Trends in Anaesthesia and Critical Care 28 (2019) 45–46

Contents lists available at ScienceDirect

Trends in Anaesthesia and Critical Care

journal homepage: www.elsevier.com/locate/tacc

Dry air positive pressure ventilation in acute pulmonary edema: Is it equivalent to diuretics?

Sir,

Acute pulmonary edema is a medical emergency in which hypoxemia and breathlessness are major concerns and it requires immediate management [1]. The fluid accumulated in the lungs impairs gas exchange and respiratory compliance [2]. It is associated with a variable clinical condition that includes cardiovascular, respiratory, renal, cerebral, trauma to the skull or chest, infections and shock.

Various methods used to treat fluid overload are fluid restriction, positive pressure ventilation, diuretics, vasodilators and dialysis. The role of diuretics and dialysis may become limited if acute pulmonary edema is associated with shock and in such patient inotropic support is required to maintain the cardiac output. We describe the role of dry air invasive positive pressure ventilation in two patients to promote insensible water loss in patient having acute pulmonary edema with cardiogenic shock.

Case 1 was a patient with acute pulmonary edema in cardiogenic shock (Cardiac index, L/min/m^2 < 2.2). Case 2 was a patient with acute pulmonary edema with underlying chronic kidney disease (Grade IV CKD) e GFR < 25ml/min/m2. In both of the cases the underlying cause of pulmonary edema was evaluated followed by standard treatment for pulmonary edema (beta blockers, angiotensin-converting enzyme (ACE) inhibitors, diuretics). Due to severe dyspnea, both of the patients tracheas were intubated and positive pressure ventilation i.e. PCV (pressure controlled ventilation) mode was used. We observed pink froth in both cases, as watery secretion coming through the tracheal tube at the time of intubation. The details of initial clinical features, vitals and biochemical parameters are shown in Tables 1 and 2.

We have used dry air during positive pressure ventilation for an initial period of 24 hours in both of these patients of acute pulmonary edema by bypassing the humidifier chamber from the ventilatory circuit in view of accumulated water inside the airway providing humidity to dry air and preventing over humidification. Written informed consent was obtained from both of the patients for publication. We observed hemodynamic improvement and radiological variables like heart rate (HR), mean arterial pressure (MAP), oxygen saturation (SPO_2), Chest X ray and lung ultrasound (B-lines) in both patients (Table 1). We also observed improvement in frothing, arterial blood gas and ventilatory variables (Table 2). The main purpose of dry air ventilation in such patients is to promote insensible loss of accumulated water inside the airway, improve gaseous exchange across the alveoli and prevents hypoxic insult during initial period followed by switching over to conventional humidified air ventilation. Then we applied moist air ventilation after 24 hours to both these patients by connecting humidifier chamber to the breathing circuit.

1. Discussion

In mechanically ventilated patients dry and cold inspiratory air is passed from the ventilator to the humidifier chamber where air floats above the water surface and absorbs heat and humidity in the form of water vapor (pass-over-procedure). However, in case of pulmonary edema over humidification of air might occur when air passes this accumulated fluid inside lungs resulting in increased secretions, atelectasis and altered pulmonary function leading probably to arterial hypoxemia.

A healthy adult individual evaporates about 200–300 ml of water per day but this value may exceed several times during dry air ventilation. Breathing dry room temperature gases has been calculated to result in a maximum loss of 42.3 KJ/hr which is small in relation to basal energy production [3]. Mebius concluded that a water content of 25–30 mg/litre and a temperature of around 32 °C in the inspired gas should be adequate to preserve mucociliary function [4]. Gilston further added that they avoid excessive humidity, overheating, bacterial contamination and ventilator problems from condensation [5]. The dry air ventilation for long duration (more than 24 hours) results cilia dysfunction, destruction of airway epithelium and reduction in lung function which lead to atelectasis, hypoxemias & bronchoconstriction [2]. However in both of our cases we have not noted any complication related to dry air ventilation, blockade of HME filter and patients were comfortable during ventilation.

When liquid flow into heat and moisture exchangers (HME) or filters either from patient (sputum or pulmonary edema), or from the breathing system (if condensation is present) results in increase resistance to gas flow, increased work of breathing, increased peak airway pressure and, in some cases, complete occlusion, preventing adequate ventilation of the lungs. To reduce this risk, the filter should be placed at a level higher than that of the patient's lungs, with the filter layer in a vertical orientation, should be changed if obstructed or used for 48 hours. We found good improvement in both vitals and ventilatory parameter in the above two patients within 24 hours without any circuit blockade. In both of these cases we observed double benefits, the first one is the use of positive pressure ventilation to prevent diffusion of water out of pulmonary capillaries and second one is dry air which promoted insensible loss of accumulated water

https://doi.org/10.1016/j.tacc.2019.06.002
2210-8440/

Table 1
(Hemodynamic & radiological variables).

Variables	Case 1								Case 2							
Diagnosis	Acute pulmonary edema with cardiogenic shock (Cardiac index, L/min/m^2 < 2.2)								Acute pulmonary edema with chronic kidney disease (Grade IV CKD) e GFR < 25ml/min/m^2							
Time	0hr	1hr	4hr	8hr	12hr	16hr	20hr	24hr	0hr	1hr	4hr	8hr	12hr	16hr	20hr	24hr
HR (beat/min)	130	132	140	122	110	100	90	90	124	110	120	135	110	100	90	90
MAP (mmHg)	50	65	60	65	70	70	75	70	110	110	100	90	70	70	70	75
SpO_2 (%)	85	100	100	100	100	100	100	100	89	100	100	100	100	100	100	100
O_2 flow (L/min)	15lit								15lit							
FiO_2 (%)		100	100	100	100	80	80			100	100	100	100	90	80	60
Chest X ray	+++	+++	++	++	++	+	+	+	+++	+++	+++	++	++	+	+	+
Bilateral chest crepts	+++	+++	++	+	+	+	+	+	+++	+++	+++	++	++	+	+	+
Lung scan (B-lines)	>5	>5	>5	>4	>4	3–4	3–4	3–4	>5	>5	>5	>5	>5	>4	>3	>3

+++ (Worst).
+ (Improved).

Table 2
Ventilatory & Arterial blood gas variables.

Diagnosis	Case 1								Case 2							
	Acute pulmonary edema with cardiogenic shock (Cardiac index, L/min/m2 < 2.2)								Acute pulmonary edema with chronic kidney disease (Grade IV CKD) e GFR < 25ml/min/m^2							
Time	0hr	1hr	4hr	8hr	12hr	16hr	20hr	24hr	0hr	1hr	4hr	8hr	12hr	16hr	20hr	24hr
Froth present	YES	YES	YES	YES	NO	NO	NO	NO	YES	YES	YES	YES	YES	YES	NO	NO
pH	7.28	7.35	7.36	7.38	7.35	7.38	7.36	7.37	7.2	7.24	7.29	7.32	7.35	7.34	7.36	7.38
pCO_2(mm Hg)	50	40	33	35	36	32	31	32	25	22	24	28	26	24	26	24
pO_2 (mm Hg)	55	89	100	120	160	150	160	120	50	76	92	120	140	150	160	120
PIP (cm H_20)	15	18	>50	19	20	16	20	15	14	15	19	>48	20	18	18	15
PEEP (cm H_20)	10	8	8	7	7	7	6	6	8	8	7	7	6	6	5	5

inside the airway. We suggest dry air invasive positive pressure ventilation for the initial 24 hours period as a modality to be investigated in future trails in the patient, having acute pulmonary edema with cardiogenic shock or with chronic kidney disease. This was a pilot study on two patients, and the use of dry air is not a proven therapy for lung edema. Its use can further only be accepted after conducting a well-designed randomized controlled clinical trial which show improved survival of patients and faster recovery, as well as well-balanced negative effects of that therapeutic option by compensating for all possible bias of a simple observation in these two cases.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.tacc.2019.06.002.

Conflict of interest

Nil.

Source of Funding

Nil.

References

[1] A. Baird, Acute pulmonary oedema - management in general practice, Aust. Fam. Physician 39 (2010) 910–914.
[2] W.S. Colucci, Treatment of acute decompensated heart failure: components of therapy, UpToDate. Wolters Kluwer. Updated (5 December 2016). www.uptodate.com/contents/treatment-of-acute-decompensated-heart-failure components- of-therapy.
[3] F.E. Ralley, J.G. Ramsay, J.E. Wynands, G.E. Townsend, D.G. Whalley, P. DelliColli, Effect of heated humidified gases on temperature drop after cardiopulmonary bypass, Anesth. Analg. 63 (1984) 1106–1110.
[4] C. Mebius, A comparative evaluation of disposable humidifiers, *Ada* Anaesthesiologica Scandinavica 27 (1983) 403–409.
[5] A. Gilston, Hygroscopic condenser humidifiers: value for money? Anaesthesia 39 (1984) 1031–1032.

Amarjeet Kumar, Neeraj Kumar*
Department of Trauma & Emergency, AIIMS, Patna, India

Chandni Sinha, Ajeet Kumar
Department of Anaesthesia, AIIMS, Patna, India

* Corresponding author. Department of Trauma & Emergency, All India Institute of Medical Sciences, Patna, India.
E-mail address: neeraj.jlnmc@gmail.com (N. Kumar).

4 March 2019