

Acute Respiratory Distress Syndrome (ARDS) Related to SmokingErhan Önalın¹, Nevzat Gözel², Faruk Kılınç³, Fatih Demircan⁴, Ramazan Ulu⁵, Yusuf Şener⁶, Emir Dönder²¹Ercis State Hospital, Department of Internal Medicine, Van, Turkey²Firat Medical Faculty, Department of Internal Medicine, Elazığ, Turkey³Dicle Medical Faculty, Department of Endocrinology, Diyarbakır, Turkey⁴Etic Life Medical Center, Department of Internal Medicine, İstanbul, Turkey⁵Firat Medical Faculty, Department of Nephrology, Elazığ, Turkey⁶Firat Medical Faculty, Department of Endocrinology, Elazığ, Turkey***Corresponding author**

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Abstract: Advanced age and smoking are the most important etiological causes in determining morbidity and mortality in acute respiratory distress syndrome (ARDS). In this case, a 83-year-old male patient developed ARDS with previous smoking history. The patient referred to the emergency service with a complaint of non-productive cough, progressive respiratory failure and orthopnea after smoking, and then a PA chest radiograph revealed two-sided common infiltration. Considering the likelihood of ARDS, biphasic positive airway pressure (BIPAP) was applied to the patient without any response, and therefore the patient was intubated and connected to mechanical ventilator. After improving ventilator parameters, the patient was taken from ventilator and discharged from the hospital with a good general medical condition. We hereby stress out in this case presentation that people elder than 65 years old with a history of smoking are likely to develop ARDS.

Keywords: Smoking, ARDS, BIPAP, Mechanical ventilation.

INTRODUCTION

Cigarette smoke includes more than 4,000 substances, some of which are pharmacologically active, antigenic, cytotoxic, mutagenic and carcinogenic (Table 1). 92 – 95 % of the main flow of smoke is in the

form of gas with a particle amount between 0.3 and 3.3 billion per 1 mL. The average particle diameter is between 0.2 and 0.5 mm, suggesting the ability of being inhaled [1].

Table-1: Some of substances in cigarette smoke [2]

Particle phase	Main effect	Gas phase	Main effect
Tar	Mutagenic/carcinogenic	Carbon monoxide	Adverse effect on oxygen binding ability to hemoglobin
Nicotine	Dose-based stimulant or depressor on parasympathetic N-cholinergic receptors	Nitrogen oxides	Irritant, pro-inflammatory, ciliotoxic
Aromatic hydrocarbons	Mutagenic/carcinogenic	Aldehydes	Irritant, pro-inflammatory, ciliotoxic
Phenols	Irritant, mutagenic/carcinogenic	Hydrocyanic acid	Irritant, pro-inflammatory, ciliotoxic
Cresol	Irritant, mutagenic/carcinogenic	Acrolein	Irritant, pro-inflammatory, ciliotoxic
Benzo(a)pyrene	Mutagenic/carcinogenic	Nitrosamines	Mutagenic/carcinogenic

Smoking has adverse effects on central and peripheral airways in lungs, alveoli, and capillary and lung immune system. In addition, many studies showed a gradual transformation of pseudo stratified cilia epithelial to squamous metaplasia, carcinoma in situ and invasive bronchogenic carcinoma. In smokers,

several respiratory disorders were found. In general, FEV1 values were lower and the decrease rate of FEV1 is more rapid in smokers as compared to non-smokers. Both of these effects suggest a dose-response relation with women being more dramatically than men. The presence of these findings in a middle-aged person is

considered as the most important feature of severe COPD risk. Respiratory complaints increased remarkably in among smokers. There exist a dose-response relation among chronic cough, phlegm, wheezing and dyspnea. The changes responsible to these symptoms include cilia loss in airway epithelium, mucous gland hypertrophy, increased number of goblet cells and increased permeability. Smoking is the main risk factor for COPD; however it still remains unclear by which mechanism it leads to COPD. It is suggested to have led to proteolytic and anti-proteolytic imbalances, bronchial hypersensitivity as well as to inflammatory effects. About 10 to 15 % of smokers manifest a remarkable airway obstruction clinically. Besides, exposure to dust and smoke along with respiratory tract infections at childhood could be other risk factors. Consequently, the mortality due to COPD, pneumonia and influenza is remarkably more common among smokers [1].

The present paper reports the clinical and radiological findings of a patient with ARDS who applied to the emergency service due to respiratory distress and hospitalized to general intensive care unit with diagnosis of ARDS, followed by receiving biphasic positive airway pressure (BIPAP) and then mechanical ventilation (MV) therapy.

CASE PRESENTATION

A male patient of 83 years old with the history of smoking more than 50 years applied to the emergency service with complains of progressive respiratory failure, nonproductive cough and agitation. Under the oxygen mask of 6 lt/min, the analysis from the first arterial blood gas resulted in the following values as 7.06 pH, pO₂: 32 mmHg, pCO₂:40.6 mmHg, HCO₃: 11.3 mmol/L and BE:8mmol/L. Taking the PA chest radiograph into account, the patient was pre-diagnosed with ARDS (Image 1). During follow-up, the patient was hospitalized to intensive care service due to deteriorated overall condition considering any possible need of mechanical ventilation.

During physical examination, the patient's performance status was poor and showed a tendency to sleeping with a respiratory rate of 38/minute, blood pressure of 100/60 mmHg, pulse of 110/minute and body temperature of 36.9 °C. During auscultation, common thin rales were heard at the lower sides of both lungs. Some biochemical parameters were high including urea: 82mg/dl (n:17-43), creatine:1,9 mg/dl (n:0,81-1,44), troponin:0,22 IU/L (n:0,001-0,004), CPK:112 IU/L (n:0-49), and C-reactive protein:10,8 mg/dl (n:0-5). No growth was found in urea and blood cultures. However, there was a common infiltration at both lungs when examined the PA chest radiograph (Image 1).

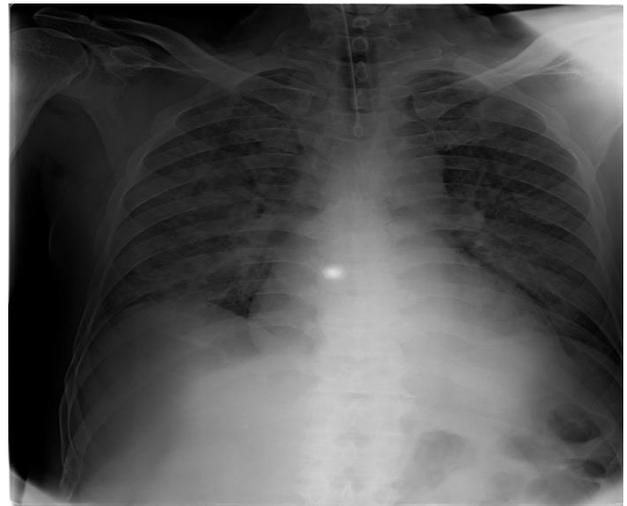


Fig-1: Radiograph of PA chest

Sinus tachycardia was detected in electrocardiogram (ECG) from the patient. From the arterial blood gas obtained during emergency, the results were as follows: pH= 7.06, PaO₂= 32 mmHg, PaCO₂= 40.6 mmHg, SaO₂= 68% (during the introduction of oxygen nasally at 6 L/minute) and HCO₃= 11.3 mEq/L. The patient was diagnosed with ARDS due to the presence of two-sided common infiltration in chest radiography, lower value of PaO₂/FiO₂ under 200, and the absence of any loading findings for left heart failure in portable echocardiograph (EKO) and ECG.

At first, the patient received a BIPAP treatment on the pressure levels of IPAP 15 cmH₂O/EPAP 5 cmH₂O as non-invasive mechanical ventilation. However, as the patient was quite agitated and tachypneic, tended to sleeping, and unable to normalize the levels of blood gases, he was intubated and connected to mechanical ventilation. The treatment procedure was such that after 120 mg methylprednisolone, the patient was administered 1 x 60 mg intravenous methylprednisolone, ceftriaxone 2 x 1 g and ciprofloxacin 2 x 500 mg (intravenous) as antibiotic therapy, budesonid 200 mcg 4 x 1 and ipratropiumbromid 0,5 mg + salbutamol sulphate 2,5 mg 4 x 1 (inhaler) and salbutamol 100 mcg 4 x 2 (inhaler) as bronchodilator therapy. Due to agitation, a midazolamine fusion of 0.04 mg/kg was started and the patient was sedated. The patient started to receive therapy in such a manner that the initial settings of MV was in volume-controlled "Assist Control (A/C)" mode with respiratory rate of 12 / minute, positive end-expiratory pressure (PEEP) of 5 cm H₂O, tidal volume of 6 mL/kg, 100 % of O₂, I:E ratio of 1:3 and in flow triggered. The patient showed an increased oxygen saturation with an excess of 90 %. The oxygen amount was gradually decreased to 50 % so as to keep the saturation rate over 90 % in the patient under paralytic treatment. The paralytic treatment was ended after an observed improvement in radiological, blood gas and

vital findings. The patient was disconnected from MV at the 7th day. After clinical follow-up for 1 week, he discharged from the hospital upon clinical and radiological improvement in his condition. During the outpatient controls after one week, physical examination and radiological findings were completely observed in normal state.

DISCUSSION

ARDS generally appears under the presence of risk factors including aspiration, sepsis or multiple blood transfusion. In chest radiographs, common alveolar infiltrates are observed due to pulmonary edema and atelectasia. Table 2 summarizes the criteria for diagnosis of ARDS [2].

Table 2- The definitions of ALI and ARDS

Acute respiratory distress
Hypoxemia; PaO ₂ / FiO ₂ < 300 mmHg for ALI, PaO ₂ / FiO ₂ < 200 mmHg for ARDS
Bilateral consolidation at Standard chest radiograph
Clinical findings of left ventricular failure or PCWP < 18 mmHg

ALI = acute lung injury; **ARDS** = acute respiratory distress syndrome; **PCWP**= pulmonary capillary pressure;

Any change in gas for ARDS reflects the ratio of PaO₂/FiO₂, being associated with the patient's prognosis. NIMV can be applied to hemodynamically stable patients during early periods [3].

Smoking has adverse effects on central and peripheral airways in lungs, alveoli, and capillary and lung immune system. In addition, many studies showed a gradual transformation of pseudo stratified cilia epithelial to squamous metaplasia, carcinoma in situ and invasive bronchogenic carcinoma. In smokers, several respiratory disorders were found. In general, FEV1 values were lower and the decrease rate of FEV1 is more rapid in smokers as compared to non-smokers. Both of these effects suggest a dose-response relation with women being more dramatically than men. The presence of these findings in a middle-aged person is considered as the most important feature of severe COPD risk. Respiratory complaints increased remarkably in among smokers. There exist a dose-response relation among chronic cough, phlegm, wheezing and dyspnea. The changes responsible to these symptoms include cilia loss in airway epithelium, mucous gland hypertrophy, increased number of goblet cells and increased permeability. Smoking is the main risk factor for COPD, however it still remains unclear by which mechanism it leads to COPD. It is suggested to have led to proteolytic and anti-proteolytic imbalances, bronchial hypersensitivity as well as to inflammatory effects. About 10 to 15 % of smokers manifest a remarkable airway obstruction clinically. Besides, exposure to dust and smoke along with

respiratory tract infections at childhood could be other risk factors. Consequently, the mortality due to COPD, pneumonia and influenza is remarkably more common among smokers [1].

No cardiac pathology was found in EKO, ECG and cardiac enzyme examinations in our case. Although diuretic therapy and NIMV were carried out, no improvement was observed. Therefore, MV treatment was performed with ARDS diagnosis ignoring the possibility of cardiogenic pulmonary edema.

Treatment procedure for the patients having massive pulmonary edema resulted from increased microvascular permeability is multimodal including respiratory management comprising higher PEEP and inversely proportional ventilation, surfactant therapy and even extracorporeal membrane oxygenation (ECMO) alternatives. Additionally, topical and systemic corticosteroids may be used [4-7]. Those cases successfully treated for ARDS have under risk of bronchiolitis obliterans and bronchiolitis obliterans organizing pneumonia within 1-4 weeks following the exposure [8]. Therefore, we invited our patient for outpatient controls and maintained the follow-up with a complete response obtained in both clinical and radiological manners during follow-up.

As a consequence, ARDS is a clinical manifestation of non-hydrostatic pulmonary edema presenting with deep hypoxemia, resulted from a number of causes with a higher rate of morbidity and mortality. ARDS can occur depending on several etiological reasons. We are in opinion that it could be advantageous to consider the increased likelihood of ARDS to occur incorporating the age factor being over 65, when taken into account that smoking is common in our country.

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