CASE REPORT

Obstructive Sleep Apnea in a patient of Idiopathic Pulmonary Fibrosis (IPF)

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Indian J Sleep Med 2007; 2.1, 32-34

Keywords: Obstructive sleep apnea, Idiopathic pulmonary fibrosis

Introduction

Obstructive sleep apnea (OSA) syndrome is said to be present when apnea hypopnoea index is greater than 15 events per hour. Association of obstructive sleep apnea with idiopathic pulmonary fibrosis is not so common. Here we report such a case in a 65 year old female.

Case report

A 62 years old female non smoker was admitted to our department with the complaints of dry cough, breathlessness lasting for one and half years. The resting pulse rate was 102/min and blood pressure was 112/74 mm Hg and her respiratory rate was 34/min. Her general examination revealed obesity with weight of 94 kg (Body Mass Index 32), clubbing and cyanosis. On chest examination there was bilateral inspiratory 'Velcro' type crepts. Her chest x-ray revealed volume reduction and pulmonary fibrosis (Fig-1). Her High Resolution Computerized Tomography thorax revealed reticulonodular shadows, ground glass opacity, honey combing, interstitial thickening, affecting predominantly basal parts in both lungs, suggestive of idiopathic pulmonary fibrosis (Fig-2). Spirometry showed FEV1/FVC to be 86 % of predicted values and reduced FVC and FEV1 suggestive of restrictive pattern.

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Her haemoglobin was 13.3 gm %; total leucocyte count 11,200/cmm: neutrophils 81%, lymphocytes 18% and monocytes 1 %. Her serum protein was 6.9 gm/dl. Smear was negative for acid fast bacilli. Her serum ACE, serum rheumatoid factor, C - reactive protein and ANA (anti nuclear antibody) titre were within normal limit. Arterial

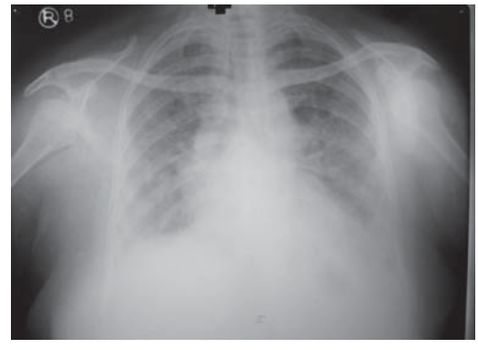

Fig 1: Chest x-ray revealed volume reduction and pulmonary fibrosis

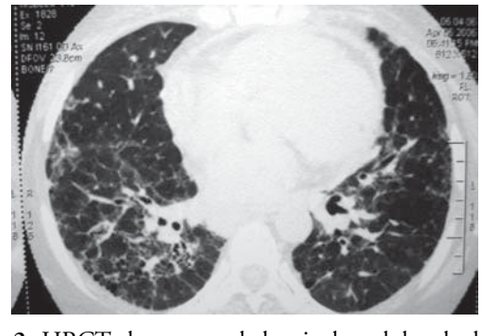

Fig 2: HRCT thorax revealed reticulonodular shadows, ground glass opacity, honey combing, interstitial thickening, affecting predominantly basal parts in both lungs, suggestive of idiopathic pulmonary fibrosis

Blood Gas revealed pH; 7.34 PO_2; 38 mm Hg, PCO_2; 25.4 mm Hg, O_2 saturation; 68 %(without oxygen).

During hospital stay her attendants also gave history of loud snoring, nocturnal awakenings, morning headache and day time sleepiness. So to rule out obstructive sleep apnea her polysomnography was done. Her polysomnography revealed apnea hypopnoea index of 29.4 /hours. Thus diagnosis of idiopathic pulmonary fibrosis with obstructive sleep apnea was made. She was put on oxygen 2 litre/ min, nasal C-PAP at setting of 6mm of H_2O (5 hours/night) and 50 mg of prednisolone per day. Her daily assessment of ABG revealed dramatic clinical improvements (shown in table-1)

Table1: Arterial blood gas & pulmonary artery pressure parameters before and after CPAP therapy

S. No.	ABG parameter	Prior to CPAP	On CPAP (Show night)				
			Day-1	Day-2	Day-3	Day-4	Day-5
1	PH	7.34	7.44	7.43	7.41	7.40	7.36
2	PO2 (mmHg)	38	43.4	47.4	68.3	66.8	71.6
3	PCO2 (mmHg)	25.4	35.1	31.6	28.8	31	35
4	O2 saturation	79%	87%	94.9%	93.3%	96%	98.7%
5	Pulmonary Hypertension	63 mm Hg					42 mm Hg

Discussion

Pulmonary fibrosis, literally means abnormal formation of fibre like scar tissue in lung. The histopathological classification of idiopathic interstitial pneumonias (IIPs) has evolved over time, most recently codified in American Thoracic Society/ European Thoracic Society (ATS/ERS) 2002 consensus classification statement[1]. This classification separates the idiopathic interstitial pneumonias into seven clinico-pathological entities(in order of their relative frequency) : idiopathic pulmonary fibrosis (47-64%) : non specific interstitial pneumonia (14-36 %): respiratory bronchiolotis interstitial lung disease(10-17%) :desquamated interstitial pneumonia: cryptogenic organizing pneumonia (4-12%) : acute interstitial pneumonia(<2 %) : lymphocytic interstitial pneumonia(<2 %). Idiopathic pulmonary fibrosis is the most common interstitial lung disease of unknown etiology. The incidence is estimated 10.7 cases per 100,000 per year for males and 7.4 cases per 100,000 per year for females[2-3]. Approximately two-thirds of patients of IPF are over the age of 60 years at the time of presentations, while present case was 52 years old. Most patients with IPF have gradual onset of dyspnoea (due to hypoxaemia) and non productive cough. On examination bibasilar fine inspiratory crepitations (so called Velcro crackles) are most characteristic. Digital clubbing is seen in 25-50 % of patients (as in present case). High-resolution computed tomography (HRCT) is significantly more sensitive and specific for the diagnosis of IPF[4].

Hypoxaemia might be a critical factor in the pathogenesis of sleep related respiratory disorder, due to hypoxia induced brain stem depression[5]. Among the sleep related disorders obstructive sleep apnea is very common. The obstructive sleep apnea (OSA) syndrome is said to be present when apnea hypopnoea index is greater than 15 events per hour[6] (in the present case it was 29.4/hour). The pathogenesis of OSA includes both anatomic (narrowing of airways or bony abnormality such as retrognathism or micrognathia) and a neurological components[7]. OSA is more common among male than females (M: F 2:1). OSA affects 2-6 % of middle ages males and 1-2 % of middle age females.

Risk factors for OSA are obesity(as in present case), neck size (collar size >17 inches in males and 15 inches in females), in present case it was 16.1 inches, tonsillar hypertrophy, deviated nasal septum, retrognathia or micrognathia, specific genetic disorder (e.g. Treacher Collins, Downs Syndrome, Alpert syndrome), genetic predisposition, endocrine disorder(hypothyroidism, acromegaly), alcohol, sedatives or hypnotics[8].

Clinical features of OSA are loud or habitual snoring (as in present case), witnessed apnea, nocturnal awakenings, gasping or choking episodes , nocturia, unrefreshing sleep, morning headache, excessive day time sleepiness, automobile/car accidents, irritability, memory loss, personality changes, decreased libido.

Diagnosis of OSA is made by polysomnography-i.e. sleep study. A diagnostic polysomnography study entails a whole night of recording during sleep. Patients found to have sleep apnea return on subsequent night for second sleep study, during which the level of CPAP necessary to abolish sleep related breathing events is determined. The other type of sleep study is split night polysomnography. The scientific rationale for split night polysomnography is that AHI in the first half of night is highly indicative of OSAS. Split night polysomnography is effective in 78 % of patients with OSA[9].

At present the treatment of choice for the patients with OSA is nasal CPAP[10]. It reduces the number of apneic and hypoxic episodes during sleep and reduces day time

sleepiness and improves neuropsychiatric function. The optimal pressure of 5 to 20 cm H_2O is needed to abolish the apneic episodes, snoring and oxyhaemoglobin saturations in all postions and during REM sleep[11]. Philips et al shown that breathing 3 litre per minute of oxygen produced significant improvements in sleep related disorders without NIPPV[12]. While a study from Mumbai has shown that nocturnal oxygen therapy has a beneficial effect on the OSA and ILD[13].

This case report highlights a rare combination of interstitial lung disease and Obstructive sleep apnea. If an idiopathic pulmonary fibrosis (IPF) patient is obese and has history of snoring then obstructive sleep apnea (OSA) must be ruled out.

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