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FREQUENCY OF PULMONARY HYPERTENSION IN PATIENTS WITH OBSTRUCTIVE SLEEP APNEA (OSA).

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ABSTRACT;
Background; Obstructive sleep apnea (OSA) is commonly complicated by left heart disease and is associated with pulmonary hypertension (PH). This study was done to determine the frequency of pulmonary hypertension in OSA to ascertain current magnitude of the problem. **Material and methods:** This cross-sectional study was done at Department of Medicine, Nishtar Hospital Multan. A total of 95 patients with OSA were included in this study. Once registered in the study, all the relevant baseline investigations were done like echocargiography to diagnose Pulmonary Hypertension. History was taken like diabetes, hypertension, smoking and other sociodemographic factors was inquired and statistical analysis was performed by entering all the data in SPSS version 20. **Results;** Of these 95 study cases, 60 (63.25) were male patients and 35 (26.8%) were female patients. Mean age of our study cases was 36.57 ± 6.06 years. Of these 95 study cases, 20 (21.1%) had diabetes and hypertension was noted in 42 (44.2%) of our study cases. Mean height of our study cases was 156.39 ± 12.92 centimeters while mean weight of our study cases was 74.59 ± 17.27 kilograms. Mean body mass index (BMI) of our study cases was 27.33 ± 4.07 kg/m^2 and obesity was present in 40 (42.1%) of our study cases. Mean disease duration was 26.32 ± 10.97 months and history of smoking was present in 21 (22.1%). Pulmonary hypertension was present in 43 (45.3%) of our study cases. **Conclusion;** Very high frequency of pulmonary hypertension was observed in our study among patients with obstructive sleep apnea. Pulmonary hypertension was significantly associated with diabetes, disease duration, smoking and obesity. All clinicians treating such patients should investigate for pulmonary hypertension for early diagnosis and management which will decrease morbidity and improve quality of life of these patients.
Keywords; Obstructive sleep apnea, Pulmonary hypertension, Arterial hypertension.

INTRODUCTION:

Obstructive sleep apnea (OSA) is a form of sleep-disordered breathing that is increasingly prevalent, in part due to the increasing rate of obesity and enhanced public awareness. An estimated 40 million Americans experience OSA, with a 3%–7% prevalence among men and a 2%–5% prevalence among women. OSA is an independent risk factor for systemic hypertension, stroke, myocardial ischemia, congestive heart failure, arrhythmias, and sudden cardiac death [1]. Pulmonary Hypertension (PH) is present in 12%–34% of patients with OSA. However, the role of OSA in the development of PH and its mechanisms, independent of left ventricular failure, are not entirely clear. Patients with PH present with symptoms of dyspnea on exertion, fatigue, lightheadedness, chest pain, syncope, palpitations, and/or lower extremity edema. Obstructive sleep apnea (OSA) is commonly complicated by left heart disease and is associated with PH [2, 3]. Pulmonary hypertension (PH) is a hemodynamic and pathophysiological state that can be found in multiple clinical conditions [4]. Pulmonary hypertension (PH) is the major cardiac complication of the disease; it is defined by a mean pulmonary artery pressure at rest (mPAP) above 25 mmHg, as assessed by right heart catheterization [5, 6]. Echocardiography is the first screening method for PH and it should be widely used, as it can also appreciate the cardiac consequences of PH, especially on the right ventricle [7]. In current practice the detection of PH is made mostly by echocardiography, as right heart catheterization, the "gold standard" diagnostic method for PH, is an invasive and expensive procedure, considered only in patients referred to surgical procedures. The development of pulmonary hypertension is a poor prognostic sign in patients with obstructive sleep apnea (OSA) and affects both mortality and quality of life. Although pulmonary hypertension in OSA is traditionally viewed as a result of apneas and intermittent hypoxia during sleep, recent studies indicate that neither of these factors correlates very well with pulmonary artery pressure. Human data show that pulmonary hypertension in the setting of OSA is, in large part, due to left heart dysfunction with either preserved or diminished ejection fraction. Longstanding increased left heart filling pressures eventually lead to pulmonary venous hypertension. The combination of hypoxic pulmonary vasoconstriction and pulmonary venous hypertension with abnormal production of mediators will result in vascular cell proliferation and aberrant vascular remodeling leading to pulmonary hypertension. These changes are in many ways similar to those seen in other forms of pulmonary hypertension and suggest shared mechanisms. The majority of patients with OSA do not receive a diagnosis and are undertreated. Appreciating the high prevalence and understanding the mechanisms of pulmonary hypertension in OSA would lead to better recognition and management of the condition [8]. Abou Shehata from Egypt [9] has documented 44.4 % pulmonary hypertension in patients with OSA.

MATERIAL AND METHODS:

Consecutive 95 patients with OSA were recruited in this study from Department of Medicine, Nishtar Hospital, Multan, Pakistan. Known cases with left heart failure, asthma, pulmonary embolus, lung cancer and COPD, patients with ARF and CRF and pregnant ladies were excluded from our study. All the relevant baseline investigations were done like echocargiography to diagnose Pulmonary Hypertension. Apnea: Cessation of airflow ≥10-sec as measured by nasal pressure in presence of thoracic efforts in overnight polysomnography. Hypopnea: 50% reduction in airflow for 10 seconds or a discernable change in airflow with either a ≥3% oxyhemoglobin desaturation or an arousal in overnight polysomnography. Apnea / Hypopnea index [AHI]: Number of apneas and hypopneas occurring per hour during sleep calculated by overnight polysomnography. Obstructive Sleep Apnea [OSA]: AHI of more than 5 events per hour. Pulmonary hypertension: It was defined as positive when both of the following conditions are present on echocardiography;

1. Pulmonary artery systolic pressure (PASP) >35 mm Hg.
2. *Tricuspid regurgitant velocity (*TRV) > 3.4

Statistical analysis was performed by entering all the data in SPSS version 20.

RESULTS;

Our study comprised of a total of 95 study cases with Obstructive sleep apnea who met inclusion criteria of our study. Of these 95 study cases, 60 (63.25) were male patients and 35 (26.8%) were female patients. Mean age of our study cases was 36.57 ± 6.06 years (with minimum age of our study cases was 31 years while maximum age was 52 years). Mean age of the male patients was 37.67 ± 6.67 years while that of female patients was 34.69 ± 4.29 years (p=0.020). Our study results have indicated that majority of our study

cases i.e. 83 (87.4%) were aged less than 45 years. Of these 95 study cases, 40 (42.1%) belonged to rural areas while 55 (57.9%) from urban areas, 39 (41.1%) had monthly family income up to Rs. 30000 and 56 (58.9%) had monthly family income more than 30000 rupees. Of these 95 study cases, 20 (21.1%) had diabetes and hypertension was noted in 42 (44.2%) of our study cases. Mean height of our study cases was 156.39 ± 12.92 centimeters while mean weight of our study cases was 74.59 ± 17.27 kilograms. Mean body mass index (BMI) of our study cases was 27.33 ± 4.07 kg/m^2 and obesity was present in 40 (42.1%) of our study cases. Mean disease duration was 26.32 ± 10.97 months and history of smoking was present in 21 (22.1%). Pulmonary hypertension was present in 43 (45.3%) of our study cases

DISCUSSION;

The development of pulmonary hypertension is a poor prognostic sign in patients with obstructive sleep apnea (OSA) and affects both mortality and quality of life [10, 11]. Although pulmonary hypertension in OSA is traditionally viewed as a result of apneas and intermittent hypoxia during sleep, recent studies indicate that neither of these factors correlates very well with pulmonary artery pressure [12-14]. Our study comprised of a total of 95 study cases with Obstructive sleep apnea who met inclusion criteria of our study. Of these 95 study cases, 60 (63.25) were male patients and 35 (26.8%) were female patients. A study conducted by Haqqee et al [15] from Karachi also reported 66% male gender predominance in patients with obstructive sleep apnea which is close to our study results. Hussain et al [16] from Karachi also reported 87 % male gender preponderance which is in compliance with our study results. A study conducted by Ullah et al [17] from Rawalpindi also reported 58 % male gender predominance which is close to our study results. A study conducted by Lee et al [18] in Korea also reported 86 % male gender predominance which is in compliance with our study results.

Mean age of our study cases was 36.57 ± 6.06 years (with minimum age of our study cases was 31 years while maximum age was 52 years). Mean age of the male patients was 37.67 ± 6.67 years while that of female patients was 34.69 ± 4.29 years (p=0.020). Our study results have indicated that majority of our study cases i.e. 83 (87.4%) were aged less than 45 years. Hussain et al [16] from Karachi also reported 49.71 ± 9.82 years mean age of the patients with OSA which is similar to our study results. A study conducted by Ullah et al [17] from Rawalpindi also reported 53 ± 12.1 years mean age of the OSA patients which is higher than that our study results. The reason for this difference is due to our methodology as we included only patients with ages ranging from 30 – 60 years. A study conducted by Lee et al [18] in Korea also reported 48.9 ± 11.7 years mean age of the OSA patients which is close to our study results.

Of these 95 study cases, 40 (42.1%) belonged to rural areas while 55 (57.9%) from urban areas, 39 (41.1%) had monthly family income up to Rs. 30000 and 56 (58.9%) had monthly family income more than 30000 rupees. Of these 95 study cases, 20 (21.1%) had diabetes and hypertension was noted in 42 (44.2%) of our study cases. A study conducted by Haqqee et al [15] from Karachi also reported 35 % hypertension in patients with Obstructive sleep apnea (OSA) which is close to our study results. Hussain et al [16] from Karachi also reported 60 % hypertension and 23 % diabetes in OSA which is in compliance with our study results.

Mean height of our study cases was 156.39 ± 12.92 centimeters while mean weight of our study cases was 74.59 ± 17.27 kilograms. Mean body mass index (BMI) of our study cases was 27.33 ± 4.07 kg/m^2 and obesity was present in 40 (42.1%) of our study cases. A study conducted by Haqqee et al [15] from Karachi also reported similar results. Hussain et al [16] from Karachi also reported 32.32 ± 7.53 kg/m^2 mean BMI which is in compliance with our study results. A study conducted by Lee et al [18] in Korea also reported 26 ±3.4 kg/m^2 which is close to our study results. Obesity is considered a major risk factor for the development and progression of OSA. The prevalence of OSA in obese or severely obese patients is nearly twice that of normal-weight adults. Furthermore, patients with mild OSA who gain 10% of their baseline weight are at a sixfold-increased risk of progression of OSA, and an equivalent weight loss can result in a more than 20% improvement in OSA severity. Moreover, the higher prevalence of OSA in obese subjects is not limited to adults; recent data show that obese children have a 46% prevalence of OSA when compared with children seen in a general pediatric clinic (33%). This finding is further aggravated by the obesity epidemic among children and adolescents. In fact, there are data suggesting that children and adolescents with OSA have more than a sixfold-increased risk of having metabolic syndrome, when compared with children and adolescents without OSA. These findings highlight the need to develop screening and prevention for these conditions, even as early as in childhood.

Mean disease duration was 26.32 ± 10.97 months and history of smoking was present in 21 (22.1%). Hussain et al [16] from Karachi also reported smoking in 53 % patients which is quite higher than our study results.

Cigarette smoking and alcohol have been suggested as possible risk factors for obstructive sleep apnea. Epidemiologic investigations show that current smoking is associated with a higher prevalence of snoring and obstructive sleep apnea. Even exposure to second-hand smoke has been independently linked with habitual snoring. Because former smokers do not manifest the increased risk for obstructive sleep apnea, airway inflammation and damage due to cigarette smoke could alter the mechanical and neural properties of upper airway and increase its collapsibility during sleep.

Pulmonary hypertension was present in 43 (45.3%) of our study cases. Abou Shehata from Egypt [9] has documented 44.4 % pulmonary hypertension in patients with OSA which is close to our study results. Another study done in USA by Minai et al [19] reported 70 % pulmonary hypertension in OSA patients indicating its high prevalence in these patients.

CONCLUSION;

Very high frequency of pulmonary hypertension was observed in our study among patients with obstructive sleep apnea. Pulmonary hypertension was significantly associated with diabetes, disease duration, smoking and obesity. All clinicians treating such patients should investigate for pulmonary hypertension for early diagnosis and management which will decrease morbidity and improve quality of life of these patients.

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