The use of overnight pulse oximetry for obstructive sleep apnoea in a resource poor setting in Sri Lanka

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Abstract

Objective: To study patients with symptoms suggestive of obstructive sleep apnoea using overnight oxymetry, and describe their symptoms, risk factors and co-morbidities.

Methodology: Overnight oxymetry was carried out on consecutive patients presenting with snoring and daytime sleepiness. Daytime sleepiness was assessed with Epworth sleepiness score (ESS). Their body mass index (BMI), presence of hypertension, neck circumference, oropharyngeal Mallampathi index, thyroid status, co-morbidities, alcohol, smoking, and medication use were noted. The number of oxygen desaturations below 4% of the baseline, per hour of sleep (oxygen desaturation index: ODI) was calculated and a plot of oxygen saturation versus time was analyzed for graph morphology. Association of ODI with other clinical parameters was statistically analyzed.

Results: Forty five patients (15 females) with a mean age of 44 (±13) years underwent overnight oxymetry. Their mean BMI was 27.62 (±8.82) kg/m2, 11(24%) were obese (BMI ≥30kg/m2) and 21 (46%) were hypertensive. They complained of snoring (91%), nocturnal waking up (82%), nocturnal choking (35 %), witnessed apnoeic episodes (31%), waking up tired (16%), nocturia (31%) and waking up with headache (20%). Mean ODI was 9.32 (±9.66)/hr. Twenty six (57%) patients had an abnormal ODI above 5/hr with saw tooth appearance in oxymetry tracings and 33% of them had an ODI ≥10/hr. Hypertension, nocturia and ESS showed a significant association with ODI (P 0.05). ODI significantly correlated with ESS (P=0.02) and BMI (P=0.006).

Conclusion: Sleep apnoea is prevalent than expected, though largely unrecognized. Obesity, higher Epworth score, nocturia and hypertension indicated risk factors for OSAS. Use of widely available pulse oximetry is a practical, low cost approach to investigate sleep disordered breathing in resource poor settings.

Introduction

Obstructive sleep apnoea syndrome (OSAS) is characterized by instability of the upper airway during sleep, which results in markedly reduced (hypopnoea) or absent (apnoea) airflow at the nose and or mouth with accompanying desaturation of oxyhaemoglobin. These episodes are terminated by brief micro-arousals that result in sleep fragmentation and diminished amounts of slow wave and rapid eye movement sleep[1]. Diagnosis of OSAS requires both the characteristic clinical features and objective demonstration of sleep-disordered breathing since clinical prediction models alone have failed to establish sleep apnoea which require therapy. The "gold standard" for the diagnosis of OSAS is polysomnography, which provides detailed information on sleep stage, air flow and oxygen saturation, heart rate and rhythm, body position, muscle tone and contraction etc[1]. However, these studies are resource intensive, and allow only a minority of affected patients to be assessed even in the affluent settings. Hence given the high prevalence of OSAS and limited availability of polysomnography, simplified approaches to the diagnosis of OSAS have become essential. Oximetry appears to be the most reliable and predictive component of polysomnography[2]. It has shown to be a less expensive and effective substitute for polysomnography with substantial accuracy when the pretest probability for obstructive OSAS is high and is shown to be as good as polysomnography in detecting clinically significant OSAS requiring intervention[2]. Frequency, depth and length of oxygen desaturation can be measured with oximetry. Oxygen saturation vs time waveform morphologies can help discriminate between obstructive apnoeas and hypopnoeas, as well as between obstructive and central apnoeas. The failure to recognize clinically significant OSAS is of particular importance as this is associated with significant morbidity and mortality. Locally, sleep investigation is at its infancy and snoring is not scientifically evaluated for most patients. It is difficult to justify the significantly higher expense of polysomnography for all the patients. However, the widely available oximetry should not certainly be denied to our patients and we endeavour to use pulse oxymetry in a resource poor setting to explore sleep disordered breathing among our patients.

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Methodology

With institutional approval we offered overnight pulse oximetry to consecutive patients presenting to Respiratory Unit, General Hospital, Kandy, with symptoms of snoring and daytime somnolence and fatigue for a duration of one year from December 2008 to December 2009. Their severity of daytime sleepiness was assessed using Epworth sleepiness score obtained via physician administered questionnaire. Patients were considered to have significant daytime sleepiness if the score is above 10. We noted their known risk factors for getting OSAS and co-morbid disease conditions including a prior history of hypertension, BMI, blood pressure, neck circumference, oropharyngeal assessment with Mallampati index, thyroid function tests and alcohol and medication use. Hospital admitted patients, one at a time was let asleep overnight in an undisturbed room with a alarm muted Dolphin 2100 pulse oximeter probe fixed on to their finger, having given detailed instructions on its placement. Oxygen saturation (SaO_2) and pulse rate was captured every two seconds for 8 hours from 8.00 pm to 5.00 am. Cumulative time with SaO_2 less than 90%, and the total recording time of the study was recorded. The number of oxygen desaturations of 4% below the baseline per hour of sleep (oxygen desaturation index – ODI) was calculated using computer software. Baseline was defined as the mean oxygen saturation in the 2 minutes preceding the onset of an event and an event was considered to last 10 seconds or longer. Artifactual data with poor pulse rate recording were excluded from analysis. A plot of oxygen saturation versus time was drawn and graph morphology analyzed. Patients with ODI more than 10 were diagnosed as having sleep apnoea. Association of ODI with clinical parameters was analyzed statistically using chi square test and Fisher's exact test when the patient number was low, and a correlation sought between ODI and BMI, Epworth score, Mallampati score neck circumference and neck length. Pearson correlation was used for continuous data and Spearman correlation for ranked data. SPSS 17.0 was used for statistical analysis.

Results

Clinical demographics

Fourty five patients were studied with overnight pulse oximetry whose mean age (±SD) was 44 (±13) years. 15 were females. Their mean BMI was 27.62 (±8.82) kg/m^2. 21 (46%) patients were hypertensive and 11 (24%) were obese (BMI ≥30 kg/m^2), 13 (28%) were overweight (Table 1).

Snoring was the commonest complaint (91%). Waking up in the night (82%), nocturnal choking (35%), Witnessed apnoeic episodes (31%), waking up tired (16%) and waking up with headache (20%) were among other complaints. 14 (31%) had nocturia.

Oxymetry results

Average oxymetry duration was 8.5 (±1.02) hours. They exhibited an ODI of 9.32 (±9.66)/hr. Any ODI above 5/hr was considered abnormal and an ODI of 15 to 30/hr moderate and above 30/hr was considered severe. 26 (57%) Patients had an abnormal ODI above 5 and 33% had ODI, 10/hr or above (Table 2). Oxymetry tracings of all the 24 patients with ODI above 5/hr showed a saw tooth appearance.

Further we evaluated the association between clinical variables and oximetry outcome (Table 3). Hypertension and nocturia showed a significant association with ODI above 10/hr ($P<0.05$). This association was not significant when cut off ODI was 5/hr. Similarly an Epworth score of 13 or above showed a significant association with ODI above 10/hr. However, Epworth score of 11 did not show a significant association with ODI.

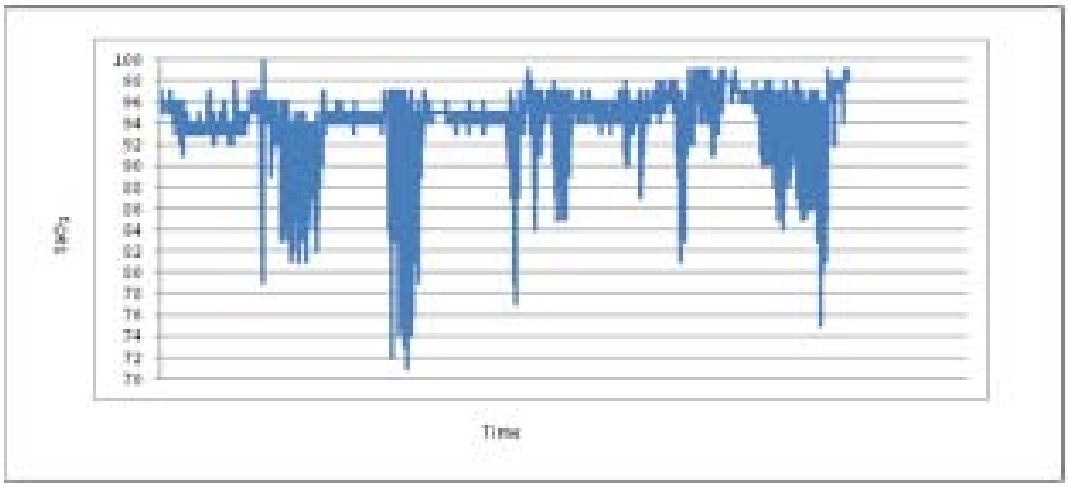

Figure 1. Oxymetry tracing of a patient showing saw tooth appearance, ODI was 26.

Table 1. Co-morbid disease of the patients presenting with snoring and sleepiness

Co-morbid illness	*No. of Patients*
Hypertension	21 (46%)
Diabetes	7 (15%)
Hypothyroidism	4 (8%)
Ischemic heart disease	2 (4%)
Stroke	1 (2%)
Peripheral vascular disease	4 (8%)
Allergic rhinitis	19 (42%)
Nasal polyps	6 (13%)
Enlarged adenoids	3 (6%)
Asthma	16 (35%)
COPD	1 (2%)
Micrognathia	1 (2%)
Depression	1 (2%)
Alcohol	6 (13%)
Smoking	2 (4%)
Insomnia	9 (20%)
Nocturia	14 (31%)

Table 2. BMI, ESS and oxymetry results in different severity groups of OSAS

ODI	*<5/hr*	*5-10/hr*	*10-15/hr*	*15-30/hr*	*>30/hr*
No of patients	19	11	4	9	2
Total desaturation (hours)	0.23±0.26	1.35±1.93	1.53±1.19	8.54±0.75	7.57
No of desaturation events	14.7±12.1	61.27±15.52	84±29.47	186.44±38.80	263±49.5
BMI mean±SD (kg/m^2)	23.91±11.52	28.15±4.10	33.04±4.82	31.73±4.10	23.9
Epworth sleepiness score	8.8±5.7	9.1±4.7	12.6±0.5	14±5.8	18±4

Table 3. Clinical features of patients and their corresponding oximetry results

Clinical feature	*No of patients*	*ODI <10/hr N = 30*	*ODI≥10/hr N = 15*	*P value*
Male sex	30	18	12	0.67
Witnessed apnoea	16	9	7	0.22
Waking up tired	14	18	9	0.89
Insomnia	9	5	4	0.43
Nocturia	15	7	8	0.04
Epworth score ≥13 (3 missing data)	16	7	9	0.02
Hypertension	31	10	11	0.01
Body mass index >30 (3 missing data)	15	7	8	0.37
Alcohol	6	4	2	0.16

Table 4. Correlation of ODI with clinical parameters

Clinical feature	*Correlation coefficient*	*p value*
BMI	0.04	0.006
Mallampathi score	-0.145	0.334
Epworth	0.349	0.020
Neck length	-0.273	0.186
Neck circumference	0.025	0.907

All the patients having ODI above 5/hr complained of snoring always or time to time. Moderate to severe ODI patients had snoring every day. Of the two patients with a severe ODI above 30 one patient was hypothyroid, and the other had micrognathia and nasal polyps.

The patient with COAD had a positive ODI of 17.9. His oxymetry tracing showed sawtooth appearance without sustained desaturations.

ODI significantly correlated with Epworth sleepiness score (P<0.05) and BMI (P=0.02) (Table 4).

Discussion

We were able to positively diagnose sleep apnoea with overnight oxymetry in a suspected group of patients complaining of snoring and day time sleepiness. Male to female ratio in this consecutive group of snorers was 2:1. Similar male female ratios are found in larger population surveys, snoring being commoner in males[3]. Their age range was 18 to 73, with a mean age of 44.4 years. Male to female ratio in patients with a positive test was 2.25:1.

A 4% drop of saturation below baseline was taken as a desaturation event in the calculation of ODI. There is no universally accepted definition of an oxygen desaturation in sleep-disordered breathing. However, in most publications, an oxygen desaturation is defined as a decrease of ≥4% from baseline SaO_2[2]. Though one definition is in common use for an oxygen desaturation, no such uniform definition exists for a normal or abnormal ODI. There are generally three cutoff points for an abnormal ODI that appear to mirror the

definition of an abnormal apnoea-hypopnoea index. The thresholds for an abnormal ODI are either ≥5, ≥10, or ≥15 desaturations per hour. There is little evidence of one definition having greater validity than the others[2,4]. Therefore it was difficult to define abnormality in the case of mild positivity of ODI 5 - 10 /hr in this series of patients without confirmatory polysomnography. There were 57% patients with ODI of 5/hr or above and 33.33% with ODI of 10/hr or above. A significant association of ODI with other clinical predictors of sleep apnoea, such as hypertension, obesity and nocturia was observed only at ODI 10/hr. Therefore ODI 10/hr seems to indicate a greater degree of severity of sleep apnoea with its resulting manifestations. Further investigation with polysomnography would have been ideal for those patients with marginally positive test on oxymetry. However, it has been shown that oxymetry would be enough to diagnose patients warranting initiation of therapy in comparative treatment based studies with polysomnography[2].

In population studies up to 34% of habitual snoring patients have had a positive test when the diagnosis was made with polysomnography[5,6]. In our sample 65% of the habitual snoring men had a positive test. This group of patients had a higher likelihood of sleep apnoea since, apart from being snores other inclusion criteria such as daytime sleepiness was also taken into account. There was no statistically significant association with habitual snoring and a positive test at any of the ODI levels. Sleep apnoea is found only in about 1.4% snorers and it is known that snoring does not necessarily indicate that apnoea is present. Alternatively snoring is not essential for the occurrence of sleep apnoea[6]. In our series of patients also, one patient who did not complain of snoring had a positive test with ODI 11/hr. If snoring alone is not predictive enough as mentioned, other parameters become important in deciding whom to test.

In recruitment of patients to our study, to assess daytime sleepiness we used Epworth score in which the patient is considered sleepy if the score was 11 or above[7,8]. There was a significant correlation between ODI and Epworth score. A total Epworth score of 13 showed significant association with a positive ODI above 10/hr. This was not true with Epworth 11 which is the accepted cut off value. We had to adjust the questionnaire to suit the particular individual, at times, since all the items in the questionnaire were not applicable. For instance driving in traffic lights, regular attendance in public places were not daily routines of our patients which would explain the difference in clinically significant cut off value in our series. A validated questionnaire suited to our setup seemed essential during the study.

Only complaint that had a significant association with a positive result was nocturia which is a recognized manifestation of OSA though the exact pathogenic mechanism is not clear. Increased venous return to right ventricle caused by negative intrathoracic pressure generated during increased ventilatory effort and hypoxic episodes is thought to increase ANP levels causing diuresis. Positive intra-abdominal pressure with the effort, which is transmitted to the bladder, is thought to contribute as well[9,10,11].

Hypertension and BMI was significantly associated with a positive test of ODI above 10/hr. The BMI showed a significant correlation with ODI as has been observed in other studies. We have studied a group consisting more of overweight patients since 73% were overweight with BMI above 25 kg/m^2. There was no significant correlation in ODI with Mallampathi score, neck circumference or neck length. Possible mechanisms whereby OSAS may contribute to hypertension in obese individuals include sympathetic activation, hyperleptinemia, insulin resistance, elevated angiotensin II and aldosterone levels, oxidative and inflammatory stress, endothelial dysfunction, impaired baroreflex function, and by effects on renal function[12]. The coexistence of OSAS and obesity has implications for cardiovascular dysfunction in obese individuals. The presence of resistant hypertension and the absence of a nocturnal decrease in blood pressure in obese individuals prompt OSAS, especially if clinical symptoms also are suggestive[12].

Two patients in this study showed a strongly positive test with ODI above 30/hr. As expected strong co morbidity contributing to their sleep apnoea was observed, since one of the patients was hypothyroid and the other had micrognathia and nasal polyps.

Visual analysis of oxygen saturation versus time graph revealed saw tooth appearance compatible with sleep apnoea including the graph of the patient with chronic obstructive airways disease.

During this study 12 patients had inadequate recording for analysis and had to undergo repeat testing. This was mainly during the initial phase of the study due to technical errors of recording. Few patients had probe dislodgement during the night. There were no observers during the night and the alarms had to be muted to avoid disturbance. An observed overnight study would overcome these pitfalls and would add other information regarding snoring and apnoea as well. However, even in developed world trend in overnight oxymetry studies is turning towards unobserved and home based studies.

Most of the patients studied did not recognize their snoring and sleepiness as pathological. Patients had to undergo education on sleep hygiene. A sleep clinic was established and the diagnosed patients were followed up with lifestyle modifications and necessary

medical treatment. Though marginally positive patients with ODI 5 to 10 were referred for polysomnography, none of them could afford it.

This study was an initial step towards a preliminary sleep lab, with utilization of already existing resources, well suited to our setting without additional cost. Its results also give evidence that sleep apnoea exist in large proportions, undiagnosed and unsuspected with its associated morbidities. Snoring could no more be considered natural. Other supportive elements like sleep questionnaire should be developed and validated. Oxymetry alone is shown to detect clinically significant sleep apnoea and this is an era where worldwide sleep disordered breathing investigation is shifting towards unobserved and low cost oxymetry based tests. Polysomnography essentially have a role in arbitrary and milder cases in specialized centers. Oxymetry however would be a better practical low cost tool for widespread use in resource poor setting in Sri Lanka.

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