CASE REPORT

Management of a Case of Obstructive Sleep Apnea with Mandibular Advancement Surgery

Gaurav Pratap Singh[1], Jayan Balakrishnan[2], Abhijeet Kadu[3]

Abstract

Aim: The aim of this case report is to highlight the use of maxillomandibular surgical advancement procedures to treat the symptoms of obstructive sleep apnea (OSA) in addition to improving esthetics, function, and stability in patients with skeletal jaw discrepancies.

Background: Obstructive sleep apnea is a condition which is frequently encountered by the orthodontist both in children and adults due to the strong correlation with various craniofacial risk factors. Although "continuous positive airway pressure (CPAP) therapy" is the gold standard for management of symptoms of OSA, patients with underlying skeletal discrepancies of the jaws can benefit from maxillomandibular surgical advancement procedures which can provide long-term benefits.

Case description: This case describes the interdisciplinary management of a 28-year-old man suffering from moderate OSA. The cause of OSA was determined to be multifactorial including lifestyle choices in addition to craniofacial risk factors of a skeletal class II jaw bases, retrognathic mandible, horizontal growth pattern, and increased submental fat deposition. The case was managed by orthosurgical line of treatment with mandibular advancement surgery to correct the underlying skeletal deformity. Post-orthognathic surgery, occlusion was settled and case was finished with class I skeletal and dental relationship with optimal functional occlusion and good esthetics. Posttreatment evaluation also revealed a marked improvement in sleep parameters with a downgrade from moderate-to-mild OSA.

Conclusion: The result highlights how suitable and timely intervention in cases of OSA can have favorable outcomes and the value of maxillomandibular advancement techniques in amelioration of OSA.

Clinical significance: Maxillomandibular surgical advancement procedures can be of great benefit to patients who suffer from symptoms of OSA owing to the underlying skeletal jaw discrepancies.

Keywords: Apnea–hypopnea index, Obstructive sleep apnea, Metabolic syndrome, Syndrome Z.

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Introduction

"Sleep is that golden chain that ties health and our bodies together."
– Thomas Dekker

The above quote certainly rings true in this age of sedentary lifestyles, occupational disorders, and disturbed eating habits. Quality sleep is important for mental and physical wellbeing. Poor quality sleep and sleep disorders are associated with a wide spectrum of comorbidities as obesity, hypertension, cardiac dysfunction, chronic obstructive pulmonary disease, restrictive lung diseases, metabolic disease, muscular dystrophy, kyphoscoliosis, hypothyroidism, and pituitary tumors.[1]

American Academy of Sleep Medicine divides the sleep-related breathing disorders into four major types as follows: Obstructive sleep apnea disorders, central sleep apnea disorders, sleep-related hypoventilation disorders, and sleep-related hypoxemia disorders.[2]

Obstructive sleep apnea is the condition that the orthodontist most frequently encounters and as the clinician deals with the craniofacial skeleton, this places him/her at a strategic advantage in early detection and screening of OSA and related sleep-disordered breathing (SDB) conditions.

Obstructive sleep apnea is defined as interrupted airflow despite persistent respiratory effort. It occurs several times every hour during sleep. Breathing continues but the airflow is blocked. This is due to the complete or partial collapse, and/or complete or partial obstructions, of the upper airway (UA) during sleep but not during wakefulness. With reduced airflow, gaseous exchange is impaired. Sleep is fragmented due to recurrent arousals.[2]

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Obstructive sleep apnea is a relatively common predicament. Various estimates peg the prevalence at 2–5% of population. Incidence increases with age and men are twice as likely to have OSA compared to women.

Although not sacrosanct, there are certain clinical features and craniofacial characteristics which can hint toward the existence of SDB in a patient. Clinical features include, but not limited to, greater prevalence of hypertension, obesity, large neck circumference, and a structurally abnormal UA.[3] It has also been observed that the patients with class II mandibular retrusion have the lowest airway

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values in terms of nasal airway volume, oropharyngeal volume and minimum axial cross-sectional area.[4]

Currently, CPAP is the gold standard in the treatment of OSA in adults. Other measures including oral appliances (OAs) and surgical management. Orthognathic surgery for OSA has shown to provide significant and long-term enlargement of velopharyngeal and overall oropharyngeal airway. It has to be appreciated that presently maxillomandibular advancement (MMA) surgery is the only treatment option that can improve polysomnography (PSG) parameters equivalent to CPAP therapy. It also becomes the primary line of treatment in patients with dentofacial deformities and those who are not amenable to CPAP.[5]

The intimate relationship shared between the orthodontist and changes in the UA dictates the importance of a multidisciplinary approach. From orthodontist's screening and referral to having an institutionalized framework which involves a patient care team comprising the orthodontist, respiratory physician, ear, nose, and throat (ENT) specialist and maxillofacial surgeon; teamwork and cooperation among specialists yield the best results for the patient.

Through this case report, we would like to present a case report of a diagnosed case of OSA who was referred to the orthodontic department of our institution for craniofacial evaluation. We highlight the judicious use of orthognathic surgery to ameliorate the condition as well as provide and improvement in esthetics and functional efficiency of the stomatognathic system.

Case Description

A 28-year-old male patient was referred to the Department of Orthodontics from the Department of Respiratory Medicine for craniofacial evaluation of risk factors associated with SDB as per the established protocols in the institution. The patient was a diagnosed case of moderate OSA with an apnea–hypopnea index (AHI) of 28 as diagnosed with overnight PSG which is the gold standard for diagnosis of OSA.[6]

Extraoral frontal examination revealed a reduced lower anterior facial height (LAFH) and a non-consonant smile arc. Profile examination revealed a convex facial profile, reduced LAFH, lower lip trap, deep mentolabial sulcus, an increased chin throat angle, and increased submental fat deposition (Fig. 1).

The intraoral examination revealed gingival recession with respect to 12, 11, and 21, traumatic deep bite, dental caries with respect to 14 and 15, class II molar and canine relation bilaterally, increased overjet and overbite, spacing in maxillary anterior tooth segment and a deep curve of spee (Fig. 2).

Cephalometric analysis showed that the patient had class II skeletal bases with orthognathic maxilla, retrognathic mandible, reduced LAFH and a horizontal growth pattern (Fig. 3 and Table 1) Functional analysis showed that the patient was a predominant mouth breather and exhibited difficulty in mastication due to an impinging deep bite. Pretreatment airway analysis was also carried out which revealed reduced oropharyngeal and hypopharyngeal airway space.

The general examination revealed that the patient had an increased body mass index (BMI) of 28 kg/m^2, increased neck circumference along with sub-mental fat deposition. Further probing revealed a history of habitual snoring, confirmed by spouse.

Based on the above findings, the patient was assessed using the Berlin Questionnaire and the Epworth Sleepiness Scale which revealed the patient to be at high risk of OSA.

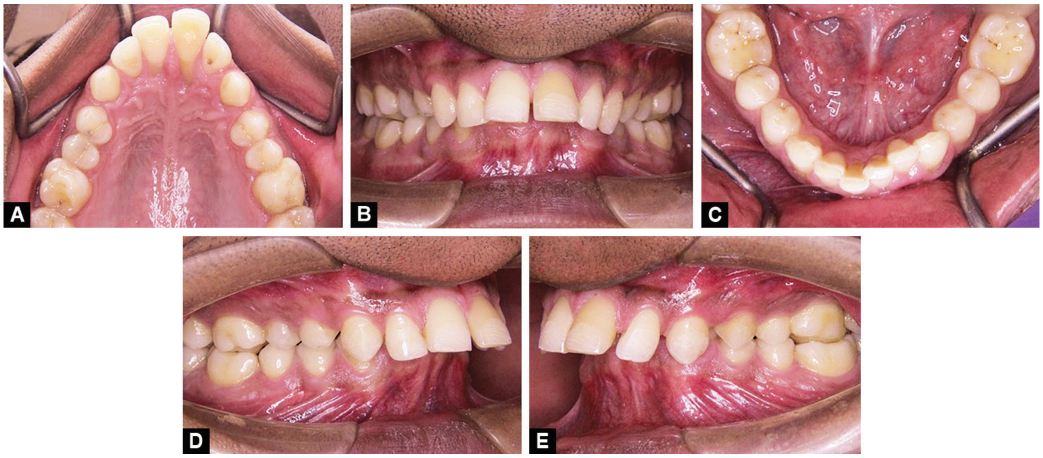
Treatment Objectives

The treatment objectives for the patient included correction of underlying pathology, improvement of profile, improvement of smile esthetics, correction of skeletal bases, and leveling and alignment of the dentition.

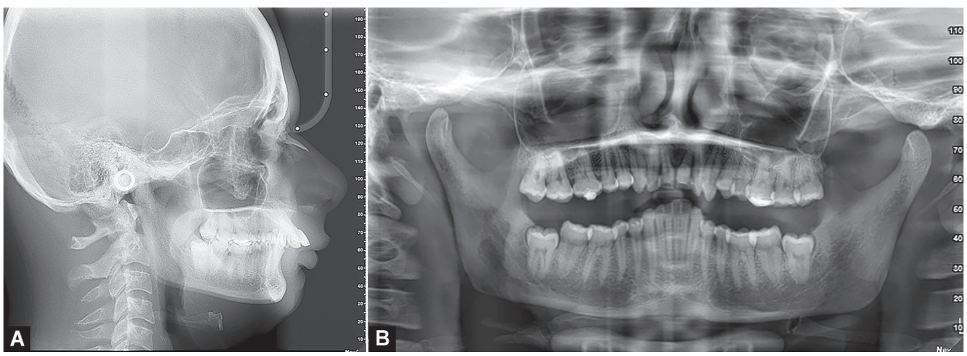
Treatment Plan

In view of the underlying OSA and the clinical, functional and radiological findings, a holistic treatment plan was agreed upon to simultaneously address all of the patient's complaints. An orthosurgical line of management was agreed upon after a consensus with all the stakeholders including the respiratory physician and the oral and maxillofacial surgeon. This was assessed to be the more beneficial plan for the patient, instead of long-term CPAP therapy, keeping in mind the underlying dentofacial deformity and the young age of the patient.

Figs 1A to C: Pretreatment extraoral photographs



Figs 2A to E: Pretreatment intraoral photographs. (A) Maxillary dentition occlusal view; (B) Mandibular dentition occlusal view; (C) Frontal view of dentition in occlusion; (D) Right lateral view of dentition in occlusion; and (E) Left lateral view of dentition in occlusion



Figs 3A and B: Pretreatment lateral cephalogram and OPG

Table 1: Cephalometric comparison of pre-, mid-, and posttreatment lateral cephalograms

Parameters	*Pretreatment*	*Presurgical*	*Posttreatment*
SNA	84°	84°	84°
SNB	77°	77°	81°
ANB	7°	7°	3°
UI-NA	8 mm/35°	4 mm/26°	4 mm/28°
LI-NB	1 mm/9°	2 mm/13°	4 mm/23°
SN-GoGn	17°	19°	28°
FMA	12°	13°	20°
IMPA	89°	94°	92°
Co-A	86	86	86
Co-Gn	113	112	115
A-N Perp	+3	+3	+3
Pog-N Perp	−2	−2	+2
Lips to E line	+3/0	0/0	−1/+1
Nasolabial angle	119° (79/40)	122° (81/41)	117° (77/40)
UI-SN	122°	109°	113°

A-N Perp, point A to nasion perpendicular; ANB, A point – nasion – B point; Co-A, condylion to A point; Co-Gn, condylion to gnathion; FMA, Frankfurt mandibular plane angle; IMPA, incisor mandibular plane angle; LI-NB, lower incisor to nasion – B point; Lips to E line, lips to esthetic line; Pog – N Perp, pogonion to nasion perpendicular; SNA, sella nasion plane to A point; SNB, sella nasion plane to B point; UI-NA, upper incisor to nasion – A point; UI-SN, upper incisor to sella nasion plane

Informed consent was obtained from the patient. Presurgical orthodontics was to be limited to levelling and alignment followed by the surgical phase which included bilateral sagittal split osteotomy (BSSO) for mandibular advancement. Postsurgical orthodontics was to settle the occlusion followed by retention phase.

Treatment plan also included patient counselling for weight reduction protocol in conjunction with medical management.

Treatment Progress

Presurgical Orthodontic Phase

Banding and bonding were carried out using 0.022″ McLaughlin, Bennett, and Trevisi (MBT) pre-adjusted edgewise appliance (PEA). All the third molars were extracted initially to allow for adequate bone healing prior to the osteotomy procedure. Leveling and alignment was carried out through progression of archwires and both the arches were placed on stabilizing archwires of 0.021″ × 0.025″ stainless steel (Figs 4 to 6).

Cephalometric prediction tracing was carried out which indicated for a mandibular advancement of 7 mm with clockwise rotation to benefit LAFH and skeletal deep bite (Fig. 7).

Based on clinico-cephalometric evaluation and prediction tracing, mock surgery was carried out and surgical splint was fabricated in accordance (Fig. 8).

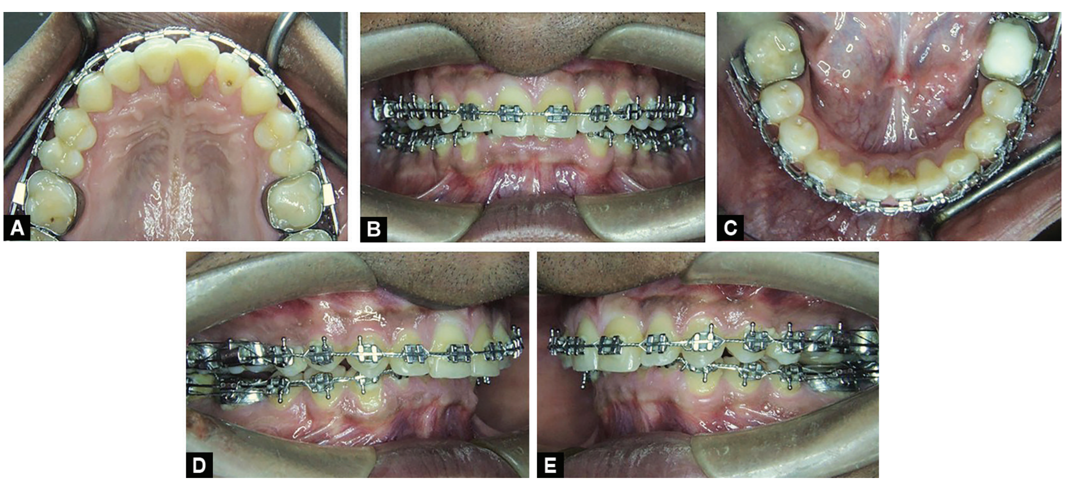
Surgical Phase

Patient was taken up for surgery under general anesthesia and mandibular advancement was carried out with BSSO. Mandibular advancement of 7 mm with clockwise rotation, as guided by surgical splint, followed by rigid fixation using titanium plates and screws was carried out (Fig. 9).

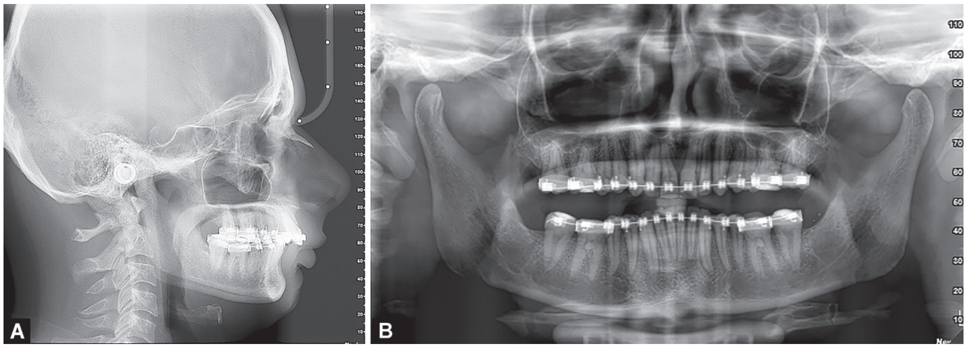
Postsurgical Phase

Postsurgical healing was uneventful (Fig. 10). The final splint was fixed intraorally for a period of 4 weeks to aid in neuromuscular adaptation along with guiding elastics. The complete orthosurgical procedure was completed in 16 months (Fig. 11).

Figs 4A to C: Presurgical extraoral photographs



Figs 5A to E: Presurgical intraoral photographs. (A) Maxillary dentition occlusal view; (B) Mandibular dentition occlusal view; (C) Frontal view of dentition in occlusion; (D) Right lateral view of dentition in occlusion; and (E) Left lateral view of dentition in occlusion



Figs 6A and B: Presurgical lateral cephalogram and OPG

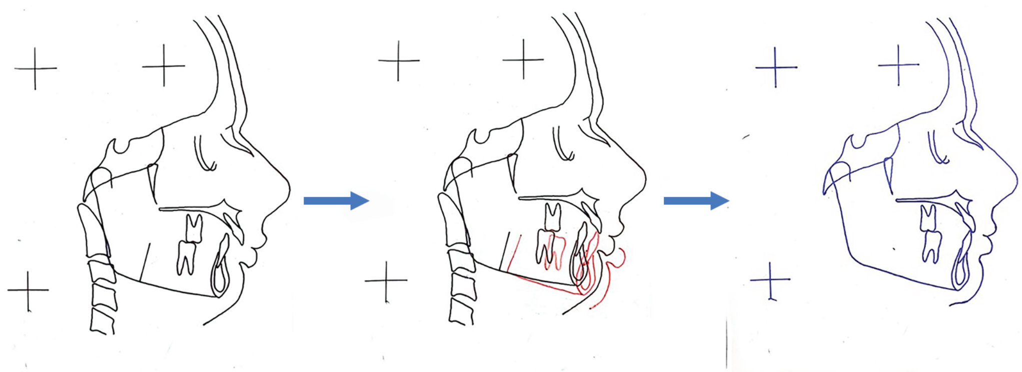
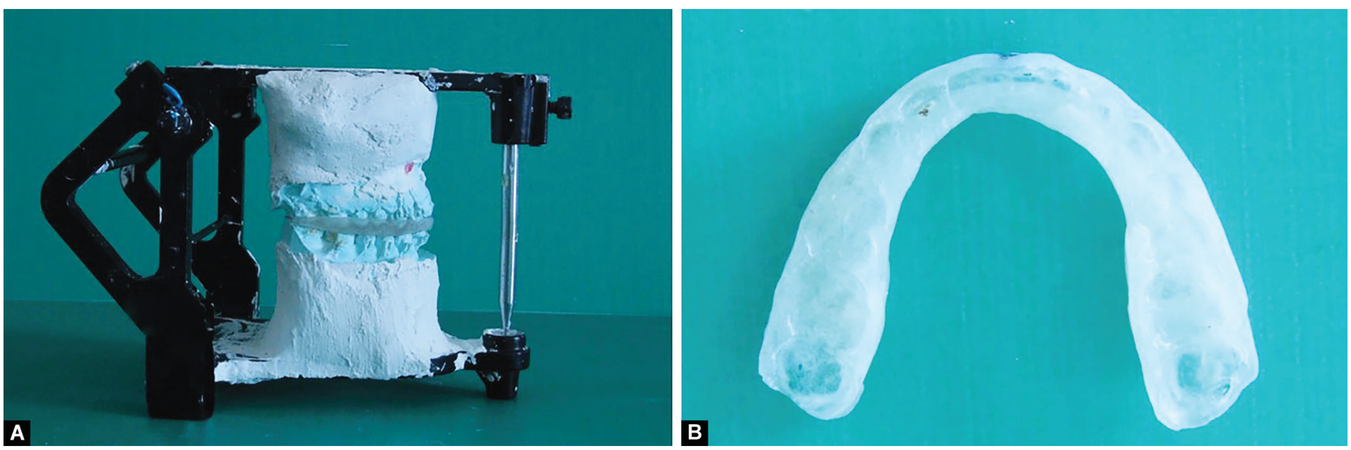


Fig. 7: Presurgical cephalometric prediction tracing



Figs 8A and B: Mock surgery and fabrication of surgical splint

During the postsurgical phase, tooth No. 15 developed secondary caries and had to be taken up for root canal treatment.

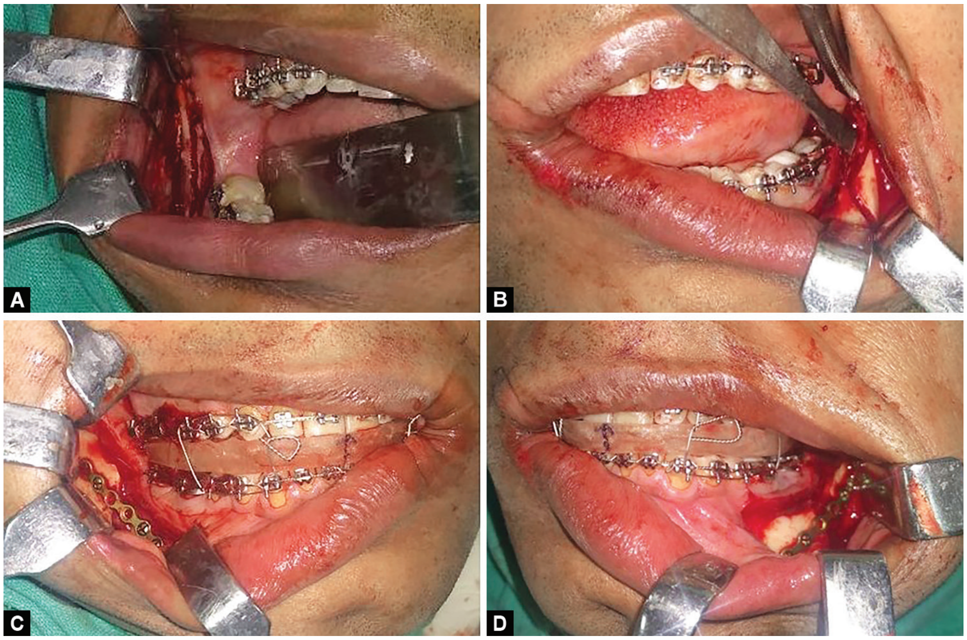
Results

Patient was referred to the respiratory physician for posttreatment PSG. The results showed significant improvement with a post treatment AHI of 8 compared with pretreatment value of 28 (Fig. 12). Patient also reported an improvement in quality of life (QOL) parameters including reduced lethargy, improved stamina, and better sleep hygiene.

Posttreatment results showed a dramatic improvement in hard and soft tissues. Extraoral frontal photograph showed an improvement of the LAFH and improvement in smile esthetics. Profile photographs also showed a marked improvement with improved LAFH, chin prominence and optimization of mentolabial and nasolabial angles. Intraoral photographs showed a commensurate improvement (Figs 13 and 14).

Posttreatment cephalometric analysis confirmed the clinical findings with improvement in LAFH, incisal angulations, and maxilla–mandibular relationships (Fig. 15 and Table 1). Posttreatment airway analysis also showed an improvement in nasopharyngeal, oropharyngeal, and hypopharyngeal airway space (Table 2).

Posttreatment orthopantomogram showed that root parallelism had been achieved (Fig. 15).



Figs 9A to D: Bilateral sagittal split osteotomy and mandibular advancement

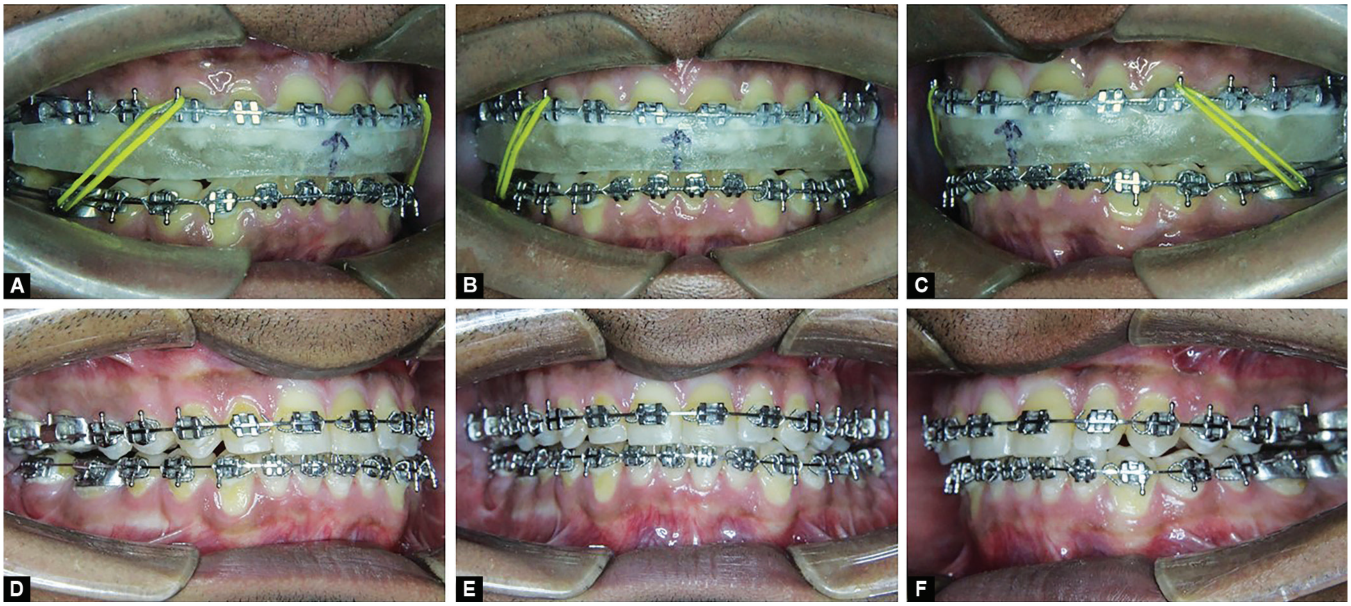
Figs 10A to C: One-week postsurgical extraoral photographs

Cephalometric superimposition revealed anterior and clockwise movement of the mandible in line with our treatment objectives of simultaneous correction of mandibular retrognathism and reduced LAFH (Fig. 16).

Discussion

Obstructive sleep apnea has been previously defined as interrupted airflow despite persistent respiratory effort. As discussed previously, OSA is associated with certain hard/soft tissue obstructions in the nasopharyngeal and oropharyngeal spaces leading to impaired breathing. As the orthodontist has an in-depth working knowledge of the craniofacial skeleton, he/she is in an advantageous position to evaluate and assess craniofacial risk factors contributing to OSA. In our institution, we have well defined the protocols for referral of patients to the orthodontist for assessment of these risk factors.

There are certain clinical and cephalometric features which predispose toward OSA. Clinical features include obesity, increased neck circumference, low waist–hip ratio, and high serum cholesterol concentration.[7] The World Health Organization (WHO) recommends cutoff points to indicate obesity at BMI more than or 30 kg/m^2 for Caucasian adults and more than or 28 kg/m^2 for Asian adults.[8] A neck circumference of more than or 43 cm for men and more than or 37 cm for women is a risk factor for OSA.[9] Certain other clinical factors include hypersomnolence, fatigue, irritability, mood swings, etc. Questionnaires are useful clinical tools which can be used to screen suspected patients before referring them for confirmatory tests. These include Friedman classification,[10] Modified



Figs 11A to F: Postsurgical alignment and leveling of dentition

Mallampati test,[11] Kushida index,[6] Berlin questionnaire,[12] STOPBang questionnaire,[13] and Epworth Sleepiness scale.[14]

Cephalometrics are a useful clinical tool which can be used to identify compromise in the airway. As far back as 1984, McNamara described his cephalometric technique which included assessment of the UA.[15] Other cephalometric variables which are suggestive of an airway compromise include mandibular retrognathism and an inferiorly positioned hyoid bone among others.

In our case, clinical features of obesity and increased neck circumference along with positive cephalometric features rung alarm bells as the cause of compromised airway. Further probing revealed a history of snoring and daytime fatigue. Use of the Berlin questionnaire helped to objectify the clinical features and indicated the need for a comprehensive and multidisciplinary treatment plan. This 10-question questionnaire is considered to be a very accurate method of predicting OSA.[12]

Currently, the gold standard of management of OSA in children and adults, in the absence of obvious nasal obstruction, is CPAP for pneumatic splinting of the airway and prevent its closure during sleep. However, CPAP is associated with certain disadvantages which include its burly and cumbersome nature, noise, high cost, need for electricity and reduced compliance which hamper its wide acceptance especially among developing countries.

The role of the orthodontist is wide and emerging in management of SDB. The orthodontist can not only help in early screening and identification of SDBs but also be part of the interdisciplinary team for management of cases of OSA. Orthodontist can advise and deliver on OA as well as surgical treatment which can be aimed at both hard and soft tissue procedures. Indications for use of OAs include patients with mil-to-moderate OSA with AHI $\geq$5 to $\leq$30 events per hour,[16,17] posture-dependent or supine-related OSA,[18] retrognathic mandible, single-site obstruction at the retroglossal region, and an oropharyngeal lumen that is narrowed anteroposteriorly but not laterally.[9]

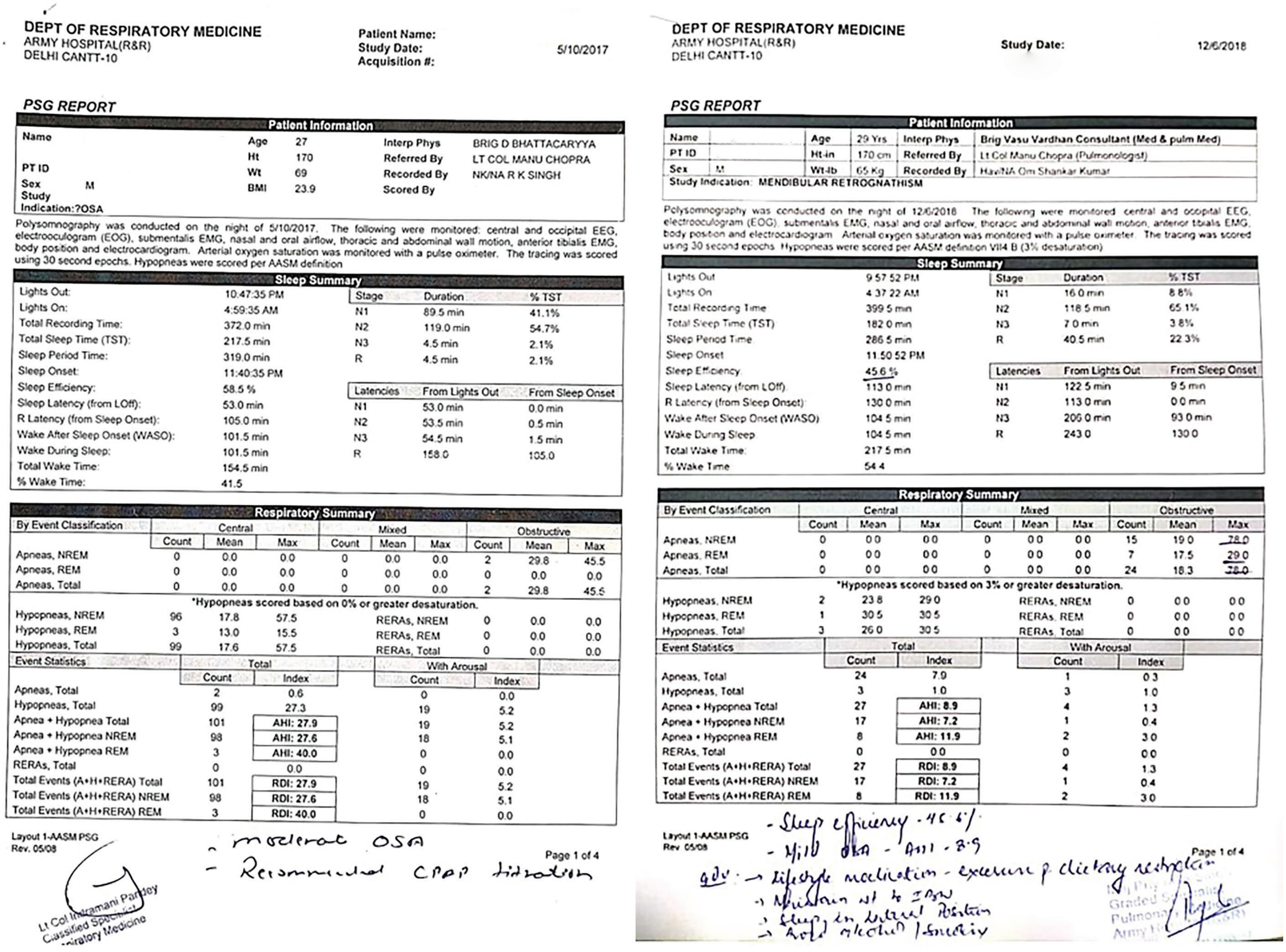
Oral surgical procedures for OSA include MMA procedures. Soft tissue response to skeletal advancement is variable and ranges from 30 to 70%. The MMAs result in the enlargement of the posterior airway and a decrease in laxity of the pharyngeal tissues. Overall, this procedure had a published short-term success rate of 97,[19] and 100%,[20] while a long-term success rate of 90% after a mean follow-up period of 4 years has also been reported.[21] Maxillary and mandibular advancement can improve PSG parameters that are comparable with CPAP therapy in the majority of patients.[5] It has been shown that for each millimeter of maxillary and mandibular bone advancement there was a 0.76-mm increase in the retropalatal region and a 1.2-mm increase in the pharynx in the retrolingual region. In addition, MMA promoted a significant repositioning of the hyoid bone in the cranial direction.[22]

Long-term follow up of MMA in cases of OSA have shown results to be stable and significant improvement noted in OSA parameters in addition to sleep quality and overall patient QOL.[23,24]

Our patient was indicated for MMA, even in the absence of SDB, with a skeletal deep bite, skeletal class II relation owing to a retrognathic mandible, reduced LAFH leading to impaired esthetics and function. The presence of OSA was a further impetus to proceed with orthosurgical line of management. Post-surgery, there was a significant reduction in AHI along with dramatic improvement in extraoral and intraoral features and alleviation in both esthetic and functional parameters. This validated our line of management as well as highlighted the importance of multidisciplinary teamwork and role of orthodontist in cases of OSA.

Conclusion

Through this case we wanted to highlight not only the use of orthognathic procedure for alleviation of SDB but also stress upon the pivotal role that an orthodontist can play in diagnosis and management of SDBs and OSA in particular. It has to be stressed upon that the orthodontist can be but only one pillar in the edifice of OSA management. Other pillars include the respiratory physician, sleep medicine specialist, oral and maxillofacial surgeon, and otorhinolaryngologist. A team which is closely coordinated, clearly communicating and efficiently practicing can



DEPT OF RESPIRATORY MEDICINE
ARMY HOSPITAL(R&R)
DELHI CANTT-10

Patient Name:
Study Date: 5/10/2017
Acquisition #:

PSG REPORT

Patient Information					
Name	Age	27	Interp Phys	BRIG D BHATTACARYYA
	Ht	170	Referred By	LT COL MANU CHOPRA
PT ID	Wt	69	Recorded By	NK/NA R K SINGH
Sex M	BMI	23.9	Scored By	
Study Indication:?OSA				

Polysomnography was conducted on the night of 5/10/2017. The following were monitored: central and occipital EEG, electrooculogram (EOG), submentalis EMG, nasal and oral airflow, thoracic and abdominal wall motion, anterior tibialis EMG, body position and electrocardiogram. Arterial oxygen saturation was monitored with a pulse oximeter. The tracing was scored using 30 second epochs. Hypopneas were scored per AASM definition

Sleep Summary

		Stage	Duration	% TST
Lights Out:	10:47:35 PM	N1	89.5 min	41.1%
Lights On:	4:59:35 AM	N2	119.0 min	54.7%
Total Recording Time:	372.0 min	N3	4.5 min	2.1%
Total Sleep Time (TST):	217.5 min	R	4.5 min	2.1%
Sleep Period Time:	319.0 min			
Sleep Onset:	11:40:35 PM			
Sleep Efficiency:	58.5 %	Latencies	From Lights Out	From Sleep Onset
Sleep Latency (from LOff):	53.0 min	N1	53.0 min	0.0 min
R Latency (from Sleep Onset):	105.0 min	N2	53.5 min	0.5 min
Wake After Sleep Onset (WASO):	101.5 min	N3	54.5 min	1.5 min
Wake During Sleep:	101.5 min	R	158.0	105.0
Total Wake Time:	154.5 min			
% Wake Time:	41.5			

Respiratory Summary

By Event Classification	Central Count	Central Mean	Central Max	Mixed Count	Mixed Mean	Mixed Max	Obstructive Count	Obstructive Mean	Obstructive Max
Apneas, NREM	0	0.0	0.0	0	0.0	0.0	2	29.8	45.5
Apneas, REM	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0
Apneas, Total	0	0.0	0.0	0	0.0	0.0	2	29.8	45.5

*Hypopneas scored based on 0% or greater desaturation.

Hypopneas, NREM	96	17.8	57.5	RERAs, NREM	0	0.0	0.0
Hypopneas, REM	3	13.0	15.5	RERAs, REM	0	0.0	0.0
Hypopneas, Total	99	17.6	57.5	RERAs, Total	0	0.0	0.0

Event Statistics	Total Count	Total Index	With Arousal Count	With Arousal Index
Apneas, Total	2	0.6	0	0.0
Hypopneas, Total	99	27.3	19	5.2
Apnea + Hypopnea Total	101	AHI: 27.9	19	5.2
Apnea + Hypopnea NREM	98	AHI: 27.6	18	5.1
Apnea + Hypopnea REM	3	AHI: 40.0	0	0.0
RERAs, Total	0	0.0	0	0.0
Total Events (A+H+RERA) Total	101	RDI: 27.9	19	5.2
Total Events (A+H+RERA) NREM	98	RDI: 27.6	18	5.1
Total Events (A+H+RERA) REM	3	RDI: 40.0	0	0.0

Layout 1-AASM PSG
Rev. 05/08

- moderat OSA
- Recommended CPAP titration

Page 1 of 4

Lt Col Indramani Pandey
Classified Specialist
Respiratory Medicine

DEPT OF RESPIRATORY MEDICINE
ARMY HOSPITAL(R&R)
DELHI CANTT-10

Study Date: 12/6/2018

PSG REPORT

Patient Information					
Name	Age	29 Yrs	Interp Phys	Brig Vasu Vardhan Consultant (Med & pulm Med)
PT ID	Ht-in	170 cm	Referred By	Lt Col Manu Chopra (Pulmonologist)
Sex M	Wt-lb	65 Kg	Recorded By	Hav/NA Om Shankar Kumar
Study Indication: MENDIBULAR RETROGNATHISM				

Polysomnography was conducted on the night of 12/6/2018. The following were monitored: central and occipital EEG, electrooculogram (EOG), submentalis EMG, nasal and oral airflow, thoracic and abdominal wall motion, anterior tibialis EMG, body position and electrocardiogram. Arterial oxygen saturation was monitored with a pulse oximeter. The tracing was scored using 30 second epochs. Hypopneas were scored per AASM definition VIII4 B (3% desaturation)

Sleep Summary

		Stage	Duration	% TST
Lights Out	9 57 52 PM	N1	16 0 min	8 8%
Lights On	4 37 22 AM	N2	118 5 min	65 1%
Total Recording Time	399 5 min	N3	7 0 min	3 8%
Total Sleep Time (TST)	182 0 min	R	40 5 min	22 3%
Sleep Period Time	286 5 min			
Sleep Onset	11 50 52 PM			
Sleep Efficiency	45 6 %	Latencies	From Lights Out	From Sleep Onset
Sleep Latency (from LOff)	113 0 min	N1	122 5 min	9 5 min
R Latency (from Sleep Onset)	130 0 min	N2	113 0 min	0 0 min
Wake After Sleep Onset (WASO)	104 5 min	N3	206 0 min	93 0 min
Wake During Sleep	104 5 min	R	243 0	130 0
Total Wake Time	217 5 min			
% Wake Time	54 4			

Respiratory Summary

By Event Classification	Central Count	Central Mean	Central Max	Mixed Count	Mixed Mean	Mixed Max	Obstructive Count	Obstructive Mean	Obstructive Max
Apneas, NREM	0	0 0	0 0	0	0 0	0 0	15	19 0	78 0
Apneas, REM	0	0 0	0 0	0	0 0	0 0	7	17 5	29 0
Apneas, Total	0	0 0	0 0	0	0 0	0 0	24	18 3	78 0

*Hypopneas scored based on 3% or greater desaturation.

Hypopneas, NREM	2	23 8	29 0	RERAs, NREM	0	0 0	0 0
Hypopneas, REM	1	30 5	30 5	RERAs, REM	0	0 0	0 0
Hypopneas, Total	3	26 0	30 5	RERAs, Total	0	0 0	0 0

Event Statistics	Total Count	Total Index	With Arousal Count	With Arousal Index
Apneas, Total	24	7.9	1	0 3
Hypopneas, Total	3	1 0	3	1 0
Apnea + Hypopnea Total	27	AHI: 8.9	4	1 3
Apnea + Hypopnea NREM	17	AHI: 7.2	1	0 4
Apnea + Hypopnea REM	8	AHI: 11.9	2	3 0
RERAs, Total	0	0 0	0	0 0
Total Events (A+H+RERA) Total	27	RDI: 8.9	4	1 3
Total Events (A+H+RERA) NREM	17	RDI: 7.2	1	0 4
Total Events (A+H+RERA) REM	8	RDI: 11.9	2	3 0

Layout 1-AASM PSG
Rev 05/08

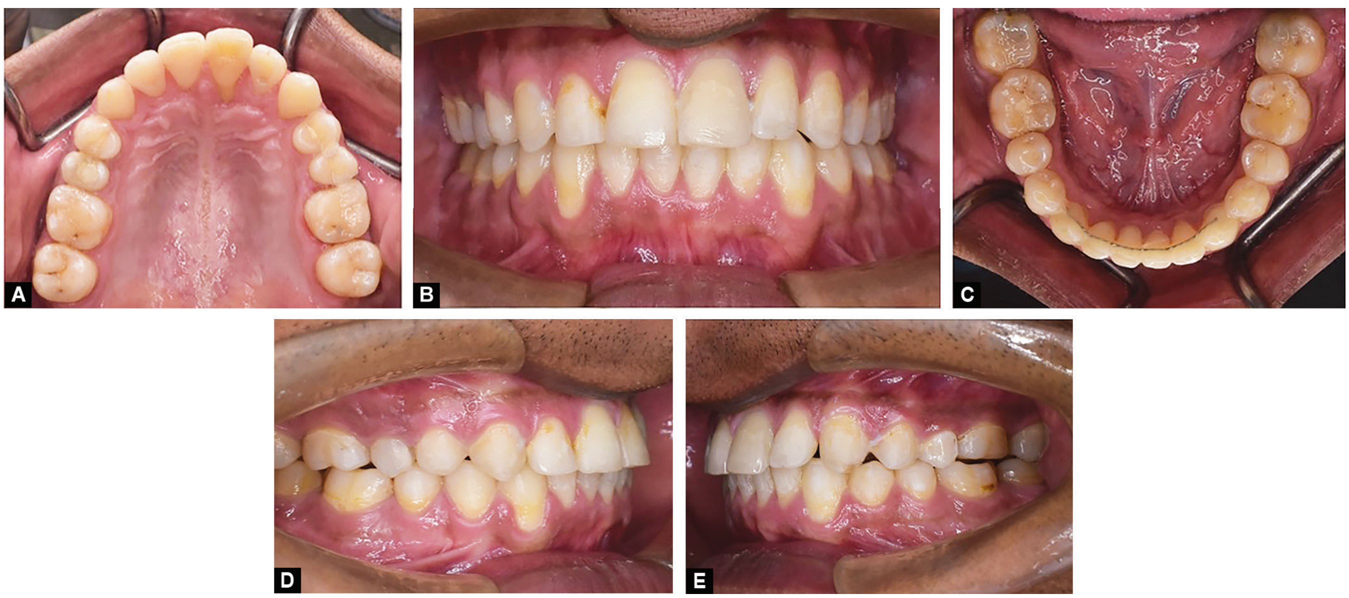
- Sleep efficiency - 45.6%
- Mild OSA - AHI - 8.9

Adv: → lifestyle modification - exercise & dietary restriction
→ Maintain wt to IBW
→ Sleep in lateral position
→ Avoid alcohol / smoking

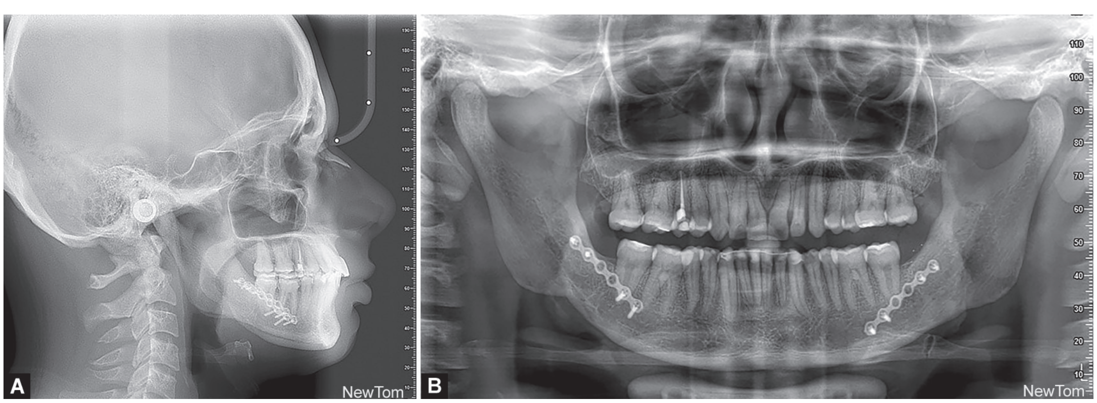
Page 1 of 4

Fig. 12: Pretreatment and posttreatment overnight PSG report

Figs 13A to C: Posttreatment extraoral photographs



Figs 14A to E: Posttreatment intraoral photographs. (A) Maxillary dentition occlusal view; (B) Mandibular dentition occlusal view; (C) Frontal view of dentition in occlusion; (D) Right lateral view of dentition in occlusion; and (E) Left lateral view of dentition in occlusion



Figs 15A and B: Posttreatment lateral cephalogram and OPG

Table 2: Airway analysis. Pretreatment and posttreatment

Parameter	*Norms*	*Pretreatment*	*Post-surgical*
Nasopharyngeal airway space	25.9 + 2.6 mm	22 mm	26 mm
Oropharyngeal airway space	10.1 + 3.1 mm	8 mm	13 mm
Hypopharyngeal airway space	18.7 + 2.6 mm	8 mm	15 mm
Hyoid distance	<15 mm	13 mm	9 mm

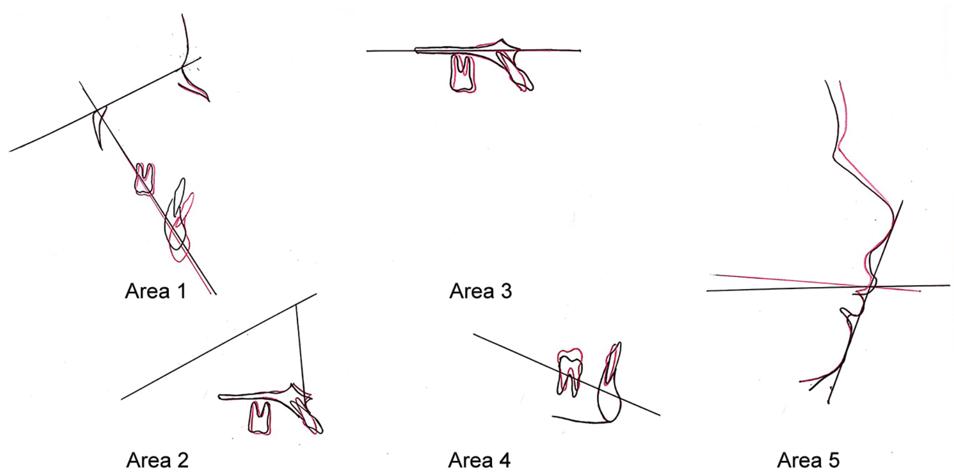


Fig. 16: Ricketts' area-wise superimposition. Black, pretreatment; Red, posttreatment

bring the best of care to a patient populace which is only going to increase in times ahead.

Clinical Significance

- Interdisciplinary management is the key to achieving the best and sustainable results in cases of OSA.
- Orthodontist can play a key role in early diagnosis and management of cases of OSA.
- Mandibular advancement devices and maxillomandibular surgical procedures are valid adjuncts to management of cases with OSA.
- Maxillomandibular surgical advancement procedures can provide long-term benefit in symptoms of OSA in addition to correction of underlying skeletal discrepancies.

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