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 (SaO2) 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 SaO2 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 PaCO2 - 37 mmHg and partial pressure of oxygen (PaO2) - 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 SaO2 of 80%, with bilateral crepitation’s and wheeze, leading to intubation and ventilation with a fraction of inspired oxygen (FiO2) of 1. Standard and invasive hemodynamic and oxygenation monitoring were instituted (FloTrac Edward Life Sciences) [Table 1].
By 4th day, patient was requiring higher positive end-expiratory pressure (PEEP) of 18, FiO₂ 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 PaO₂/FiO₂ ratio and absence of cardiac cause, a diagnosis of severe ARDS with lung contusion was made according to Berlin definition. Management was started according to ARDSNet protocol. By 5th day, PaO₂ was 38 with FiO₂ 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 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 PaO₂ hypothermia was planned with a cooling mattress (HIICO - Variotherm 550). Therapeutic hypothermia was initiated as recommended by American Heart Association used in post cardiopulmonary resuscitation patients. 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, PaO₂ improved, and pressure control was reduced from 20 mmHg to 16 mmHg, while maintaining PEEP at 18 cm of H₂O. FiO₂ was gradually reduced to 0.85 by that evening. By 7th day, PaO₂ improved to 275 mmHg, passive rewarming started and normothermia achieved by 24 h.

By 11th day, PEEP was brought to 13 cm of H₂O and FiO₂ to 0.6. The I: E ratio was reverted to 1:2 and sedation gradually stopped. On 12th 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.

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![Figure 2: X-ray chest on 3rd day of accident](image1)

![Figure 3: Computerized tomography scan chest on 3rd day of admission](image2)

### Table 1: Induced Hypothermia Period: Interventions and Data

<table>
<thead>
<tr>
<th>Day</th>
<th>Time</th>
<th>Ventilator mode</th>
<th>pH</th>
<th>PaO₂</th>
<th>FiO₂</th>
<th>PaO₂/FiO₂ ratio</th>
<th>PCO₂</th>
<th>Position</th>
<th>Temperature °C</th>
<th>PT/INR/APTT</th>
</tr>
</thead>
<tbody>
<tr>
<td>4</td>
<td>7.00 AM</td>
<td>PC-15, PEEP-10</td>
<td>7.38</td>
<td>79</td>
<td>1</td>
<td>79</td>
<td>43</td>
<td>Supine</td>
<td>38</td>
<td>18.2/1.39/34.6</td>
</tr>
<tr>
<td></td>
<td>10.00 AM</td>
<td>PC-16, PEEP-18</td>
<td>7.32</td>
<td>57</td>
<td>1</td>
<td>57</td>
<td>44</td>
<td>Supine</td>
<td>38</td>
<td>18.2/1.39/34.6</td>
</tr>
<tr>
<td></td>
<td>1.00 PM</td>
<td>PC-20, PEEP-18, IRV 1:1</td>
<td>7.31</td>
<td>70</td>
<td>1</td>
<td>70</td>
<td>43</td>
<td>Supine</td>
<td>38</td>
<td>18.2/1.39/34.6</td>
</tr>
<tr>
<td></td>
<td>7.00 PM</td>
<td>PC-20, PEEP-18, IRV 1:1</td>
<td>7.36</td>
<td>109</td>
<td>1</td>
<td>109</td>
<td>42</td>
<td>Supine</td>
<td>38</td>
<td>18.2/1.39/34.6</td>
</tr>
<tr>
<td>5</td>
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<td>PC-20, PEEP-18, IRV 2:1</td>
<td>7.20</td>
<td>38</td>
<td>1</td>
<td>38</td>
<td>66</td>
<td>Lateral</td>
<td>38</td>
<td>19.2/1.49/not done</td>
</tr>
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<td>12.00 PM</td>
<td>PC-20, PEEP-18, IRV 2:1</td>
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<td>54</td>
<td>1</td>
<td>54</td>
<td>91</td>
<td>Lateral</td>
<td>37</td>
<td>19.2/1.49/not done</td>
</tr>
<tr>
<td></td>
<td>8.00 PM</td>
<td>PC-20, PEEP-18, IRV 2:1</td>
<td>7.26</td>
<td>67</td>
<td>1</td>
<td>67</td>
<td>71</td>
<td>Lateral</td>
<td>35</td>
<td>19.2/1.49/not done</td>
</tr>
<tr>
<td>6</td>
<td>8.00 AM</td>
<td>PC-16, PEEP-18, IRV 2:1</td>
<td>7.41</td>
<td>147</td>
<td>1</td>
<td>147</td>
<td>47</td>
<td>Lateral</td>
<td>35</td>
<td>21.3/1.71/37.1</td>
</tr>
<tr>
<td>7</td>
<td>9.00 AM</td>
<td>PC-16, PEEP-15, IRV 2:1</td>
<td>7.61</td>
<td>275</td>
<td>0.85</td>
<td>323</td>
<td>29</td>
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<td>35</td>
<td>19.9/1.57/40.1</td>
</tr>
<tr>
<td>8</td>
<td>9.00 AM</td>
<td>PC-16, PEEP-15, IRV 2:1</td>
<td>7.38</td>
<td>80</td>
<td>0.7</td>
<td>114</td>
<td>57</td>
<td>Supine</td>
<td>37</td>
<td>17.7/1.34/41.6</td>
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<tr>
<td>9</td>
<td>9.00 AM</td>
<td>PC-16, PEEP-13, IRV 2:1</td>
<td>7.37</td>
<td>161</td>
<td>0.7</td>
<td>230</td>
<td>56</td>
<td>Supine</td>
<td>37</td>
<td>21.2/1.70/42.9</td>
</tr>
</tbody>
</table>

**PEEP:** Positive end-expiratory pressure; **PC:** Pressure control; **IRV:** Inverse ratio ventilation; **PaO₂:** Partial pressure oxygen; **FiO₂:** Fraction of inspired oxygen; **PCO₂:** 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. 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.

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 hypercapnia show that pH as low as 7.15 is well-tolerated. Conservative fluid therapy titrated to lower central venous pressure decreases ventilator days. Prone position in severe ARDS improves oxygenation and significantly decreases 28-day and 90-day mortality.

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. Induction of hypothermia has been shown to mitigate post anoxic brain injury and minimize renal and cardiac insults as well. It has been reported that hypothermia was effective in improving oxygenation and survival in septic patients with ARDS. 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