Case Report

Induced hypothermia for trauma-related ARDS

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Abstract

We report a case of 27-year-old male with lung contusions related acute respiratory distress syndrome (ARDS) managed by ARDSNet guidelines and additional hypothermia. On 4th day, post trauma partial pressure of oxygen dropped to 38 mm of mercury (Hg), not improving even on high positive end-expiratory pressure of 18 cm water (H_2O), inverse ratio ventilation and fraction of inspired oxygen of 1. Extracorporeal membrane oxygenation was ruled out due to the risk of hemorrhage from trauma sites. Thereafter, hypothermia along with muscle paralysis was considered to reduce total body oxygen consumption. Patient's condition improved under hypothermia, and he was extubated and taken up for fracture fixation surgeries and discharged later in stable condition.

Keywords: Acute respiratory distress syndrome, hypothermia, hypoxia, trauma

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Introduction

Acute respiratory distress syndrome (ARDS) is characterized as nonpneumonic and noncardiogenic situation caused by increased permeability of vessels and severe arterial hypoxemia. It can be caused by both direct and indirect causes.[1] Early identification and implementation of standardized therapy are mandatory. We are reporting a case of ARDS managed additionally with hypothermia.

Case Report

A 27-year-old male patient was admitted after road traffic accident (RTA),with fractures of left femur, tibia, elbow, pelvis, and chest injury. The patient was initially managed at other hospital and shifted to our hospital on 3rd day after RTA.

On admission, patient was alert with heart rate, blood pressure and respiratory rate of 128 min, 90/50 mmHg and 24/min respectively, and oxygen saturation (SaO_2) of 58% on room air which improved to 100% with oxygen supplementation. Chest-X-ray (CXR) on the day of accident showed no rib fractures or lung contusion [Figure 1]. Echocardiogram was unremarkable.

Later in the morning the SaO_2 dropped to 90–92% on 5–6 L oxygen and noninvasive ventilation was initiated. Arterial blood gas (ABG) showed pH - 7.37, partial pressure of carbon $PaCO_2$ - 37 mmHg and partial pressure of oxygen (PaO_2) - 57 mmHg. CXR showed bilateral infiltrates [Figure 2], computerized tomography scan chest confirmed contusion and consolidation [Figure 3]. By evening, patient became restless and tachypneic (40/min) with SaO_2 of 80%, with bilateral crepitation's and wheeze, leading to intubation and ventilation with a fraction of inspired oxygen (FiO_2) of 1. Standard and invasive hemodynamic and oxygenation monitoring were instituted (FloTrac Edward Life Sciences) [Table 1].

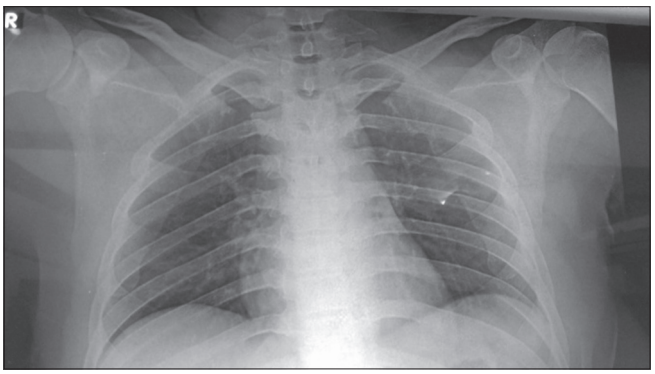

Figure 1: X-ray chest on the day of accident

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By 4th day, patient was requiring higher positive end-expiratory pressure (PEEP) of 18, FiO_2 of 1 and inverse ratio ventilation (1:1) to maintain saturation. Endotracheal suction contained blood. Routine cultures were sent, and antibiotics instituted according to unit protocol.

On the basis of CXR, low PaO_2/FiO_2 ratio and absence of cardiac cause, a diagnosis of severe ARDS with lung contusion was made according to Berlin definition.[2] Management was started according to ARDSNet protocol. By 5th day, PaO_2 was 38 with FiO_2 of 1, so inspiratory: Expiratory (I: E) ratio was further reversed (2:1).

Patient was given lateral position as prone position was not possible due to fracture upper and lower limbs. The patient received diuretics and albumin, and total fluid intake was restricted to 1800 ml/day. Our center has extracorporeal membrane oxygenation (ECMO) facility and the case fulfilled institutional criteria for the same. But in view of relative contraindication t-hat is, trauma with multiple bleeding sites[3] and on advice from a leading ECMO center the same was ruled out to prevent hemorrhage. As a last measure and to prevent organ damage from low PaO_2, hypothermia was planned with a cooling mattress (HICO - Variotherm 550). Therapeutic hypothermia was initiated as recommended by American Heart Association used in post cardiopulmonary resuscitation patients.[4] Hypothermia was instituted by cooling to 35°C over a 12 h period and continued on 6th day. Nasopharyngeal temperature was monitored and maintained at 35°C. On 6th day, PaO_2 improved, and pressure control was reduced from 20 mmHg to 16 mmHg, while maintaining PEEP at 18 cm of H_2O. FiO_2 was gradually reduced to 0.85 by that evening. By 7th day, PaO_2 improved to 275 mmHg, passive rewarming started and normothermia achieved by 24 h.

By 11th day, PEEP was brought to 13 cm of H_2O and FiO_2 to 0.6. The I: E ratio was reverted to 1:2 and sedation gradually stopped. On 12th day, patient was weaned with continuous positive airway pressure of 7. By 15th day, patient was weaned off ventilator and on 16th day, he was extubated.

Patient was taken up for fixation of fractures later. The patient developed cognitive dysfunction, which eventually recovered over time and was discharged from hospital after 1 month of admission.

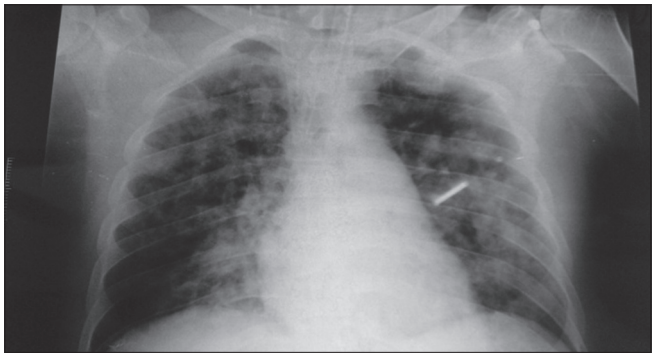

Figure 2: X-ray chest on 3rd day of accident

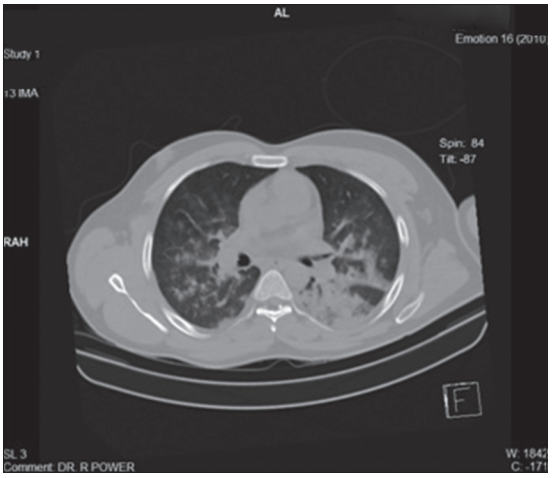

Figure 3: Computerized tomography scan chest on 3rd day of admission

Table 1: Induced Hypothermia Period: Interventions and Data

Day	Time	Ventilator mode	pH	PaO_2	FiO_2	PaO_2/FiO_2 ratio	PCO_2	Position	Temperature °C	PT/INR/APTT
4	7.00 AM	PC-15, PEEP-10	7.38	79	1	79	43	Supine	38	18.2/1.39/34.6
	10.00 AM	PC-16, PEEP-18	7.32	57	1	57	44	Supine	38	
	1.00 PM	PC-20, PEEP-18, IRV 1:1	7.31	70	1	70	43	Supine	38	
	7.00 PM	PC-20, PEEP-18, IRV 1:1	7.36	109	1	109	42	Supine	38	
5	9.00 AM	PC-20, PEEP-18, IRV 2:1	7.20	38	1	38	66	Lateral	38	19.2/1.49/not done
	12.00 PM	PC-20, PEEP-18, IRV 2:1	7.12	54	1	54	91	Lateral	37	
	8.00 PM	PC-20, PEEP-18, IRV 2:1	7.26	67	1	67	71	Lateral	35	
6	8.00 AM	PC-16, PEEP-18, IRV 2:1	7.41	147	1	147	47	Lateral	35	21.3/1.71/37.1
7	9.00 AM	PC-16, PEEP-15, IRV 2:1	7.61	275	0.85	323	29	Supine	35	19.9/1.57/40.1
8	9.00 AM	PC-16, PEEP-15, IRV 2:1	7.38	80	0.7	114	57	Supine	37	17.7/1.34/41.6
9	9.00 AM	PC-16, PEEP-13, IRV-2:1	7.37	161	0.7	230	56	Supine	37	21.2/1.70/42.9

PEEP: Positive end-expiratory pressure; PC: Pressure control; IRV: Inverse ratio ventilation; PaO_2: Partial pressure oxygen; FiO_2: Fraction of inspired oxygen; PCO_2: Partial pressure carbon dioxide; PT: Prothrombin time; INR: International normalized ratio; APTT: Activated partial thromboplastin time

Discussion

Acute respiratory distress syndrome is currently defined by "Berlin definition" as sudden in onset, life-threatening lung failure where alveoli get filled with liquid and collapse thereby decreasing compliance.[5] Laboratory test to diagnose ARDS includes ABG, CXR and echocardiography to exclude cardiac cause and tests for possible infection. The mortality rate reported is 27% in mild, 32% in moderate and 45% in severe ARDS.[6]

Most ARDS related deaths are due to multiorgan failure while refractory hypoxemia accounts for only 16% of deaths. Treatment is guided by ARDSNet guidelines and etiological management. Trials of permissive hypercarbia show that pH as low as 7.15[7] is well-tolerated. Conservative fluid therapy titrated to lower central venous pressure decreases ventilator days.[8] Prone position in severe ARDS improves oxygenation and significantly decreases 28-day and 90-day mortality.[9]

The relationship between metabolic activity and temperature is explained as Q10. This measure is the ratio of two metabolic rates separated by 10°C and is the multiple by which the global metabolism decreases by a change of 10°C. The initial prospective randomized controlled trials of Induced hypothermia with severe traumatic brain injury in humans and other studies in animal models with anoxic brain injury produced promising results.[10] Induction of hypothermia has been shown to mitigate post anoxic brain injury and minimize renal and cardiac insults as well.[11] It has been reported that hypothermia was effective in improving oxygenation and survival in septic patients with ARDS.[12] Going by the literature reports of hypothermia in ARDS and guidelines of postresuscitation hypothermia for cerebral protection, we deployed this technique as a last resort. Our aim was to reduce oxygen consumption of brain and other organs and prevent multiorgan damage in the face of resistant hypoxia to standard and advanced modes of ventilation. This effort seems to have fructified as our patient ultimately recovered without any residual organ dysfunction or damage.

Conclusion

Though our patient had significant hypoxia, he never had anoxia as in cardiac arrest. We have not witnessed any hypothermia related complications in our case. Our case report opens up the possibility of induced hypothermia as a modality of therapy for multiorgan protection in refractory hypoxia due to ARDS where there are limitations to other strategies. A well-planned trial may be the way forward to validate this technique.

References

1. Fröhlich S, Murphy N, Boylan JF. ARDS: Progress unlikely with non-biological definition. Br J Anaesth 2013;111:696-9.
2. Ferguson ND, Fan E, Camporota L, Antonelli M, Anzueto A, Beale R, *et al*. The Berlin definition of ARDS: An expanded rationale, justification, and supplementary material. Intensive Care Med 2012;38:1573-82.
3. ELSO Guidelines for Cardiopulmonary Extracorporeal Life Support. Ann Arbor, MI, USA: Extracorporeal Life Support Organization; Version 1.3; November, 2013. p. 4.
4. Nolan JP, Morley PT, Vanden Hoek TL, Hickey RW, Kloeck WG, Billi J, *et al*. Therapeutic hypothermia after cardiac arrest: An advisory statement by the advanced life support task force of the International Liaison Committee on Resuscitation. Circulation 2003;108:118-21.
5. Bernard MS, Artigas A, Brigham KL, Carlet J, Falke K, Hudson L, *et al*. Report on the American-European Consensus Conference on acute respiratory distress syndrome: Definitions, mechanisms, relevant outcomes, and clinical trial coordination. Am J Respir Crit Care Med 1994;149:818-2.
6. Fanelli V, Vlachou A, Ghannadian S, Simonetti U, Slutsky AS, Zhang H. Acute respiratory distress syndrome: New definition, current and future therapeutic options. J Thorac Dis 2013;5:326-34.
7. Kress JP, Marini JJ. Acute respiratory distress syndrome: Adjuncts to lung-protective ventilation. Semin Respir Crit Care Med 2001;22:281-92.
8. National Heart, Lung, and Blood Institute Acute Respiratory Distress Syndrome (ARDS) Clinical Trials Network, Wiedemann HP, Wheeler AP, Bernard GR, Thompson BT, Hayden D, *et al*. Comparison of two fluid-management strategies in acute lung injury. N Engl J Med 2006;354:2564-75.
9. Guérin C, Reignier J, Richard JC, Beuret P, Gacouin A, Boulain T, *et al*. Prone positioning in severe acute respiratory distress syndrome. N Engl J Med 2013;368:2159-68.
10. Bernard S, Buist M. Induced hypothermia in intensive care medicine. Anaesth Intensive Care 1996;24:382-8.
11. Polderman KH. Induced hypothermia and fever control for prevention and treatment of neurological injuries. Lancet 2008;371:1955-69.
12. Villar J, Slutsky AS. Effects of induced hypothermia in patients with septic adult respiratory distress syndrome. Resuscitation 1993;26:183-92.

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