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Original Article

A Comparative Study on Sleep SpO_2 between Normal and Early Emphysematous Patients

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Abstract

Background: Emphysema is one of the disastrous maladies across the globe. The principal causes of this disease are air pollution, toxic gas inhalation, and habitual smoking. Early detection of this condition which principally affects the middle-aged persons might halt the progression of this disease. Hence, this simple study was undertaken to evaluate the practicability of this novel method.

Materials and Methods: Using a pulse oximeter, SpO_2 was determined in 30 early emphysematous patients of both sexes. They were diagnosed clinically by a competent chest physician and also correlated with computerized pulmonary function tests and chest computed tomography scans. The test (pulse oximetry) was done when the person was sleeping. This was compared with age-matched normal healthy persons whose pulse oximetry was also done during sleep.

Result and Discussion: Study showed a significant difference in SpO_2 percentage during sleep between normal and early emphysematous persons ($P < 0.05$). Observation showed that this was more intense during rapid eye movement stage of sleep.

Conclusion: Since sleep hypoxemia as reflected by simple SpO_2 determination is more pronounced in emphysematous patients, it can be used as a simple test for susceptible persons for early detection of emphysema and taking early preventive measures.

Key words: Emphysema, Sleep hypoxemia, SpO_2

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is now regarded as a global malady of grave concern because of its persistently increasing morbidity and mortality.[1] In 2000 alone more than 2.5 million deaths occurred globally because of COPD.[2] Furthermore, at the present moment it ranks as the third leading cause of death in the United States.[3] This disease is supposed to be the fifth leading disease burden worldwide by the year 2020.[4] COPD basically encompasses two distinct clinicopathological entities, viz., emphysema and chronic bronchitis.[1] A progressive, persistent airflow obstruction as revealed by progressively increasing forced expiratory volume 1 (FEV_1) and FEV_1/forced vital capacity ratio, is the hallmark of COPD.[1]

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Although a minor reversible component of airflow obstruction is noted occasionally, but for the most part the airflow obstruction is irreversible in COPD.[5,6] The disease "emphysema" is a pathological diagnosis and is defined as "a state of the lungs in which there is gross alveolar wall destruction with irreversible enlargement of the air spaces distal to the terminal bronchioles and without any evidence of fibrosis."[7,8] In COPD airflow obstruction is almost always associated with an abnormal inflammatory response of the lungs to gases or noxious inhaled particles.[9] Pathologically speaking, the hallmark of emphysema is the breakdown of yellow elastic tissue of the lungs which is mainly made of the protein "elastin." Loss of elastin leads to loss of integrity of the alveolar wall. The initial trigger is an insult with noxious particles and gases of which cigarette smoke is the most common factor. However, this happens in an accelerated way in the susceptible persons, that is, those who are genetically predisposed to this

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disease. It has long been observed that a severe deficiency of the enzyme α_1-antitrypsin leads to a preponderance of panacinar emphysema. Normally, a full blown emphysema occurs in the sixth or seventh decade of life, and that also mostly in smoking (in 90% of case). However, in persons with severe deficiency of α_1-antitrypsin, emphysema sets by fourth or fifth decade of life. Both a loss of protease-antiprotease balance and oxidative stress are implicated in the etiology of pulmonary emphysema.

It has long been postulated that in the pathophysiology of emphysema, the initial insult begins with the destruction of alveolar walls. However, recently McDonough *et al.* have challenged this concept, giving a new suggestion that the narrowing and disappearance of the terminal bronchioles precede alveolar destruction and lead to the latter condition, thus giving rise to the occurrence of centrilobular and panlobular emphysema.[10] In their study, 78 patients were investigated with microcomputer tomography to track their alveoli and terminal bronchiolar walls phase wise in different stages of the disease.

During sleep, even in a normal person, there is a decrease in ventilation, tidal volume and chemo-responsiveness to blood CO_2. However, this does not result in hypoxemia, because the drop in PaO_2 occurs on the flat potion of the oxyhemoglobin dissociation curve. However, in emphysema, oxygenation during wakefulness may already be on the steep portion of the oxyhemoglobin dissociation curve, leading to hypoxemia during sleep as tidal volume falls. The most pronounced hypoxemia occurs during the rapid eye movement (REM) stage of sleep because of the generalized muscle hypotonia that accompanies this stage.

Having thought of this affair, the authors decided to measure SpO_2 during the REM stage of sleep in normal and emphysematous patients using a pulse oximeter which is so simple a device that even a nurse can apply it correctly on patients.

MATERIALS AND METHODS

Study Design

It was an institution-based, observational and cross-sectional study.

Study Setting

The study was done in the indoor of the General Medicine and Chest Medicine Wards, in MGM Medical College and LSK Hospital, Kishanganj, Bihar, India.

Time Line

The study was done between the periods January 01, 2016 and December 31, 2016.

Study Population

The study was done in normal adult healthy subjects of both sexes (as control) and on patients with proven diagnosis of emphysema but without any other obvious illness. The age limit was 40-60 years.

Methodology

After taking permission from the Heads of the Departments of Medicine and Chest Medicine, the Principal, the Director and the Chairman of the Ethical Committee, the study was formally commenced. Formal written consents of all subjects were also taken after explaining the procedures and the purpose of the study.

For each subject through clinical examination was done followed by routine blood testing to exclude any unwanted disease and also plasma α_1-antitrypsin was estimated. Then, digital spirometry was performed in all controls and patients as also routine chest X-ray (posterior-anterior), electrocardiogram and thoracic computed tomography scan. Other relevant tests were done as and when necessary.

In confirmed early emphysematous patients and in controls, sleeping SpO_2 especially during REM stage of sleep was performed by a standard pulse oximeter. The whole data obtained were then analyzed and compared.

RESULTS

The results obtained are given in Table 1 and Figure 1.

DISCUSSION

Our studies show that as expressed in Table 1 and Figure 1, the emphysematous population has a significantly lower sleep time SpO_2 as compared to that in normal healthy population.

Sleep time hypoxemia is observed in several pulmonary and other disorders. Palma *et al.*[11] observed in a 2008 publication that there is significant sleep time hypoxemia in hepatopulmonary syndrome (HPS). Furthermore, observations in patients with primary pulmonary

Table 1: The mean±SD of percentage SpO_2 (sleep time) in normal and emphysematous populations

Population	SpO_2 in percentage (mean±SD)	*t* value	*P* value
Control	98.23±0.94	9.83	0.01
Early emphysematous patients	88.17±3.89		

SD: Standard deviation

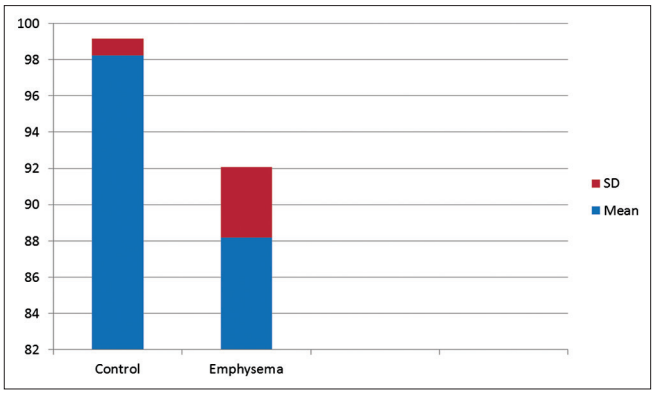

Figure 1: Column diagram showing the comparison of mean sleep time SpO_2 between normal and emphysematous populations

hypertension[12] and in a recent study with a small host of non-HPS patients of cirrhosis support that oxygen saturation may be influenced during sleep.[13]

Sleep produces several alterations in respiratory mechanics and gas exchange, including breathing pattern instability, hypoventilation, upper airway obstruction, ventilation-perfusion mismatch, and decreased hypoxic and hypercapnic ventilatory responses.[14]

The notion that pulse oximetry in chronic obstructive emphysema can prove a significant lowering of oxygen saturation during sleep was first suggested 55 years ago.[15] This was further substantiated by early nocturnal polygraphic studies on emphysema patients and still later by polysomnographic studies. Other studies included intermittent blood gas tension measurements during sleep. All these started after the advent of reliable transcutaneous pulse oximeter development. Several studies were performed to show that COPD patients did experience a worsening of hypoxemia, particularly during REM sleep.[16,17]

The initial studies focused almost entirely on patients with severe cases who were clearly hypoxemic even during day time. In our study, we have highlighted effect of REM sleep in early emphysema, though similar studies began earlier where data appeared for nocturnal hypoxemia in COPD patients with little or no hypoxemia during day time (PaO_2 >80 mm Hg).[18]

Douglas *et al.* studied transient hypoxemia during sleep in chronic bronchitis and emphysema. In their study, arterial oxygenation, breathing pattern, and electroencephalogram were investigated during sleep in patients with chronic bronchitis and emphysema as also in healthy subjects for comparison. All patients with "blue bloaters syndrome" had episode of sleep time transient hypoxemia lasting 1-100 min, during which time their oxygen saturation reduced more than 10% than day time resting stage. On the contrary, patients with "pink puffer syndrome" did not show such hypoxemia nor did the healthy subjects. Hypoxemia episode in their studies also occurred mainly during the REM stage of sleep. It was suggested by these workers that the cause of these hypoxemia episodes resides in a combination of hypoventilation and impaired ventilation/perfusion (V/Q) ratio. It was also suggested that these phenomena might also lead to pulmonary hypertension and secondary polycythemia.[19]

The key drivers of hypoxemia are V/Q mismatch, respiratory muscular hypotonia, exercise, sleep, and chronic emphysema. Since, along with exercise, sleep is also an exaggerating factor, concomitant sleep disorder may place a small but significant number of COPD patients in a further increased risk of pulmonary as well as secondary cardio-vascular complications.[20]

CONCLUSION

Sleep hypoxemia as reflected by the simple test "pulse oximetry" with the determination of sleep time SpO_2, is found to be quite significant and more pronounced in patients with emphysema as compared to normal subjects. This phenomenon is noticeable even in early emphysematous patients, and therefore this test can be applied for early detection of emphysematous patients. However, further studies on a massive scale might be envisaged to come to a more conclusive evidence.

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