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INTERNATIONAL JOURNAL OF ADVANCED RESEARCH (IJAR)

Article DOI: 10.21474/IJAR01/20139
DOI URL: http://dx.doi.org/10.21474/IJAR01/20139

RESEARCH ARTICLE

SUCCESSFUL MANAGEMENT OF PATIENT OF INHALATIONAL INJURY INDUCED ARDS

Pravin Singh Thakur, Raghavendra Vagyannavar, Apoorva Singh, Amit Pandey, B.P Singh and Pragyasri Jain

Manuscript Info

Manuscript History
Received: 28 October 2024
Final Accepted: 30 November 2024
Published: December 2024

Abstract

Background: Involvement in fire accidents can be associated with inhalational injury resulting from inhalation of smoke or chemical products of combustion, which can further lead to ALI and ARDS Here we present a case of successful management of a 22 year old patient who suffered from CO poisoning with inhalational injury induced ARDS.

Case Description: A 22-year-old male came in with alleged history of involvement in a fire accident at a hotel. Initially suspected of having CO poisoning along with severe acute lung injury and acute respiratory distress syndrome. Patient was initially managed on HFNC but shifted to IMV and managed according to ARDS protocol. Over a period of 12 days, we were able to successfully treat him and move him out of the ICU.

Discussion and Conclusion: Treatment of CO poisoning is established as immediate removal of victim from the exposure and administration of high flow 100% O2. Since, there is no single management protocol for inhalational injury, this case was treated as per ARDS management protocol with bronchoscopy and bronchoalveolar lavage, which helped in significantly improving the patient' s oxygenation status and earlier recovery than expected. Hence, we hypothesize that employment of early bronchoscopy and bronchoalveolar lavage helps in accelerating the recovery of patients suffering from inhalational injury induced ARDS.

Introduction:-

One of the leading causes of accidental poisonings is exposure to carbon monoxide. It is produced due to combustion of organic matter when there is insufficient supply of oxygen. Mild poisoning causes flu like effects, headachesand vertigo. Severe poisoning can cause CNS and CVS toxicity even death.

Pulmonary injury resulting from inhalation of smoke or chemical products of combustion is known inhalation injury[(1)]. It leads to release of pro-inflammatory cytokine and toxin, alterations in regional blood circulation and perfusion[(3)(4)]. This leads to cast formation, surfactantreduction, rise in airway resistance, and decrease in pulmonary compliance[(5)]. These all leads to acute lung injury and ARDS[(6)]. Here we were able to successfully treat a 22 year old patient who suffered from CO poisoning with inhalational injury induced ARDS.

Corresponding Author:- Pravin Singh Thakur

Case Report

A 22-year-old male came in with alleged history of involvement in a fire accident at a hotel. He was found lying unconscious in his room and brought out after half hour smoke inhalation . When Patient reached our emergency department, he was on O2 support 12 L/min via NRBM, altered sensorium with GCS of E2V2M5. Patient's blood pressure was 105/53 mm Hg, heart rate was 102 beats/min, respiratory rate was 32/min, temperature 100.5 F, SPO2 was 83% at 12litre/min O2. Patient had no visible signs of external burn injury or trauma. On Systemic examination B/L crepts present .Pupils were bilaterally and equally reactive to light. There was no neck rigidity or any cranial nerve deficit at the time of presentation.There was no evidence of seizures, vomiting, urinary and bowel incontinence and substance abuse in the past. Patient was shifted to ICU for further management. ABG s/o type1 respiratory failure with pO2-40.3. Considering patients history and neurological status, CO poisoning was suspected, hence managed with HFNC on 100% Fio2 and sweep gas flow rate of 40 l/min O2. After 12 hours post admission patient was intubated due to increased respiratory distress and fall in spo2 and taken on invasive mechanical ventilation at CMV mode with 100% Fio2, 6ml/kg tidal volume and PEEP 8. Patient was taken on midazolam + fentanyl infusion. HRCT chest s/o features of ARDS (inhalational injury), which includes wedge shaped consolidation in B/L lower lobe involving basal segment with air bronchogram, irregular patchy ground glass opacities through both lung fields sparing apices and peribronchial haze associated with peribronchial and perivascular cuffing (Fig 1), chest x ray PA view findings were also s/o ARDS (Fig 2).

NCCT Head done revealed no abnormal findings. Patient was managed according to ARDS protocol using lung protective ventilation with antibiotics, bronchodilators, mucolytics and IV and inhalational steroids .Despite this P/F ratio remained below 200, hence Prone session for 18 hours was done. On day 3 after proning there was significant improvement in volume of secretions on suction and Fio2 requirement (65%). Sedation interval was given and patient was found conscious and following commands. But I/v/o high fio2 and PEEP requirement sedation started again. Bronchoscopy was done which revealed thick and copious mucus with black cast and generalised hyperemia throughout tracheobronchial tree which was s/o inhalational injury (Fig 3). Bronchoalveolar lavage also performed simultaneously (Fig 3).

I/v/o prolonged need of ventilator support and tracheal toiletting tracheostomy done on day 5. After tracheostomy midazolam-fentanyl infusion was stopped and patient was taken on dexmetomidine infusion of 0.4mcg/kg/hr. Over next 3 days considerable improvement in PEEP and Fio2 was seen. Patient gradually weaned off pressure support and put on T-piece with O2 at 1litre/min. Sedation completely stopped and patient was advised for bedside mobilisation. Through the whole course chest physiotherapy was routinely done.

Patient was able to maintain saturation on room air by day 10 . By day 12, after confirming that patient was able to maintain airway and swallowing reflexes, patient was successfully decannulated. Further monitored for 1 day in HDU and then shifted to ward.

Discussion:-

Fatalities result from carbon monoxide poisoning due to inhalational injury at the scene of the fire due to its mechanism[7]. In a closed fire space, injury is influenced by the levels of carboxyhemoglobin[8].

Usually CO toxicity happens because CO competitively binds to the heme groupswhich leads to increase in the affinity of the remaining heme groups for oxygen. This shifts the oxygen-hemoglobin dissociation curve to the left.

For Treatment of CO poisoning it is vital to remove the victim from the exposurefollowed by administration of high-flow or 100% oxygen by a NRBM. Hyperbaric oxygen (HBO) is also used in the treatment of poisoning, but consensus has still not been reached for or against its use.

In case of our patients, diagnosis was made on the basis of history, response to supportive treatment and exclusion of other causes.

Chemicals in smoke leads to injury to lower airways.The initial proinflammatory response is triggered due to burnt biological materials that are toxic to the airways. Within minutes after an inhalation injury a 10-fold increase in bronchial blood circulation can be seen[9]. This leads to increase in permeability andbronchial epithelial damage [10]. Within 24 hours following the injury, a fall in P/Fratio ≤ 200 can be seen due to increase in pulmonary transvascular fluid[11]. Another clinical finding seen is hyperemia of the tracheobronchial tree, especially lower airways which can

be used to diagnose inhalational injury[12,13]. This is followed by copious and foamy secretion formation released by goblet cells, which later on solidify and leads to airway obstruction by formation of casts[10].

Observationsinclude char, edema of airways, necrosis of mucosa, presence of sootand inflammation of airways.

Single standard protocol for management of inhalation injury has not been established yet[2]. Thetreatment consists of supportive care usually by acute hospitalization and rehabilitation[1]. Some studies have demonstrated benefits of gravity-assisted bronchial drainage techniques, chest percussion and vibrations, which are effective in removal of secretions[14,15,16] .

Early ambulation should be priority to prevent respiratory complications in patients of inhalation injury. Chair sitting can be started even in patients on continuous ventilatory support with appropriate use of analgesics.

As it is evident from our case, patients who develop ARDS need to be managed according to ARDS net strategy along with addition of bronchoscopy and bronchoalveolar lavage. Clearing away the soot particles from bronchoalveolar mucosa greatly helps in reducing the proinflammatory response. Thus, overall helping in whole recovery process.

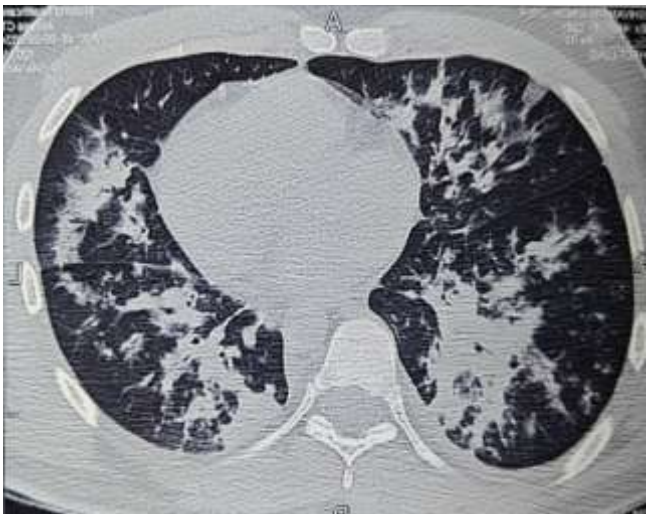

Fig. 1:- HRCT chest s/o features of ARDS (inhalational injury), which includes wedge shaped consolidation in B/L lower lobe involving basal segment with air bronchogram, irregular patchy ground glass opacities through both lung fields sparing apices and peribronchial haze associated with peribronchial and perivascular cuffing.

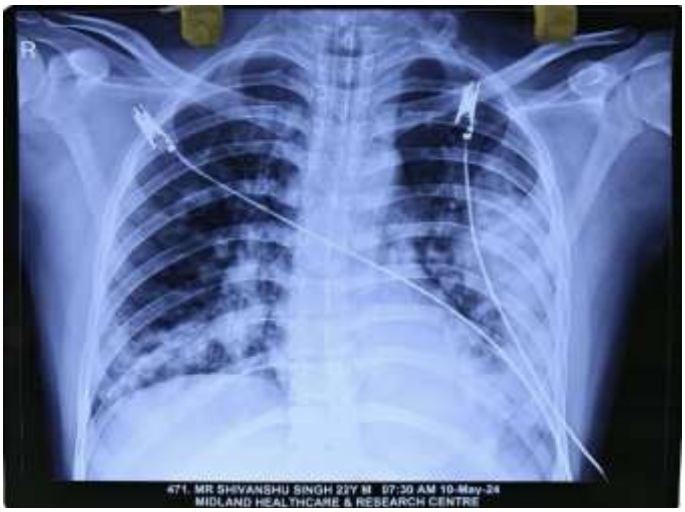

Fig. 2:- CXR showing B/L fluffy opacities.

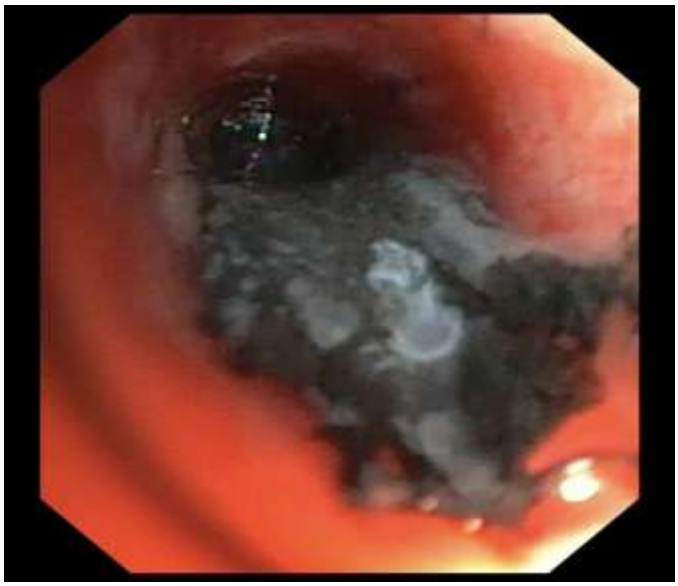

Fig 3:- Bronchoscopy revealed thick and copious mucus with black cast and generalised hyperemia throughout tracheobronchial tree.

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