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Acute pulmonary edema – Is positive pressure ventilation with dry air useful?

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Acute pulmonary edema (APE) is a life threatening emergency. The accumulation of excessive fluid in the alveolar spaces of the lungs, interferes with gas exchange and may cause hypoxaemia and respiratory failure. Patients usually present with shortness of breath, chest pain, cold, clammy skin, with blood tinged or pink frothy sputum in severe cases. It is associated with variable clinical conditions that include the cardiovascular, respiratory, renal and other systems. The pulmonary edema may be cardiogenic (also known as hydrostatic edema), where there are deranged starling forces of the pulmonary vasculature and interstitium or non-cardiogenic (also known as increased-permeability pulmonary edema, acute lung injury, or acute respiratory distress syndrome) which is due to the direct injury of the lung parenchyma or vasculature [1]. Cardiogenic and noncardiogenic pulmonary edema, though having distinct causes, may often be difficult to distinguish because of having similar clinical manifestations.

Patients with cardiogenic pulmonary edema are usually treated with diuretics, afterload reduction and treating the underlying cause which may require other treatment like coronary revascularization. Patients with noncardiogenic pulmonary edema who require mechanical ventilation, should receive lung protective ventilation strategies, including a low tidal volume and other supportive measure along with attempts at treating the cause [2] The fluid overload in cardiogenic pulmonary edema, is usually treated with fluid restriction, positive pressure ventilation (helps move the fluid out of the lungs by increasing the intrapulmonary pressure), diuretics, vasodilators and dialysis. However, in patient with associated shock, the role of these therapies may be limited, especially the use of diuretics and dialysis.

A. Kumar et al. describe the use of dry air positive pressure ventilation in two cases of APE in the journal, one with cardiogenic shock and the other with underlying chronic kidney disease [3]. Both the patients underwent tracheal intubation due to severe dyspnea and received mechanical ventilation (pressure controlled mode). There was pink frothy secretions in the tracheal tube in both cases. They used dry air during positive pressure ventilation for a period of 24 hours in both patients. This was done by bypassing the humidifier chamber from the ventilatory circuit. They hypothesized that this would prevent over humidification, promote insensible loss of the accumulated water within the airway, improve gas exchange across the alveoli and thus prevent hypoxia during acute pulmonary edema. They observed a reduction in frothing, improvement in oxygen saturation, hemodynamic parameters, arterial blood gas, chest x-ray, lung ultrasound (B-lines) and ventilatory variables in both patients.

The authors hypothesized that this strategy that promotes insensible loss of accumulated water inside the airway, would be an additional benefit to the positive pressure ventilation which prevents diffusion of water out of pulmonary capillaries. While this is an interesting concept which seems logical to work as an adjunct to positive pressure ventilation, diuretics and other supportive measures for APE, one cannot say with certainty that this contributed to the patient improvement and it was not the effect of the positive pressure ventilation, diuretics and others supportive strategies alone.

It has been shown that healthy adult individual can exceed the usual evaporation of approximately 200–300 mL of water per day, while using dry air ventilation [4]. Excessive humidity may result in bacterial colonization ventilator malfunction from condensation [5].

A water content of 25–30 mg/litre and a temperature of around 32 °C in the inspired gas has been shown to be adequate to preserve mucocilliary function [6]. However, dry air ventilation for long duration can results in ciliary dysfunction, damage of the airway epithelium and reduced lung function which leads to atelectasis and hypoxemia. The authors used the dry air ventilation only for a period of 24 hours following which humidification was resumed. They did not experience any such adverse events related to the dry air ventilation and the patients seemed comfortable. Nevertheless, one cannot make any conclusions on the safety of this approach and this need to be investigated further, in addition to the efficacy.

In conclusion, the concept of giving dry positive pressure ventilation for a short period as an adjunctive therapy with diuretics and other supportive strategies in patients with APE, seems logical and certainly merits further investigation. This strategy may be useful especially in patients with APE in shock, where they may be some limitations to use the conventional therapies which may produce hypotension. However, the duration for which this strategy can be used safely, will first need to be determined by studying it in more patients. The efficacy of this strategy needs to be determined by conducting an adequately powered randomized trial, once the safety has been established.

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Declarations

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Ethics approval and consent to participate

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Competing interests

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