VOLUME-8, ISSUE-9, SEPTEMBER-2019 • PRINT ISSN No. 2277 - 8160 • DOI : 10.36106/gjra

Original Research Paper

Anaesthesiology

MANAGEMENT OF SEVERE ACUTE RESPIRATORY DISTRESS SYNDROME IN A PERIPHERAL FIELD ARMY HOSPITAL USING A TRANSPORT VENTILATOR -"CHALLENGES OF AN ANAESTHESIOLOGIST IN A REMOTE LOCATION"- A CASE REPORT

Lt Col (dr) Ajit Bhardwaj

M.D Anaesthesia, DM Neuroanaesthesia Asst. Prof. Anaesthesiology, Command Hospital, Lucknow

ABSTRACT Since World War I, it has been recognized that some patients with massive transfusion, sepsis, severe pancreatitis and other conditions develop respiratory distress, diffuse lung infiltrates, and respiratory failure, sometimes after a delay of hours to days. A clear definition of the syndrome was developed in 1994 by the American-European Consensus Conference (AECC) on acute respiratory distress syndrome (ARDS). ARDS causes a marked increase in intrapulmonary shunting, leading to severe hypoxemia. Although a high FIO2 is required to maintain adequate tissue oxygenation and life, additional measures, like lung recruitment with PEEP, are often required. Here we describe a challenging case of severe ARDS managed in a peripheral field army hospital located in a remote area using a transport ventilator.

KEYWORDS : ARDS, Transport ventilator

INTRODUCTION

Since World War I, it has been recognized that some patients with massive transfusion, sepsis, severe pancreatitis and other conditions develop respiratory distress, diffuse lung infiltrates, and respiratory failure, sometimes after a delay of hours to days. Ashbaugh et al described 12 such patients in 1967, using the term "adult respiratory distress syndrome" to describe this condition1.

A clear definition of the syndrome was developed in 1994 by the American-European Consensus Conference (AECC) on acute respiratory distress syndrome (ARDS)2. According to the AECC criteria, ARDS is defined by the ratio of the partial pressure of oxygen in the patient's arterial blood (PaO2) to the fraction of oxygen in the inspired air (FIO2). In ARDS, the PaO2/FIO2 ratio is less than 200. In addition, cardiogenic pulmonary edema must be excluded either by clinical criteria or by a pulmonary capillary wedge pressure (PCWP) lower than 18 mm Hg in patients with a pulmonary artery (Swan-Ganz) catheter in place (2).

ARDS is associated with diffuse alveolar damage (DAD) and lung capillary endothelial injury. Early ARDS is characterized by an increase in the permeability of the alveolar-capillary barrier, leading to an influx of fluid into the alveoli. ARDS expresses itself as an inhomogeneous process. ARDS causes a marked increase in intrapulmonary shunting, leading to severe hypoxemia. Although a high FIO2 is required to maintain adequate tissue oxygenation and life, additional measures, like lung recruitment with PEEP, are often required. Here we describe a challenging case of severe ARDS managed in a peripheral field army hospital located in a remote area using a transport ventilator.

CASE REPORT

A 21 year old woman was admitted in this peripheral field hospital with complaints of fever for last seven days .She was shifted to the ICU on Day 2 of admission with difficulty in breathing.

On Examination- Patient was restless and having severe respiratory distress.

Vitals- Heart Rate (HR)-120/min
Blood Pressure- 90/60 mm of Hg
Temp-1020F
Pulse Arterial Oxygen Saturation (SPO2) of 75-80% on face mask with oxygen @10ltr/min
Respiratory Rate (RR)- 40-50/min

Systemic Examination(S/E)-

Chest- Bilateral crepts throughout both lung fields

Per Abdomen- Hepatomegaly (liver palpable 4 cm below the costal margin)

Investigations (On Admission)

TLC-11,200/mm3
DLC-
P-78 , L-18, M-1, E-3
Platelets- 1,50,000/mm3
PT C-12 Sec
T-18 Sec
PTTK- C-30 Sec
T-50 Sec
PT INR- 1.58
SGOT- 112 IU/L
SGPT- 141 IU/L
S.Bilirubin- 2.0 mg/dl
Direct bilirubin- 1.1 mg/dl
Blood Urea Nitrogen (BUN)- 24 mg/dl
Creatinine- 0.9 mg/dl
Urine Examination- NAD
Serum Amylase- 36U/L
Serum Proteins- 6.3 g/dl
Lactate dehydrogenase(LDH) - 360 U/L
Uric Acid - 5.6 mg/dl
Chest Radiograph (on admission) (Figure-1) - NAD

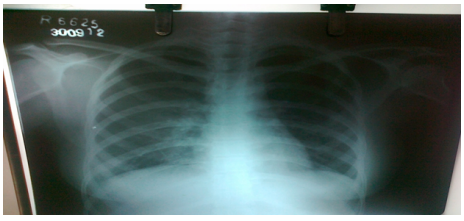

FIGURE -1

Chest Radiograph (Day-2) (Figure -2)- Bilateral Diffuse non homogenous fluffy opacities with confluence and air bronchograms

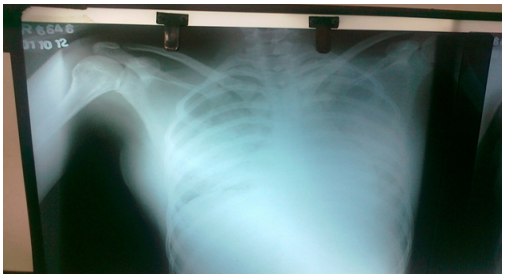

FIGURE-2

A Preliminary Diagnosis of Acute Respiratory Distress Syndrome was made based upon clinical and radiograph findings

Treatment- IV fluids to raise BP

Antibiotics- Inj Vancomycin 1gm IV BD

Inj Imipenem 1 gm IV TDS

Inj Levofloxacin 500 mg IV OD

Inj Ranitidine 50 mg IV BD

Patient developed labored breathing and SPO2 did not rise above 80% with a RR of 50/min. Patient was intubated and planned for elective mechanical ventilation.Our hospital only had an operational transport ventilator which is used for air evacuating battle injuries from peripheral field hospital to a tertiary/zonal hospitals. There were several constraints of using even the transport ventilator one of them being lack of central oxygen supplies. Transport ventilator had to be driven by jumbo oxygen cylinders which have to be continuously refilled from a place far off from field area. Also requirement of treating severe ARDS requires using sophisticated modes which are not present in a transport ventilator. Since patient was hemodynamically unstable and could not be shifted to a bigger hospital, we had to manage the patient in a field setup using a transport ventilator.

Central venous cannulation was done and CVP monitoring carried out.

Ventilator Settings:

Mode- Assist Control

Mechanical Ventilation

Tidal Volume(6 ml/kg) 330ml

RR- 18/min

FIO2- 0.7

Positive End Expiratory Pressure (PEEP)- 15 cm of Water

Patient was paralyzed with intermittent doses of Vecuronium 1mg

Propofol infusion was started @80 mg/hr for sedation

	DAY 2	DAY3	DAY 4	DAY 5	DAY 6	DAY 7	DAY9
Hb(g/dl)	7.6	8.2	9.3	8.6	8.8	9.0	10.4
TLC(cumm)	11500	11000	11800	11400	11700	9200	7900
PLATELETS (cumm)	1,50,000	1,40,000	1,56,000	1,54000	1,60,000	1,70,000	1,85,000
PTINR	1.58	1.5	1.58	1.5	_	_	1.5
PTTK(sec)	-	C-30	C-30	C- 30	_	_	C-30
		T-50	T-52	T-43	_		T-34
BUN(mg/dl)	22	23	_	44	50	31	23
CREATININE (mg/dl)	0.7	0.7	_	1.2	1.2	1.0	0.8
SGOT(U/L)	130	112	_	65	52	48	37
SGPT(U/L)	148	141	_	88	61	56	48
S BILIRUBIN (mg/dl)	2.0	1.9		1.2	1.2	1.2	1.2

Patient gradually responded to treatment and after four days of antibiotics and elective ventilation she became afebrile. Ventilator settings were regularly adjusted to reduce the FI02 gradually to 0.4 & PEEP to 08 cm of water. Chest radiograph on 4th elective ventilation day showed improvement (FIG-3) which became more evident clinically. Patient was gradually weaned off ventilatory support after stopping sedation and neuromuscular paralysis.

Patient was reversed and given Continous positive airway pressure (CPAP) and T- Piece trial before extubation on day 6 of admission Patient was placed on Non invasive ventilation with Bilevel positive airway pressure (BIPAP) in order to provide PEEP for further improvement. Intermittent BIPAP support was given for 48 h and later replaced by oxygen therapy by face mask. Patient became oxygen dependent and desaturated to a SPO2 of 83-85% on room air. With 3 days of regular chest physiotherapy and incentive spirometry it became possible to gradually reduce the FI02 and finally patient was taken off oxygen therapy. Patient was discharged 11 days after admission.FIG 4 depicts the chest radiograph one day prior to discharge.

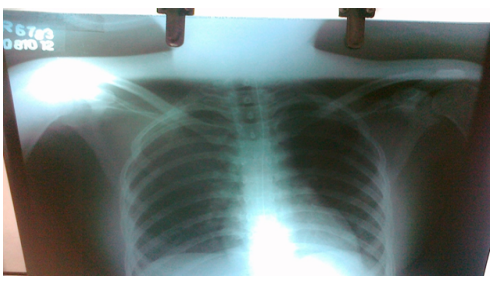

FIGURE - 4

DISCUSSION

Multiple risk factors exist for ARDS. Some of the common factors are Bacteremia, Sepsis, Trauma, Burns, Massive transfusion, Pancreatitis & Fat embolism. In our case triggering event was an infection which led to development of ARDS .

Until the 1990s, most studies reported a 40-70% mortality rate for ARDS. However, 2 reports in the 1990s, suggested mortality rates, in the range of 30-40%3,4. Possible explanations for the improved survival rates may be better understanding and treatment of sepsis, recent changes in the application of mechanical ventilation, and better overall supportive care of critically ill patients.

No drug has proved beneficial in the prevention or management of Acute Respiratory Distress Syndrome(ARDS)5-8.

Thus far, the only treatment found to improve survival in ARDS is a mechanical ventilation strategy using low tidal volumes (TV) (6 mL/kg based upon ideal body weight). An ARDS Clinical Trials Network study of a fluid-conservative strategy versus a fluid-liberal strategy in the management of patients with ARDS found no statistically significant difference in 60-day mortality between the 2 groups 72 hours after presentation with ARDS9. We used a low TV strategy along with a starting PEEP of 15 cm of water on a transport ventilator.

Mechanical Ventilation

The goals of mechanical ventilation in ARDS are to maintain oxygenation while avoiding oxygen toxicity and the complications of mechanical ventilation. Generally, this involves maintaining oxygen saturation in the range of 85-90%, with the aim of reducing the fraction of inspired oxygen

(FIO2) to less than 0.65 within the first 24-48 hours. It took us 4 days of elective ventilation to gradually reduce the FIO2 from starting value of 0.7 to 0.4 .In an ARDS Network study, patients with ARDS ventilated with a low-tidal-volume (6ml/kg) had a significantly lower mortality rate10.

CONCLUSION

Although no specific therapy exists for ARDS, treatment of the underlying condition is essential, along with supportive care, noninvasive ventilation or mechanical ventilation using low tidal volumes and an early treatment of infection.

REFERENCES

1. Ashbaugh DG, Bigelow DB, Petty TL. Acute respiratory distress in adults. Lancet. Aug 12 1967;2 (7511):319-23. [Medline].
2. Bernard GR, Artigas A, Brigham KL. The American-European Consensus Conference on ARDS. Definitions, mechanisms, relevant outcomes, and clinical trial coordination. Am J Respir Crit Care Med. Mar 1994;149 (3 Pt 1):818-24. [Medline].
3. Davidson TA, Caldwell ES, Curtis JR. Reduced quality of life in survivors of acute respiratory distress syndrome compared with critically ill control patients. JAMA. Jan 27 1999; 281 (4):354-60. [Medline].
4. Davey-Quinn A, Gedney JA, Whiteley SM. Extravascular lung water and acute respiratory distress syndrome--oxygenation and outcome. Anaesth Intensive Care. Aug 1999; 27(4):357-62. [Medline].
5. Martin-Loeches I, Lisboa T, Rhodes A, Moreno RP, Silva E, Sprung C, et al. Use of early corticosteroid therapy on ICU admission in patients affected by severe pandemic (H1N1)v influenza A infection. Intensive Care Med. Feb 2011;37 (2):272-83. [Medline].
6. Cepkova M, Matthay MA. Pharmacotherapy of acute lung injury and the acute respiratory distress syndrome. J Intensive Care Med. May-Jun 2006; 21(3):119-43. [Medline].
7. Dellinger RP, Zimmerman JL, Taylor RW. Effects of inhaled nitric oxide in patients with acute respiratory distress syndrome: results of a randomized phase II trial. Inhaled Nitric Oxide in ARDS Study Group. Crit Care Med. Jan 1998;26 (1):15-23. [Medline].
8. Griffiths MJ, Evans TW. Inhaled nitric oxide therapy in adults. N Engl J Med. Dec 22 2005; 353 (25):2683-95.
9. [Best Evidence] The NHLBI ARDS Clinical Trials Network. Comparison of two fluid-management strategies in acute lung injury. N Engl J Med. Jun 15 2006; 354 (24):2564-75. [Medline].
10. The Acute Respiratory Distress Syndrome Network. Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. N Engl J Med. May 4 2000; 342(18):1301-8. [Medline].