Clinical profile of sleep apnea syndrome
A study at a University Hospital

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ABSTRACT

Objectives: To review a series of patients with sleep apnea syndrome, to promote more awareness and alert local health professionals to early diagnosis and treatment.

Methods: We studied, prospectively, 48 consecutive patients who were managed at the university hospital from 1992 to 1996.

Results: The male:female ratio was 1.4:1. The mean interval between onset of symptoms and the diagnosis was 5.5 years (range 0.25 to 30). In over half of the patients the diagnosis was not suspected upon referral. The mean body mass index was 42.8kg/m^2, (range 25 to 76). Daytime hypoxemia was present in 28 patients (58%), while 26 (54%) had Pa CO_2 > 45 mmHg, mainly as a result of obesity-hypoventilation syndrome. Significant proportions had systemic and pulmonary hypertension (60% and 23%), and 32% had ischemic heart disease. All patients, but one, tolerated continuous positive airway pressure, but cost of the equipment led some to prefer surgical treatment that is offered free.

Conclusion: This series shows a bias towards female sex and frequent association with obesity-hypoventilation syndrome. In many cases the diagnosis was not suspected suggesting poor recognition and awareness of sleep apnea syndrome. Monitoring pulse oximetry during sleep was helpful in the diagnosis and titration of continuous positive airway pressure. Ways of providing continuous positive airway pressure under the health system need to be studied.

Keywords: Sleep apnea syndrome, obesity.

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Over the last decade vast knowledge was gained about sleep apnea syndrome (SAS). It is known now that SAS is associated with and can aggravate many medical illnesses such as systemic and pulmonary hypertension, cardiac arrhythmias, ischemic heart disease and stroke, as well as affecting psychosocial well-being.[1] Moreover, increased mortality has been documented in patients with an apnea index of more than 20.[2] Recognition and treatment of SAS is important to prevent these serious complications. While SAS is relatively common in the West affecting 2-4% of middle aged males, little is known about its prevalence in Saudi Arabia. Little also is known about the clinical features in the local population, whether this syndrome is recognized and referred early before complications occur and on patients' acceptance of treatment. Herein, we present the clinical profile and discuss the management issues of patients diagnosed in our center, to promote more awareness about SAS in this region.

Methods. This study was conducted prospectively at King Khalid University Hospital (KKUH), which serves as a general and a tertiary care hospital for the civilian population, and is located in Northwest Riyadh. Referred patients from other parts of the Kingdom are also accepted.

Inclusion criteria. All patients who were diagnosed with SAS at KKUH from 1992 to 1996 were included. Diagnosis of SAS was based on a

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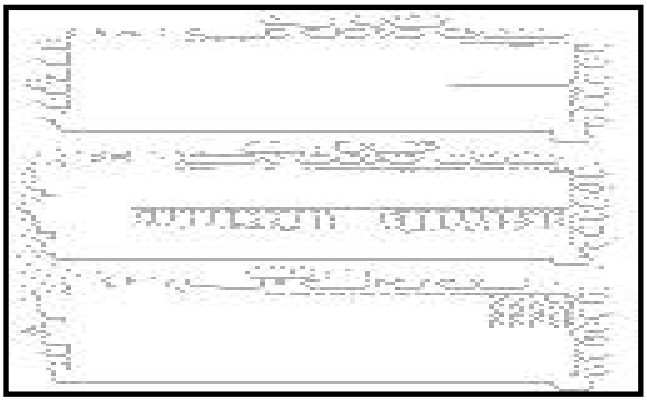

Figure 1 - Pulse oximetry recordings from patients with obstructive sleep apnea syndrome (OSAS) during sleep. (a) The typical deep pattern considered specific for OSAS. (b) Recurrent bouts of desaturation that are abolished. (c) By the application of continuous positive airway pressure (CPAP).

combination of: (1) the typical history, particularly of hypersomnolence, apneas noted by spouse or other relatives during sleep, snoring, interrupted and unrefreshing sleep; (2) typical dips in oxygen measurement (desaturation) during sleep taken by a pulse oximeter with a memory and a recorder (Figure 1), called "deed pattern";[5] which is defined as a drop in oxygen saturation of 4% or more to < 90%, (3) significant improvement in symptoms and total or near total elimination of oxygen desaturation dips recorded during full night sleep with continuous positive airway pressure (CPAP) therapy (as shown in Figure 1). Polysomnography (PSG) was available later for some patients.

Study protocol. We completed data entry for each patient that included age, sex, weight, and height, specialty of the referring doctor and his or her diagnosis, symptoms of the patients including history taken from the spouse, signs, any associated disorder, relevant investigations (including arterial blood gases (ABG), pulse oximetry, pulmonary function tests (PFT), echocardiography, PSG when available, treatment and acceptance by the patients.

Statistical analysis. The two-tailed student T test was determined using the Statpac Gold statistical package. A value < 0.05 was chosen to indicate the significance of the different variables between male and female patients.

Table 1 - Clinical parameters related to gender in patients with sleep apnea syndrome. (Mean values + standard deviation).

Variable	Male		Female		P value
	No.	Value	No.	Value	
Age (years)	28	44.8 ± 13.63	20	56.4 ± 10.1	0.0023*
BMI (kg/m^2)	28	39.0 ± 9.54	20	48.8 ± 12.7	0.0037*
Interval to diagnosis (years)	28	5.57 ± 6.56	19	5.39 ± 5.5	0.9221
PaO_2 (mmHg)	27	68.6 ± 15.4	19	54.4 ± 9.80	0.001*
$PaCO_2$ (mmHg)	27	45.7 ± 10.6	19	56.4 ± 10.4	0.0015*
Nadir sleep O_2 saturation	28	55.0 ± 17.8	19	43.6 ± 15.4	0.0279*
% FVC (of predicted)	24	79.8 ± 17.7	15	57.9 ± 14.6	0.0003*
% FEV_1 (of predicted)	24	80.5 ± 18.7	15	58.0 ± 16.1	0.0005*

*Significant P value < 0.05

Table 2 - Presenting symptoms and signs in patients with sleep apnea syndrome.

Symptom or sign	Patient no.	%
Snoring	48	100
Hypersomnia	48	100
Dyspnea	35	73
Poor quality of sleep	23	48
Impaired concentration	21	44
Chronic cough	19	40
Impotence (In males only)	13	46
Headache	12	25
Fatigue	6	13
Dizziness	4	8
Irritability	2	4
Nocturia	1	2
Papilledema	2	4

Results. A total of 83 patients were evaluated clinically and screened with over-night pulse oximetry during the study period. Of those, 48 patients satisfied the above-mentioned definition of SAS (28 males and 20 females). The mean age was 49.36 years (range 13 to 76). Male patients were significantly younger than female patients (Table 1). The mean body mass index (BMI) was 42.8kg/m^2 (range 25 to 76), and was significantly higher for females (Table 1). The mean interval between onset of symptoms and the diagnosis was 5.57 years (SD ±6.6) for males and 5.39 years for females (SD ±5.4), with no significant difference (P > 0.05, Table 1). Clinical features and associated disorders are shown in Tables 2 and 3. On presentation, the awake arterial blood gas tensions (ABG) showed that 28 patients (58%) had PaO_2< 60 mmHg and 26 (54%) patients had $PaCO_2$> 45 mmHg, while the nadir oxygen saturation during sleep was below 85% in all patients (mean 49%). Spirometry could be performed by 39 patients and showed that the mean forced expiratory volume in one second (FEV_1) was 72% (range 41 to 119) and the mean vital capacity was 72% (range 44 to 108) of the predicted values. Of these, 19 values were suggestive of a restrictive defect, 13 values were normal, 4 values were obstructive defects and 3 values showed mixed

Table 3 - Associated disorders with sleep apnea syndrome.

Disorders	Patient no.	%
Obesity (Body mass index >39 kgm-2)	45	94
Systemic hypertension (Diastolic BP > 90 mmHg)	29	60
Ischemic heart disease (Diagnosed clinically, by EKG ± coronary angiography)	15	31
Pulmonary hypertension (Systolic pulmonary artery pressure> 25 mmHg)	11	23
Hypothyroidism (diagnosed clinically and by thyroid function test)	2	4

defects. Table 1 shows that female patients had more derangement in ABG and spirometric values than males. Polysomnography was carried out for 18 patients and showed the obstructive or predominantly obstructive type of SAS (Apnea-hypopnea index 56.5, range 21-88). The specialty of the referring doctor and the diagnosis at referral are shown in Table 4. In many (28 patients, 58%), the diagnosis of SAS was not suspected. An in-hospital trial of CPAP was effective and was tolerated in all patients except one, but because of the cost of the equipment some patients did not acquire it for home use. Of those, 4 patients opted to go for surgical treatment (uvulopalatopharyngoplasty or UPPP) leading to partial improvement in one, no change in 2 and an unknown result in one patient who did not come for follow up.

Discussion. In this report we describe the characteristics of cases of SAS in a Saudi referral center. In line with Western series, snoring and day somnolence were universal.[1] Male to female ratio were estimated to be 8:1 in sleep disorder clinics, and 3:1 in the general population,[1] while in this series it was 1.4:1. Men tended to be middle aged, while women were older. The decline in hormones was postulated as a cause for the rise in incidence of SAS after menopause.[4] Obesity was present in 94% of our patients, which is higher than the 60-90% reported in previous series.[1,5] Also, there was high prevalence of dyspnea (73%) and chronic cough (40%) as presenting symptoms, and a high percentage had daytime hypoxemia (58%) and hypercapnia (54%). Daytime hypoxemia and hypercarbia are not usual features of SAS. When present it may indicate the presence of obesity-hypoventilation (Pickwickian) syndrome or chronic obstructive lung disease (COPD) co-existing with obstructive sleep apnea (OSA).[6] However, the leading cause of daytime hypoxemia in our series, based clinically, and on the pulmonary function tests, was the obesity-hypoventilation syndrome and not COPD.

Table 4 - Mode and reasons for referral.

Referring doctor	Reason for referral	Patient no.	%
Internist	Unexplained respiratory failure	14	29
	COPD	4	8
Cardiologist	Unexplained symptoms with no cardiac cause	10	21
ENT surgeon	Snoring, rule out SAS	8	17
Neurologist	Hypersomnia, rule out SAS	1	2
Different specialist	Rule out SAS	11	23

COPD: Chronic obstructive pulmonary disease
SAS: Sleep apnea syndrome

The delay between symptoms and diagnosis of SAS was similar in male and female patients (mean values were 5.57 and 5.39 years). This is an interesting point as Western series had shown that the delay in women was 9.7 ± 3.1 years and was significantly longer than in the case of men.[7] However, the average delay was close to ours (approximately 4 years) and the gender difference was not statistically significant when only obese patients were considered.[7] The index of suspicion of SAS tends to be higher in the obese than the non-obese women.[8] The fact that obesity was more prevalent and severe in our female patients probably explains the lack of difference and the more severe derangement in their ABG and spirometry. The delayed diagnosis and referral is probably because of the varied symptomatology and presentations to different specialists (as seen in Table 1). This is more likely to occur when patients present with unusual complaints, such as bed wetting because of nocturia. Poor recognition is still a problem even in Western Countries.[5]

Hypertension and coronary artery diseases are common in patients with sleep disordered-breathing,[9] which increases the cardiovascular morbidity and mortality. In our series 60% had hypertension and 31% had ischemic heart disease. Daytime hypertension is reported in 40% in SAS, while the prevalence of SAS in hypertension is about 20-30%.[10] Sleep apnea syndrome is now considered an independent risk factor for coronary artery disease. Schafer et al,[11] demonstrated that SAS was often associated with ischemic episodes that may lead to central nervous system activation and additional fragmentation of sleep. They concluded that patients with nocturnal ischemia should be screened for underlying sleep apnea. Finally, 23% of our cases had pulmonary hypertension, which is slightly higher than the 15-20% reported in most Western series.[6,12] When higher figures of pulmonary hypertension were reported (up to 42%), these were associated with

daytime hypoxemia.[5,11] The latter association is supported by our findings.

Overnight measurement of oxygen saturation by pulse oximetry was used for screening suspected cases of SAS in our center before the availability of PSG. As shown by Epstein and Dorlac,[5] the deep pattern has a sensitivity of 74% and a specificity of 89%. Values are even higher (>85%) when the BMI is >30 Kg/m^2, such as in our group of patients. Nonetheless, this should not be considered as replacement of PSG, which was shown, by the same authors, to be superior and cost effective. The lack of PSG in many of our patients is the main limitation of this study, although the diagnosis was highly likely based on clinical grounds and the response to CPAP.

Nasal continuous positive airway pressure is regarded the treatment of choice for patients with SAS.[13] While 25% of patients in a previous study in Riyadh,[14] did not accept an in-hospital trial of the CPAP system, it was tolerated by all but one of our patients, including many elderly uneducated patients. This is contrary to the common belief that such patients may not accept this highly technical mode of therapy. Although CPAP is effective, long term compliance is a problem.[15] Meurice et al found that only 68% of patients were compliant after 14 months of starting CPAP treatment.[16] The equipment is not provided free to our patients, so the cost (about 1500 to 3000$ in Saudi Arabia) was a deterrent to obtain CPAP for home use. Some opted for UPPP, which is less effective but offered free by the health system.

In conclusion, OSA in a referral center in Saudi Arabia shows a bias towards female sex, obesity, and a frequent association with Obesity-Hypoventilation Syndrome, and that CPAP therapy was well tolerated. The study is limited by the fact that it represents only one hospital experience, and that the long-term compliance to CPAP was not assessed. However, this preliminary data may serve to raise the awareness of physicians and the public of SAS and its repercussions. Surveys should be carried out to estimate the prevalence of sleep-disordered breathing in the gulf communities.

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