Severe Acute Respiratory Distress Syndrome and Oxygen Therapy

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Abstract

Pandemic of COVID-19, there was havoc for want of oxygen and ventilator to ARDS victim due to COVID-19. Many patients died in ambulance during waiting or on way to hospital for oxygen and ventilator bed. Is oxygen and ventilator crucial for ARDS survival?

Case report

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abstract

In the pandemic of COVI-19, there was havoc for want of oxygen and ventilator bed for ARDS victim due to COVID-19. Many patients died in ambulance during waiting or on way to hospital for oxygen and ventilator bed. Is is oxygen and ventilator crucial for ARDS survival. Here is cases who survive without oxygen irrespective of oxygen saturation 76% at ambient air.

Case

A 45-year-old woman reported to the outpatient department (OPD) on 21st October 2020, complaining of fever, body-ache, headache, cough, and loss of taste sensation since 5days. She started treatment prescribed by her family doctor, but the breathlessness continued to exacerbate. Her elder son of 32 years of age was suffering from COVID-19. On examination her pulse was elevated, measuring 110 per minute, blood pressure (BP) 160/80 mmHg, respiratory rate 38 per minute, bilateral crepts were heard over both the lungs. She denied having respiratory distress despite suffering from severe dyspnoea. Her temperature was 99.4 degrees Fahrenheit and oxygen saturation was 76% at ambient air. Investigation showed haemoglobin at 11.3 gms/dl, total leucocyte count at 6080/ microL (N=4000-10000), lymphocyte count at 970.8/ microL (N=1500-4000) , lymphocytes 12.2/microL (N=40-600), c-reactive protein 63mg/L (N=0-6), serum ferritin as 86.87ng/ml (N=4.63-204), D-Dimer quantification 1837.39 ng/ml (N= <500), HbA1c 5.7%, serum B-12 as 270 ng/ml(N=197-771), vitamin D 15.7ng/ml (N=30-100), TSH 7.15 mIU/L (N= <4.5). A High-Resolution Computed Tomography of the thorax revealed 75% (23/25 score) involvement of both lungs (figure A1). She was administered nasal oxygen at 4 litres per minute on an OPD basis and her spO2 raised to 84% within one hour, but relatives and the patient denied indoor hospitalization. She was advised regular treatment at home for which they agreed. Treatment given was oral favipiravir 1800 mg 12 hourly on the first day and then 400 mg twice a day for one week. , ivermectin 12mg twice a day for five days, doxycycline 100 mg twice a day for five days, aspirin 150 mg once a day after food, metformin 500 mg once a day, dexamethasone 1mg/kg body weight and subsequent tapering doses in next two weeks, low molecular weight heparin 60 mg subcutaneously twice a day for seven days. levothyroxine 25microgram on an empty stomach. Her elder son was given practical training on how to inject heparin subcutaneously from a prefilled syringe and she was

subsequently isolated in a separate room. Her nasal swab was positive for SARS-CoV2 –RNA virus. She reported on 23rd October with no complaints except for mild weakness; her SpO2 was 91% at ambient air and 88% at the end of six minutes brisk walk. Respiratory rate was 20 per minute. On 27th October, her SpO2 was 99% at rest and was 98% after a six minutes brisk walk. She reported to hospital on 7th November with no complaints, SpO2 at rest was 95%with ambient air. Her HRCT showed on 28th November she felt almost normal; her vitals too were stable with BP of 110/70 mm hg and respiratory rate 16 per minute with no crypts in chest SpO2 was 100%. Lastly seen on 9thDecember 2020 with no complaints with SpO2 at100%. Her HCRT done on 9thth December 2020 showed patchy areas intermixed with thickened interstitial (figure A2).

Discussion

Infection by SARS-COV-2 virus evokes cytokine storm resulting in liberation of excessive IL-6 and autacoids, these liberated chemicals are responsible for excessive hyper inflammation, excessive coagulation, multiple pulmonary embolism, raised antiphospholipid antibodies, alveolar endothelial damage, hyaline membrane formation, fibrosis due to liberation with nitric oxide and exudation of fluid resulting in alveoli-perfusion defect at times and preserve the lung compliance. Irrespective of severe hypoxia as seen in the present case, the patient did not complain of difficulty in respiration, a condition commonly called dead man walking or happy hypoxia. At the post-mortem, both lungs appear stiff, stony-hard, and ten-times heavier than normal; these are difficult to be cut even by sharp instruments. There is no oxygen transfusion across the lungs despite administration of >20 litres oxygen per minute inhalation and of ventilator support. The oxygen therapy is used to reverse hypoxia, but our case recovered without giving oxygen in spite of having an oxygen saturation of barely 72%. In animal studies, hyperoxia was found to cause alveolar inflammation, fibrosis and loss of diffusion capacity **(1).**Oxygen is routinely available in almost all hospitals. It has been observed that there is a rise in the reactive oxygen species, if supplemental oxygen is administered to a case of chronic obstructive pulmonary disease (COPD) **(2).** In patients with risk factors such as ARDS, and COPD, oxygen therapy should be started when SpO2 is <88% and stopped when it is >92%. Priestly wrote "excessive oxygen might burn the candle of life too quickly and soon exhaust the animal power within", thus, oxygen has come to be recognized as both essential and toxic. Inhalation of pure oxygen can lead to convulsions, asphyxiation, blindness and death **(3).**Hence, it is imperative to have a well balanced and controlled approach towards oxygen administration; the quantity should be sufficient enough to fulfil the tissue requirement of oxygen but not more. RBC free-oxygen carriers more efficiently provide oxygen to tissue. Hyperoxia leads to the formation of reactive oxygen species, enhances the ATP synthesis and vasoconstriction **(4)** . Tissue oxygen requirement is the deciding factor of auto regulation of local blood flow (1,5). Oxygen toxicity is due to excessive formation of free radicals responsible for causing lung damage such as marked exudation, congestion and edema, accompanied with intra alveolar haemorrhage, necrosis of the alveolar cells and epithelial desquamation, due to damaged capillary endothelium, thickening of the alveolar septum and loss of type-1 pneumocytes, formation of hyaline membranes and fibrosis (appendix) **(5).**

Fatal pneumonia occurred in animals after only four days of exposure to 73% oxygen(6). Lower concentration of oxygen used for a longer time like 24-48 hours results in pulmonary toxicity including tracheobronchitis, ARDS and pulmonary interstitial fibrosis(1). Thus, hypoxemia requires a careful use of oxygen and awareness of the dangers of oxygen sensitivity and oxygen toxicity.

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Legend for figure A1- HRCT 20th October 2020

Patchy areas of consolidations are noted scattered in upper and lower lobes of both lungs, CT severity score 23/40.

A2-HRC 9th December 2020

Patchy fibrotic areas intermixed with thickened interstitial are noted in upper and lower lobes of both lungs

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