ORIGINAL ARTICLE

IS SEVERITY OF OBSTRUCTIVE SLEEP APNOEA IS A SURROGATE MARKER OF CPAP TITRATION PRESSURE?

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ABSTRACT

Background: Obstructive sleep apnoea (OSA) is emerging epidemic and its presence often goes unnoticed. It may be either consequence or cause of several non-communicable life style related disorders. Identifying OSA and timely intervention makes remarkable difference quality of life and co-existing disorders. CPAP is the mainstay of treatment in dynamic obstruction related OSA. However patient undergoing CPAP titration is economical burden and time consuming. We tried to look at whether severity of OSA can predict approximate CPAP pressure required for domiciliary use.

Methodology: We retrospectively analyzed data of patients who have undergone CPAP titration and there severity of OSA based on Apnoea hypopnoea index. We subcategorised them in to mild, moderate and severe categories

Results: We looked at 17 serial patients who underwent CPAP titration. Using unpaired t test and two tailed p values, we analysed difference between mild and moderate, mild and severe and moderate and severe categories, however no statistical significance was noted with regard to CPAP titration pressure between these groups.

Conclusion: There was no correlation between CPAP pressure and severity of OSA noted in our study.

Keywords: OSA, CPAP, Airway resistance

INTRODUCTION

Obstructive sleep apnoea (OSA) with its inherent nature of complicating and co-existing with other life style disorders such as Diabetes mellitus, Cardiovascular morbidities like hypertension, arrhythmia and ischemic diseases, Dyslipidemia and other endocrinopathies, adds significant risks to already existing health hazards. OSA with repetitive hypoxia and hyperoxia creates a state of inflammation at the cellular level which possibly contributes to insulin resistance and may progress to glucose intolerance and diabetes mellitus. Due to continued neural signals to keep 'effort to breath' intact against collapsed upper air way results in disturbance of pressure mechanics in the thoracic cavity, which results in sympathetic overstimulation and arrhythmias. Persistent sympathetic stimulation may lead to development of hypertension. OSA also complicates cognitive functions and quality of life by increasing day time sleepiness. So it is imperative to diagnose and in-

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tervene OSA at right time so that maximum positive difference can be made to this group of patients.

In 2006, a population-based survey from north India had estimated the prevalence of OSAS at 3.6 per cent (males and females being 4.9 and 2.1% respectively)¹. The prevalence rates of OSA and OSAS in males were 13.4 and 4 per cent respectively whereas in females, these were 5.6 and 1.5 per cent respectively². Another hospital-based study from north India revealed an estimated prevalence of OSA and OSAS to be 4.4 and 2.4 per cent in males, whereas it was 2.5 and 1 per cent in females³.

Continuous positive airway pressure (CPAP) form the important therapeutic modality, however in country like India where awareness level and economic constraints determines the clinical course motivating patient to undergo overnight CPAP titration study following diagnostic Polysomnography becomes a tall order for the clinician. To find out scientific way to circumvent CPAP titration study, we tried looking at whether severity of sleep apnoea (vide infra), can help us empirically set the CPAP pressure required for domiciliary use.

METHODOLOGY

After obtaining ethical clearance from the institutional ethics committee, we retrospectively analysed the consecutive patients who have undergone CPAP titration for obstructive sleep apnoea. We have sub-categorized them in to mild (Apnoea Hypopnoea Index {AHI} - 5-15/hr), moderate (AHI- 15-30/hr) and severe (AHI- >30/hr) obstructive sleep apnoea. Further CPAP titration pressure which was prescribed for domiciliary CPAP use as standard protocol was taken in to consideration and correlated with severity of OSA. All were previously diagnosed as OSA patients following full night polysomnography. Full night CPAP titration was done with constant monitoring the respiratory events and snoring. We analysed the mean CPAP titration pressure required in mild, moderate and severe OSA groups. We also looked at the range of CPAP titration pressure in each group.

Statistics: we looked for statistical significance using unpaired t test and two tailed P value.

RESULTS

We enrolled 17 consecutive patients of OSA who have undergone CPAP titration for domiciliary use. Among which three had mild OSA, five were having moderate OSA and remaining eight had severe OSA (Table 1). Mean of CPAP titration pressure in mild OSA was 7.33 cm of H2O, where as in moderate OSA mean CPAP titration pressure was 8.2 cm of H20. In severe OSA mean titration pressure was 10 cm of H20, also median and mode was also 10 cm of H20.

First we correlated between mild and moderate OSA. Standard error of mean (SEM)was 1.0. The two-tailed P value equals 0.5879, conventionally this difference is considered to be not statistically significant. The mean of group one minus group two equals -0.8700. 95% confidence interval of this difference: from -4.5899 to 2.8499.

Then we compared mild & severe OSA which has SEM of 3.5.The two-tailed P value equals 0.7023 even this difference is considered to be not statistically significant. The mean of group one minus group two equals -2.6700. 95% confidence interval of this difference: from -17.6508 to 12.3108.

Table 1: CPAP distribution across groups

Severity of OSA	CI	PAP	titra	tion	press	ure (In C	m H2	20)
Mild	6	8	8						
Moderate	7	7	8	9	10				
Severe	5	6	10	10	10	10	12	13	14

Table 2: Mean, median and mode of CPAPpressure

Severity of OSA	CPAP Titration pressure				
	Mean	Median	Mode		
Mild	7.33	8	8		
Moderate	8.2	8	7		
Severe	10	10	10		

Table 3: Sex wise distribution of cases and se-verity of OSA

Severity of OSA	Male	Female	
Mild	2	1	
Moderate	2	3	
Severe	5	4	

Finally we compared between Moderate & severe OSA with SEM- 2.5, again two-tailed P value equals 0.6588 being statistically not significant. The mean of group one minus group two equals -1.800. 95% confidence interval of this difference: from - 10.405 to 6.805

DISCUSSION

To best of our knowledge our study is the first reported scientific communication correlating severity of OSA (apnoea hypopnoea index). We found out that no scientific correlation can be made between CPAP pressure required for domiciliary use and severity of OSA based on AHI. CPAP titration pressure required for domiciliary use in severe OSA patients ranged from 6cm of H2O to 14 cm H2O. Previous researchers who have worked on air way resistance in sleep apnoea have reported that, it depends on multiple factors. One group of researchers have shown that dilator muscle activation relates both to anatomical airway narrowing/collapsibility (positive end-expiratory pressure) and to airway patency (pharyngeal resistance) across a group of subjects with obstructive sleep apnoea4. These findings are consistent with the model of passive anatomical factors and muscle activation (in part driven by the negativepressure reflex) being the principal determinants of pharyngeal patency. The relationship between

critical closing pressure and genioglossus muscle activation weakens when dilator tone diminishes with loss of the wakefulness drive at sleep-onset but, for the activity of tensor palatine it becomes stronger suggesting persistence of the negativepressure reflex during early sleep. Another group of researchers who worked on Japanese population observed that aside from age and obesity, anatomical and functional abnormalities are significantly related to the severity of OSA. Predominant determinants of AHI differed depending on the severity of OSA or the magnitude of obesity⁵. CPAP is essentially used to overcome airway resistance during sleep. As it is clear from the observations made above airway resistance is the interplay of complex things and desired CPAP pressure may vary from patient to patient and age old idiom 'once size do not fit all' holds good here too.

CONCLUSION

Severity of OSA and CPAP pressure required for domiciliary use do not correlate linearly and CPAP titration should be done before domiciliary CPAP use.

REFERENCES

- Sharma SK, Kumpawat S, Banga A, Goel A. Prevalence and risk factors of obstructive sleep apnoea syndrome in a population of Delhi, India. Chest 2006; 130 :149-56.
- Reddy EV, Kadhivaran T, Mishra HK, Sreenivas V, Handa KK, Sinha S, et al. Prevalence and risk factors of obstructive sleep apnoea among middle-aged urban Indians: A communitybased study. Sleep Med 2009; 10: 913-8.
- 3. Vijayan VK, Patial K. Prevalence of obstructive sleep apnoea syndrome in Delhi, India. Chest 2006: 130 : 92S.
- R. Pierce, D. White, A. Malhotra, J. K. Edwards, D. Kleverlaan, L. Palmer, J. Trinder .Upper airway collapsibility, dilator muscle activation and resistance in sleep apnoea. Eur Respir J 2007; 30: 345–353.
- Kensaku Aihara et al. Analysis of anatomical and functional determinants of obstructive sleep apnea. Sleep and Breathing June 2012, Volume 16, Issue 2, pp 473-481