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Management of Acute Respiratory Distress Syndrome Due to Transfusion-Related Acute Lung Injury and Pulmonary Contusion in a Patient with Moderate Head Injury Post-Craniotomy Decompression, Epidural Hematoma, and Posterolateral Rib Fractures 2-6

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ABSTRACT

Introduction: Acute Respiratory Distress Syndrome (ARDS) is characterized by acute onset within seven days of an insult, leading to impaired gas exchange, respiratory distress not attributed to cardiac pump dysfunction, and diffuse bilateral opacities on chest X-ray (CXR). ARDS can result from direct lung parenchymal injury, such as pulmonary contusion, or indirect mechanisms, such as transfusion-related acute lung injury (TRALI), which triggers inflammatory mediator release, causing capillary leakage and damage to type I and II pneumocytes.

Case Description: A 50-year-old male was admitted to the Intensive Care Unit (ICU) following a craniotomy evacuation. On the second day of ICU care, after receiving four units of packed red cell (PRC) transfusion and subsequent extubation, the patient developed dyspnea, increased respiratory rate, elevated work of breathing, and desaturation. Clinical examination revealed decreased consciousness, tachycardia, tachypnea, and desaturation. Diagnostic imaging showed diffuse bilateral opacities without cardiac abnormalities. The patient was re-intubated and connected to a ventilator using a lung protective strategy. Broad-spectrum antibiotics and adequate tissue perfusion support were administered. The patient showed improvement and was discharged from the ICU.

Conclusion: ARDS, whether caused by direct insults like pulmonary contusion or indirect mechanisms like TRALI, requires a lung protective strategy to preserve healthy lung tissue. Early recognition and appropriate ventilatory management are critical for improving outcomes in such cases.

Keywords

TRALI, Pulmonary Contusion, ARDS, Lung Protective Strategy

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INTRODUCTION

Acute Respiratory Distress Syndrome (ARDS) is an acute clinical syndrome occurring within seven days of an insult, characterized by impaired gas exchange, respiratory distress not attributable to cardiac pump failure, and diffuse bilateral opacities on chest X-ray (CXR). Despite prompt management, ARDS is associated with high mortality, often due to severe hypoxemia resulting from compromised pulmonary oxygenation. A cornerstone of managing hypoxemia in ARDS involves mechanical ventilation employing a lung-protective strategy, which has been shown to improve patient outcomes [1].

One recognized etiology of ARDS is Transfusion-Related Acute Lung Injury (TRALI), a potentially life-threatening complication of blood transfusion. TRALI arises from the release of inflammatory mediators triggered by transfusion, leading to endothelial alveolar damage, plasma leakage, and immune dysregulation,

ultimately culminating in ARDS. Another distinct cause of ARDS is pulmonary contusion, a direct parenchymal lung injury resulting from mechanical trauma. Unlike TRALI, pulmonary contusion induces ARDS through immediate tissue damage rather than a mediated inflammatory cascade, highlighting the diverse pathophysiological mechanisms underlying this syndrome.

The lung-protective strategy, a critical therapeutic approach, aims to optimize oxygenation while minimizing ventilator-induced lung injury in ARDS patients. This strategy is predicated on the use of low tidal volumes and high positive end-expiratory pressure (PEEP), which have been demonstrated to enhance oxygenation outcomes compared to conventional ventilation methods [2]. Implementation of such strategies is typically conducted within the intensive care unit (ICU), where multidisciplinary expertise can be effectively leveraged.This case report elucidates the complex management of ARDS in a patient with concurrent TRALI and pulmonary contusion, compounded by moderate head injury post-craniotomy decompression, epidural hematoma, and posterolateral rib fractures 2-6. The objective is to highlight the challenges and therapeutic nuances in managing such a multifaceted clinical scenario, offering insights to improve clinical practice in similar cases.

CASE DESCRIPTION

A 50-year-old male patient, identified as Mr. S, with a height of 170 cm and weight of 65 kg, presented to the Emergency Department (ED) of RSHS following a motorcycle accident one day prior, during which he was not wearing a helmet. Initially, the patient reported no immediate loss of consciousness or respiratory distress post-accident. However, he developed a progressive headache accompanied by vomiting without seizures, prompting family members to bring him to the ED. The patient denied any history of hypertension, diabetes mellitus, or allergies to medications or food. Upon arrival, his Glasgow Coma Scale (GCS) score was E2M4V2, with vital signs indicating a heart rate of 101 beats per minute, blood pressure of 145/70 mmHg, respiratory rate of 28 breaths per minute, and oxygen saturation of 93-94% on a non-rebreather mask (NRM) at 10 liters per minute.

Table 1. Post-Operative Laboratory Findings

Parameter	Value
Hemoglobin (Hb), g/dL	7.4
Hematocrit (Ht), %	21.6%
Leukocytes (Lc), /mm³	12,800
Thrombocytes (Tc), /mm³	71,000
Sodium (Na), mmol/L	141
Potassium (K), mmol/L	4.2
Chloride (Cl), mmol/L	105
Glucose (GDS), mg/dL	154
PT/APTT, sec	14.5/36.5
INR	1.02
Urea (Ur), mg/dL	32
Creatinine (Cr), mg/dL	1.1
pH	7.39
HCO3, mmol/L	24.2
PCO2, mmHg	38.8
Base Excess (BE), mmol/L	0.1
PO2, mmHg	143.9
Saturation (Sat), %	97.8

Physical examination revealed ronchi in the right lung field on auscultation, with no wheezing, intercostal retraction, or epigastric retraction. Initial chest X-ray (CXR) confirmed right pulmonary contusion and posterolateral fractures of ribs 2-6, with no evidence of cardiomegaly. The patient was intubated in the resuscitation room and subsequently referred for emergency surgery.The emergency craniotomy, performed

approximately four hours after ED admission, involved evacuation of a 30 cc intracranial hemorrhage (ICH) clot in the left frontotemporoparietal region and a 30 cc epidural hematoma (EDH) clot in the right frontotemporoparietal region, with an estimated blood loss of 2500 cc. Intraoperative hemodynamics remained stable, with systolic blood pressure ranging from 90-130 mmHg and diastolic pressure from 55-85 mmHg, and no shock phase was observed. Fluid management included 2500 cc of crystalloids, 1000 cc of colloids, 580 cc of packed red cells (PRC), and 540 cc of fresh frozen plasma (FFP). Post-operatively, the patient was transferred to the Intensive Care Unit (ICU) with an endotracheal tube in place. Laboratory findings post-surgery are detailed in Table 1.

On the first ICU day, the patient was ventilated in Pressure Support Intermittent Mandatory Ventilation (PSIMV) mode with a respiratory rate of 12 breaths per minute, pressure support of 5 cmH2O, PEEP of 5 cmH2O, and FiO2 of 50%, achieving a tidal volume of 540-550 cc and oxygen saturation of 97%. Hemodynamics were stable without vasopressor support, and the patient remained under sedation. Management included analgesics (fentanyl and paracetamol), sedatives (propofol), and supportive therapies such as ceftriaxone, methylprednisolone, and nebulized ventolin. Laboratory parameters showed persistent anemia (Hb 7.4 g/dL) and thrombocytopenia (71,000/mm³). Hemodynamic trends are summarized in Table 2.On the second ICU day, sedation was discontinued, and the patient's GCS improved to E3M5Vet. A repeat CXR indicated improvement in the right lung contusion compared to the preoperative. Due to persistent anemia (Hb 7.4 g/dL), two units of PRC were transfused with a target Hb of 10 g/dL. Post-transfusion, Hb rose to 8.4 g/dL, and arterial blood gas (ABG) analysis showed stable oxygenation (PO2 139.6 mmHg, SpO2 97.4%). At 16:00, the patient underwent successful extubation following a spontaneous breathing trial (SBT), with stable hemodynamics and saturation of 97-98% on NRM. However, a second transfusion of two PRC units, initiated at 17:00 and completed by 00:00, was complicated by the onset of restlessness and tachypnea (36-38 breaths per minute) with increased work of breathing (WOB) by 06:00 the following day.On the third ICU day, at 07:00, the patient experienced desaturation to 56% and a decline in GCS to E2M2V2, necessitating reintubation. Frothy sputum was noted upon endotracheal tube suction, suggesting acute respiratory deterioration. Ventilator settings were adjusted to Pressure Control Ventilation (PCV) mode with a rate of 16 breaths per minute, pressure support of 16 cmH2O, PEEP of 10 cmH2O, FiO2 of 70%, and a tidal volume of 260-280 cc, achieving SpO2 of 86-89%. ABG post-intubation revealed severe hypoxemia (PO2 55.8 mmHg, SpO2 86.2%), consistent with ARDS, likely secondary to TRALI given the temporal association with transfusion. Echocardiography indicated normal cardiac output and fluid responsiveness.

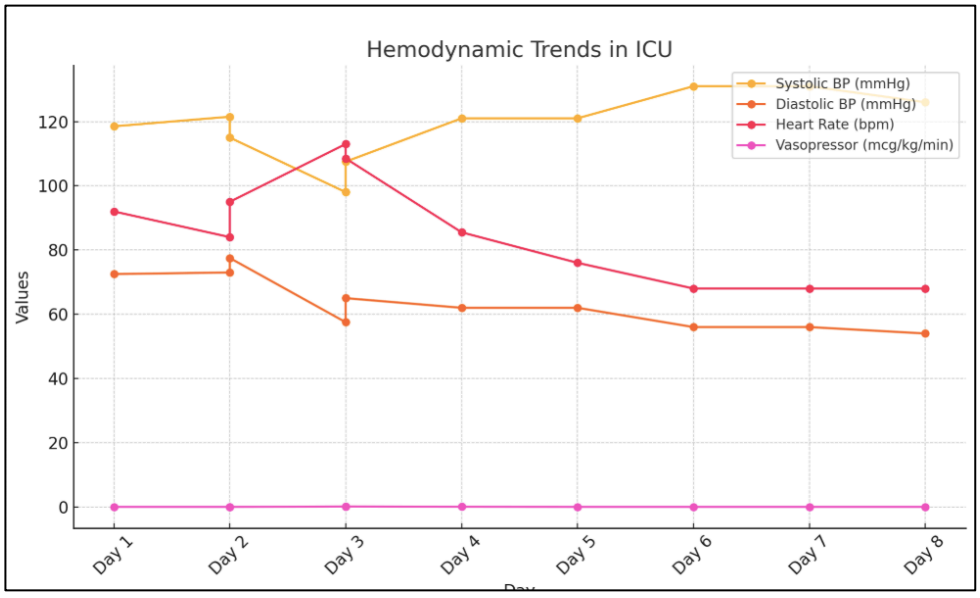

Figure 1. Hemodynamic Trends In ICU

Management adhered to lung-protective strategies, targeting a tidal volume of 4 cc/kg (260 cc), PEEP to prevent atelectrauma, and FiO2 tapering to minimize biotrauma, with additional therapies including N-acetylcysteine and repeat CXR. Hemodynamic support with norepinephrine (0.15 mcg/kg/min) was initiated due to transient instability.By the fourth ICU day, hemodynamics improved with reduced norepinephrine support (0.07 mcg/kg/min), and ventilator settings were adjusted to PSIMV with PEEP of 8 cmH2O and FiO2 of 60%, achieving SpO2 of 96%. ABG showed improving oxygenation (PO2 80 mmHg), and enteral feeding was initiated. On the fifth day, the patient developed a fever (38.5°C) with a slight leukocytosis (16,260/mm³), prompting a switch from ceftriaxone to meropenem. Ventilator weaning progressed to Pressure Support Ventilation (PSV) mode, and sedation was transitioned to dexmedetomidine. By the sixth day, fever resolved, and leukocytosis decreased (12,830/mm³), with ventilator settings shifted to CPAP mode. However, inadequate cough reflex and GCS of E2M5Vet necessitated a tracheostomy consultation. On the seventh day, post-tracheostomy, the patient was managed with a T-piece, showing stable oxygenation (SpO2 99%) and reduced ronchi. By the eighth day, the patient remained afebrile and hemodynamically stable, with plans for ward transfer following multidisciplinary evaluation. Hemodynamic trends throughout the ICU stay are detailed in Figure 1.

DISCUSSION

Obesity significantly predisposes patients to acute respiratory failure, with its severity escalating with increasing body mass index (BMI). In this case, the patient's BMI of 39 kg/m² classified her as having severe obesity, correlating with heightened airway resistance, reduced lung volume, and increased respiratory workload [6, 9]. These physiological changes, including decreased chest wall and lung compliance due to excess adipose tissue, lead to reduced functional residual capacity (FRC) and expiratory reserve volume (ERV), contributing to gas trapping and hypoxemia [9]. Obesity is both a direct cause and risk factor for respiratory complications, a finding supported by its association with chronic microatelectasis and altered ventilatory mechanics [6].

Obesity hypoventilation syndrome (OHS) in this patient, evidenced by a BMI of 39 kg/m² and pCO2 of 89.1 mmHg, is compounded by obstructive sleep apnea (OSA) and comorbidities such as heart failure and community-acquired pneumonia (CAP) [6-9]. The multifactorial pathogenesis of OHS, including increased respiratory load, diminished central drive, and leptin resistance, exacerbates hypercapnia, with untreated cases linked to a 23% mortality rate within 18 months [7]. Acute hypercapnic respiratory failure (AHRF) likely results from chronic alveolar hypoventilation worsened by infection and cardiac dysfunction, necessitating invasive mechanical ventilation [10]. Initial management with the pressure control-assist control (PC-AC) mode is aligned with recommendations for obese patients, targeting low tidal volumes and adequate PEEP to optimize compliance [1,2].

CAP, confirmed by radiographic infiltrates and clinical symptoms, contributed to the patient's respiratory decline, with early broad-spectrum antibiotics (ceftazidime and levofloxacin) adhering to the Infectious Disease Society of America (IDSA) guidelines [4]. However, subsequent Acinetobacter baumannii resistance highlights the need for culture-guided adjustments, underscoring the importance of dynamic antibiotic stewardship [4]. The role of corticosteroids remains debated, with some evidence suggesting reduced treatment failure, although not mortality benefit [4].

Congestive heart failure (CHF), driven by pulmonary hypertension (PH) and chronic hypoxaemia, further complicates the case, with furosemide and vasodilators effectively managing fluid overload [6, 10]. The successful transition to high-flow nasal cannula (HFNC) post-extubation on day 7 reflects a tailored ventilatory strategy, although the initial extubation failure emphasizes the need for meticulous readiness assessment [1,2].

Nutritional support initiated early with enteral feeding addressed catabolic stress, aligning with the ASPEN/SCCM guidelines for critically ill obese patients [3]. Overall, this case underscores the necessity of a multidisciplinary, adaptive approach for managing AHRF in OHS with complex comorbidities.

CONCLUSION

The management of acute hypercapnic respiratory failure (AHRF) in patients with obesity hypoventilation syndrome (OHS) necessitates a comprehensive strategy that incorporates noninvasive ventilation or invasive mechanical ventilation with low tidal volumes, moderate to high positive end-expiratory pressure (PEEP), and recruitment maneuvers tailored to the patient's clinical status, with the primary goals of reducing the respiratory muscle workload, correcting acidosis, alleviating hypercapnia, and improving hypoxia. Effective treatment of concomitant conditions such as community-acquired pneumonia and heart failure is critical to support patient recovery. Long-term weight loss achievable through structured dietary interventions or bariatric surgery is the most effective approach for mitigating OHS symptoms and facilitating successful weaning from mechanical ventilation, underscoring the need for a holistic and individualized treatment plan.

DECLARATIONS

None

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The Authors agree to be published in the Journal of Society Medicine.

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The authors declare no conflicts of interest in this case report.

AUTHORS' CONTRIBUTIONS

All authors made substantial contributions to the case report. RTH was responsible for patient management, data collection, and the initial drafting of the manuscript. All authors reviewed and approved the final version of the manuscript, ensuring its accuracy and integrity and being accountable for all aspects of the work.

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REFERENCE

1. Rahardjoputro R, Ernawati E, Widyaningrum NR. Efek antibiotik empiris untuk pasien pneumonia komunitas dewasa di RS X Surakarta. SENTRI J Ris Ilmiah. 2024;3(2):2311.
2. Kurniawan W, Rumende C, Harimurti K. Hipoalbuminemia pada pasien usia lanjut dengan pneumonia komunitas: prevalensi dan pengaruhnya terhadap kesintasan. J Penyakit Dalam Indones. 2017;12(48).
3. Ananda IP, Budyanra B. Determinan pneumonia status pada anak usia di bawah lima tahun. Indones J Epidemiol Kesehat Komunitas. 2024;9(2):11775.
4. Vanoni NM, Carugati M, Borsa N, et al. Management of acute respiratory failure due to community-acquired pneumonia: a systematic review. Med Sci (Basel). 2019;7(1):10.
5. Rahman IW, Prihartini A. Uji sensitivitas antibiotik terhadap pertumbuhan Kleb pneumonia dari sputum penderita infeksi saluran pernapasan bawah. J-HEST J Heal Educ Econ Sci Technol. 2022;5(3):53.
6. Esquin AM, Lemyze M, editors. Mechanical ventilation of the critically ill obese patient. 1st ed. Springer; 2018.
7. Ni Luh PD, Desak KI, Budiarsa. Sindrom pickwickian-sindrom hipoventilasi pada obesitas. Neurona. 2019;36(2):120-3.
8. Nakamura S, Kawagishi Y, Kikushima A. Acute hypercapnic respiratory failure in patients with obesity hypoventilation syndrome during the COVID-19 pandemic. Respirology Case Reports. 2023;11: e01151.

9. Neeraj MS, Georgios K. Respiratory complications of obesity: from early changes to respiratory failure. Breathe. 2023; 19:220263.
10. Sameh M, Rahma G, Amina K. Characteristics of obese patients with acute hypercapnia respiratory failure admitted in the department of pneumology: an observational study of a North African population. Sleep Disorders. 2022; 2022:5398460.