

Isolated inhalational injury: Clinical course and outcomes in a multidisciplinary intensive care unit

Jose Chacko, Nikahat Jahan, Gagan Brar¹, Ramanathan Moorthy

Abstract

Background and Aims: Although smoke inhalation is well known to cause acute lung injury, there are few reports in literature that study the evolution, clinical course and outcomes of isolated inhalational lung injury in a modern intensive care setting. A major fire disaster provided us the opportunity to study victims of isolated inhalational injury admitted to our Multidisciplinary Intensive Care Unit (MICU). **Materials and Methods:** We studied the clinical course, ICU and hospital outcomes of 13 victims of a fire disaster who required mechanical ventilation for isolated inhalational lung injury. All patients were followed up at regular intervals, and their functional status was assessed at 8 months after hospital discharge. **Results:** The Lung Injury Scores (LIS) worsened to reach a nadir on Day 3 of injury. There was a significant correlation between the LIS on Day 3 and duration of mechanical ventilation ($r = 0.8; P = 0.003$), ICU ($r = 0.8; P = 0.002$) and hospital ($r = 0.6; P = 0.02$) days. Late-onset airway complications were encountered in four patients. Three of them required long-term artificial airways – two with a tracheostomy while the third patient required surgical insertion of a “T” tube. Persistent problems with phonation occurred in two patients. At 8 months postdischarge, all patients were independent with activities of daily living; all were back to work, except for two who continued to need artificial airways. **Conclusions:** Inhalational lung injury progresses over the first few days and is worst after 72 h. Late-onset airway complications may manifest after several weeks and require repeated intervention.

Keywords: Inhalational injury, intensive care, outcomes

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Introduction

In February 2010, a major fire broke out in an eight-storied building complex located in the heart of Bangalore city. The incident occurred in the late afternoon, during peak business time. There were approximately 600 people occupying the building at the time of the incident. Although the initial fire was short lived, thick columns of smoke rose up through service ducts and engulfed several upper floors. As a consequence, many of the victims suffered major inhalational injury, although none of them had serious surface burns.

Reports of isolated inhalational lung injuries are infrequent^[1,2] and mostly studied in firefighters.^[3,4] Although acute lung injury following smoke inhalation is well established, there are few studies that analyze the clinical progression and outcomes of such injuries in a modern intensive care setting. We evaluated the temporal evolution, ventilation characteristics short and long-term outcomes in victims treated in our MICU following this disaster.

Materials and Methods

Thirty-six victims were brought to the emergency department (ER) of our hospital, located 300 m down the road from the disaster site. Of these, seven were brought dead; attempts to resuscitate two others in the ER were unsuccessful. Of the remaining 27 patients, 13 required intubation and mechanical ventilation. These patients were transferred to the MICU after initial triage and

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resuscitation, and were the subjects of this retrospective observational study. The hospital ethics committee granted approval for the study.

Subjects were seen and triaged initially at the ER by medical and nursing staff. All patients were initially administered oxygen at 16 L/min through a nonbreathing mask. A quick initial assessment was made regarding the requirement for intubation and mechanical ventilation. Five patients required intubation shortly after arrival to the ER; eight others who were considered to be in impending distress were transferred to the intensive care areas of the hospital. All ventilated patients were later on transferred to the MICU. Ventilation was commenced in the pressure-controlled mode with a tidal volume of 6–7 ml/kg. FiO_2 was set at 1.0 for the first 24 h on all patients considering the possibility of carbon monoxide toxicity. Arterial blood gas analysis was done as appropriate. Serial X-rays were done to assess the extent of lung injury. After the first 24 h, the FiO_2 was weaned down and Positive End Expiratory Pressure (PEEP) levels adjusted to maintain an SpO_2 of >90%. Sedation was maintained with an intravenous infusion of morphine and midazolam. Fiberoptic bronchoscopy and bronchoalveolar lavage was performed as clinically indicated. All patients were administered an intravenous dose of hydroxycobalamin 50 mg/kg considering the potential for cyanide toxicity. Intravenous hydrocortisone was administered in a dose of 100 mg every 8 h for 3 days. Attempts to wean and extubate were made once the $\text{PaO}_2/\text{FiO}_2$ (P/F) ratio was more than 200 on a PEEP of 5 and when the airway swelling was considered to have settled down. Noninvasive ventilation was used postextubation if required. Patients who were considered likely to require prolonged ventilator support or with significant airway burns underwent bedside percutaneous tracheostomy. Appropriate cultures were done in patients who were suspected to have septic complications.

We collected data on the baseline demographic characteristics, APACHE II score, the initial Glasgow Coma Score (GCS) and the time interval between arrival to the ER and intubation. Presence of cutaneous burns and eye injuries were noted. P/F ratios and LIS were calculated on the first 3 days of ICU admission. Bronchoscopy findings and culture reports were recorded. Data was collected on extubation failure and the use of noninvasive ventilation (NIV) after extubation. Information regarding ventilation days, duration of intensive care and hospital stay were abstracted from patient records. Periodic follow-up was carried out as clinically indicated by the ICU and otorhinolaryngology

staff either in person or by telephonic interview on all subjects. At 8 months of hospital discharge, information was specifically sought on the functional status in regard to (1) status of airway, (2) presence of dysphonia, (3) ability to carry out activities of daily living (ADL) and (4) whether returned to work or not.

Statistical analysis was carried out using the Medcalc statistical software, version 11.1.1. Frequency analysis was expressed as mean (\pm SD) or median with 25th–75th interquartile range (IQR) for continuous variables. Categorical variables were expressed as number and percentage. P/F ratios on Day 1 and Day 3 were compared using the paired Wilcoxon signed rank test for nonparametric variables. Correlations between ventilation and ICU and hospital days with P/F ratios and LIS from Day 1 to Day 3 were analyzed using the Pearson correlation coefficient (*r*). A *P*-value of <0.05 was considered significant.

Results

Demographic characteristics and features on arrival to the ER are summarized in Table 1. The study population was relatively young, with a mean age of 32.5 ± 5.4 years. There were 10 males and three females, all of whom were at work at the multistoried building when the fire broke out. All except two patients had a GCS of 15 on arrival to the ER. The others had a GCS of 13 and 14. The median time from arrival to intubation was 120 min (range = 5–360). Six patients (46.2%) had stridor or breathlessness on presentation. Singeing over the face, neck or upper chest was present in five patients, while two had evidence of superficial corneal injury.

Ventilatory parameters worsened over 48–72 h in most patients, reflecting as lower P/F ratios and higher LIS. The lowest P/F ratios were encountered on Day 3; these were significantly worse than on Day 1 (95% confidence

Table 1: Demographic and basal characteristics

Pt. no.	Age	Sex	Initial GCS	Stridor at presentation
1	26	M	15	Yes
2	36	F	15	Yes
3	43	M	15	No
4	29	M	15	No
5	28	M	15	Yes
6	32	M	15	Yes
7	30	M	15	No
8	33	F	15	No
9	40	F	13	No
10	34	M	15	Yes
11	35	M	15	No
12	31	M	14	No
13	24	M	15	Yes

interval for median: 110–225 versus 256–351. $P = 0.0012$) [Figure 1]. The LIS score worsened in six patients from Day 1 to Day 3. Ventilatory parameters seemed to have improved sufficiently in six patients by Day 3 to attempt extubation. However, three of them had to be reintubated within 24 h; two of them underwent tracheostomy and required ventilator support for another 18 days, while the third could be extubated after three more days of ventilation. NIV was applied in five patients postextubation; two of them had to be reintubated.

The median duration of ventilator support was 5 days (IQR = 2–8) and the maximum duration was 21 days. The median duration of ICU stay was 8 days (IQR = 4–10), with a maximum of 24 days; the median hospital stay was 12 days (IQR = 10–18); median APACHE II score was 13 (IQR = 11–15). Patient no. 9 spent the longest time in hospital of 70 days. Three patients underwent bedside percutaneous tracheostomy on Days 5, 6 and 8 of mechanical ventilation. Short-term outcomes are summarized in Table 2.

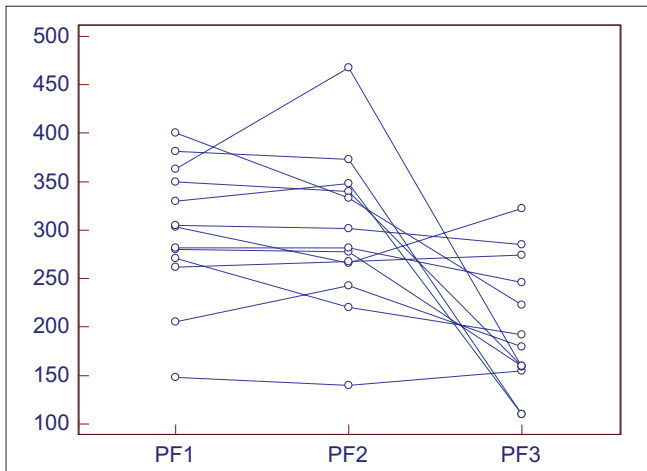


Figure 1: PaO₂/FiO₂ (P/F) ratios on Days 1, 2 and 3. PF1, PF2 and PF3 are P/F ratios on Days 1, 2 and 3

We analyzed clinical outcomes in relation to the P/F ratio and LIS. The LIS score on Day 3 correlated strongly with ventilation days ($r = 0.8$; $P = 0.003$), ICU days ($r = 0.8$; $P = 0.002$) and hospital days ($r = 0.6$; $P = 0.02$) [Figures 2–4]. There was no significant correlation of any of the above outcomes with the LIS on Day 1 or Day 2. Although the P/F ratios gradually worsened to reach the nadir by Day 3, this did not bear a significant correlation with duration of ventilation, ICU or hospital days.

The clinical course was complicated by ventilator-associated pneumonia in three patients; these patients also underwent the longest duration of ventilator support – two of them for 21 days each and one for 11 days. *Acinetobacter baumannii* was the causative organism in Patient 1, while *Pseudomonas aeruginosa* and *Escherichia coli* were grown on bronchoalveolar lavage in patient no. 3. *Staphylococcus aureus*, *Klebsiella pneumoniae* and *Stenotrophomonas maltophilia* were the incriminating organisms in patient no. 9.

Persistent or new-onset airway problems were encountered in several patients, resulting in significant disability for varying periods of time. Patient no. 4 was extubated on Day 3; however, he had complete

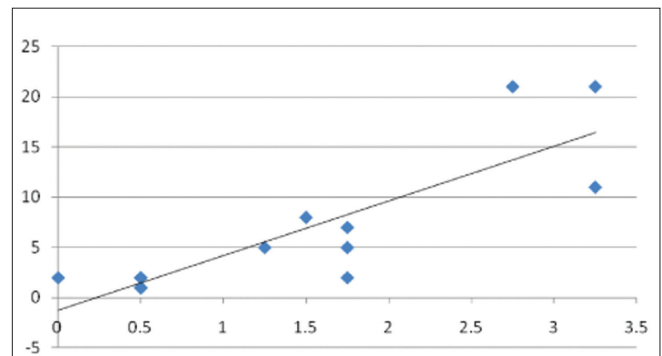


Figure 2: Correlation between Lung Injury Scores on Day 3 and ventilation days ($r = 0.8$; $P = 0.003$)

Table 2: Short-term outcomes

Patient no.	Arrival–intubation time	Ventilation days	ICU days	NIV postextubation	Hospital days	Failed extubation days	Cord involvement	Eyes	Skin
1	<15 min	11	16	Y	18		Y	Y	
2	2 h	7	9		11		Y		
3	<15 min	21	24	Y	33	3	Y		Y
4	6 h	2	4		11		Y		
5	2.5 h	2	5		9		Y	Y	Y
6	4 h	1	7	Y	10		Y		
7	2 h	1	2	Y	8				
8	<15 min	2	4		12		Y		
9	<15 min	21	24	Y	70	3	Y		Y
10	4.5 h	8	10		18				Y
11	6 h	2	4		7		Y		
12	<15 min	5	8		16				Y
13	2 h	5	8		16	3	Y		

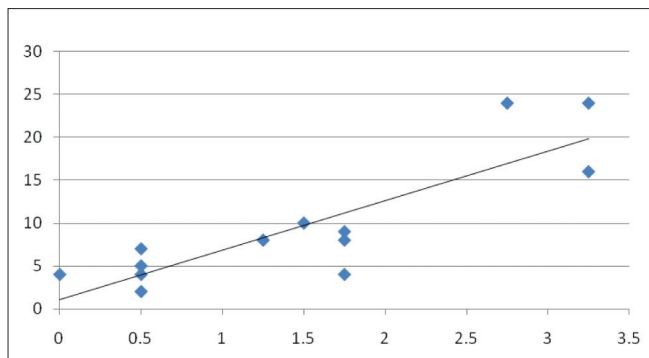


Figure 3: Correlation between Lung Injury Scores on Day 3 and ICU days ($r = 0.8$; $P = 0.002$)

aphonia. Fiberoptic bronchoscopy revealed significant inter-arytenoid and vocal cord edema, although he had no stridor. His voice began to improve after a period of 3 weeks. However, at 9 months, his voice had still not returned to normal. Patient no. 3, who was ventilated for 21 days, had significant aphonia. In addition, he started experiencing stridor on exertion by 8 weeks of hospital discharge. Fiberoptic bronchoscopy revealed an annular stenotic segment 2.5 cm below the vocal cords, which was partially dilated by rigid bronchoscopy (outer diameter 8.2 mm) with significant symptomatic relief. Patient no. 13 had hoarseness of voice at the time of discharge from hospital; however, he had no stridor at this stage. He presented 11 weeks postdischarge with increasing stridor, which worsened on exertion. Indirect laryngoscopy revealed congestion of the posterior third of the vocal cord and edema in the interarytenoid region. The cords were in a paramedian position with restricted movement. He was subjected to an open tracheostomy. On computed tomography scan, a tight subglottic stenosis, 1.5 cm long, with the narrowest part measuring 3 mm, was noted. He underwent surgical release and mitomycin application followed by repeated dilatation by rigid bronchoscopy with significant improvement of the tracheal lumen. He was successfully decannulated at 6 months with no persisting symptoms. Patient no. 12 presented 6 months after discharge with hoarseness of voice, shortness of breath and noisy breathing with exertion. On rigid bronchoscopy, a spiral stenotic segment was noted 2.5 cm below the vocal cords. In spite of progressive dilatation, he continued to have stridor. A "T" tube was inserted after 8 months of hospital discharge, which is still in place at the time of writing. Patient no. 9, who was ventilated for 21 days, had persisting hoarseness of voice postdecannulation with copious secretions. Over time, there was progressively worsening breathing difficulty characterized by stridor, which required retracheostomy at 4 months postdischarge. Bronchoscopy showed glottic as well as supraglottic inflammation. The cords were adherent

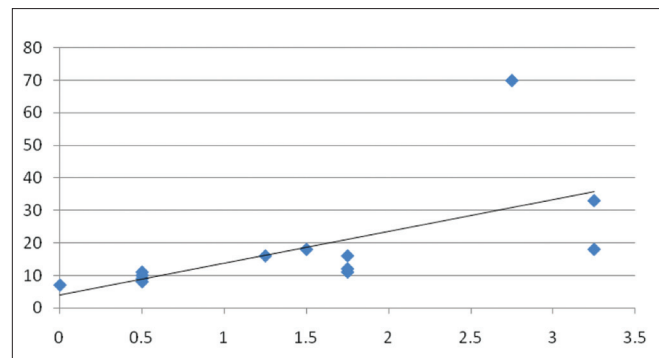


Figure 4: Correlation between Lung Injury Scores on Day 4 and hospital days ($r = 0.6$; $P = 0.02$)

together and repeated attempts to open them with laser and keel placement have so far been unsuccessful.

We evaluated the functional status of all patients at 8 months postdischarge. All subjects were independent with ADLs at this time. Two patients continued to require artificial airways – one on a tracheostomy and the other on a "T" tube awaiting further intervention to address subglottic stenosis; all except these two patients had returned back to full-time work. Two patients had persisting dysphonia, especially while attempting to raise the pitch of their voice. During the posthospitalization period, five patients reported to have sleep disturbances and significant feeling of depression.

Discussion

Respiratory failure is the most common cause of death after burn injury,^[5] with the highest mortality observed in subjects with combined burn and inhalational injury.^[6,7] Smoke inhalation can lead to major airway and parenchymal injury even in the absence of significant hypoxemia at the onset of the insult.^[8,9] Our subjects were young men and women who suffered inhalational injury without significant cutaneous burns. We intubated our patients early – five/13 patients were intubated soon after arrival in the ER; all were intubated and initiated on mechanical ventilation within 6 h of presentation. Semi-urgent intubation before the onset of significant proximal airway edema has been shown to be the safer approach.^[10,11] This may be particularly relevant in a situation where clear-cut indicators are not available to reliably predict those who might develop airway complications at a later stage.

Smoke exists in two phases – the gas and the particulate phase.^[12,13] The gas phase includes several toxins, including carbon monoxide, cyanide, aldehydes and oxidants.^[14,15] These toxins can cause mucosal injury and irritation, leading to bronchoconstriction and excessive

secretions. This type of injury evolves over the first few hours, although it may occur earlier in subjects who have irritable airways.^[16] Deposition of particles in the distal airways and the lungs results in a severe form of injury, characterized by intense mucosal inflammation.^[17,18] Failure to clear particulate material due to impaired ciliary activity perpetuates the injury process.^[19] One of the early changes of inhalational injury is the increase of pulmonary vascular resistance, possibly chemically mediated, leading to an increase of pulmonary capillary pressure and fluid leak.^[20,21] Neutrophils adhere to the alveolar capillary membrane leading to an outpouring of protease and oxidants, resulting in aggravation of lung injury.^[22] Nitric oxide upregulation may play an important role in aggravating burn and inhalational injury by inhibiting hypoxic pulmonary vasoconstriction and shunting of blood to relatively less-ventilated areas of the lung.^[23,24] Airway obstruction due to fibrin, neutrophils, mucus and epithelial debris may lead to alveolar hypoventilation, collapse and worsening hypoxemia.^[25]

We noted a progressive worsening of lung injury over time, with the worst LIS on Day 3. The P/F ratios also worsened in the first 72 h, reaching a nadir on Day 3, before beginning to show improvement in most patients. In fact, three patients who were extubated on Day 3 worsened further, requiring reintubation. These patients developed ventilator-associated pneumonia and required a longer duration of mechanical ventilation and ICU and hospital stay. It is possible that extubation failure contributed to worsening of the clinical course and longer time to recovery in these patients. The LIS score on Day 3 correlated strongly with the duration of mechanical ventilation and ICU and hospital stay. The LIS on Day 1 and Day 2 did not correlate with these outcomes. Although the P/F ratios on Day 3 were significantly worse compared with Day 1, they did not correlate with duration of ventilation or ICU or hospital stay. A previous series also reported worsening of lung injury during the first 24 h of the insult, although no mention was made of the subsequent evolution of the injury.^[1] Normal chest X-rays on initial examination followed by worsening have also been reported previously following smoke inhalation without burns.^[26] The three patients who spent the longest time on mechanical ventilation developed ventilator-associated pneumonia. Inhibition of ciliary function following smoke inhalation leads to inability to clear secretions and bacteria, leading to bacterial colonization and, very often, infection.^[27]

Late-onset airway complications requiring repeated intervention developed in four patients in our study.

All these patients had required mechanical ventilation; three of them needed a tracheostomy. None of them had airway problems initially after removal of the endotracheal/tracheostomy tube and presented with symptoms of airway obstruction weeks later – two requiring retracheostomy while the third patient required a “T” tube insertion. Late-onset vocal cord and tracheal stenosis were previously reported in six of 96 patients who suffered inhalational injury, although the time profile of evolution of these complications was not mentioned.^[2] In another study, one of 13 mechanically ventilated patients developed tracheal stenosis due to granuloma formation that required endoscopic intervention.^[1]

Thermal injuries below the glottis are relatively rare due to absorption of heat by the upper airway that acts as a heat exchanger;^[28] the low specific heat of air and reflex glottic closure that occurs in conscious subjects may also attenuate such injury.^[29] Three of our patients who had delayed airway complications had subglottic stenosis, while in the fourth case, the vocal cords were involved. Tracheal stenosis due to an endotracheal or tracheostomy tube has a predilection for narrowing at the site of the cuff. The fact that all our patients had glottic or subglottic involvement suggests that the mechanism was most likely from the inhalational injury. It is also plausible that the initial damage due to smoke inhalation might render the airways more susceptible to further injury from an endotracheal or tracheostomy tube.^[30,31] Vocal cord problems are more commonly encountered after prolonged translaryngeal intubation.^[32] One of our patients who developed vocal cord stenosis had a tracheostomy performed after only 6 days of endotracheal intubation, making it unlikely that the endotracheal tube may have been the primary cause of the problem. Although vocal cord complications have been reported following inhalational injury,^[33] late-onset significant airway compromise after 4 months of the injury, due to adhesion of the vocal cords, appears to be poorly recognized.

We treated our patients with intravenous hydrocortisone for the first 3 days after the injury. The role of steroids in inhalational injury is uncertain and inadequately studied. One human study could find no effect of steroids on lung-related morbidity or mortality.^[2] However, no harm has been reported with the short-term use of steroids in this setting, and it may have a positive effect on physiological and radiological changes caused by irritant gases like nitrogen oxides.^[31,34] All our patients received intravenous hydroxocobalamin on admission to the ICU considering the possibility of cyanide toxicity.

Cyanide toxicity should be suspected in all indoor fires with significant exposure to smoke. Hydroxocobalamin, a Vitamin B12 derivative, is a powerful chelator of cyanide and has been shown to improve survival from cyanide poisoning.^[35]

We did not have access to measurement of carbon monoxide (CO) and cyanide levels in our patients. Neurological manifestations including impaired vigilance and confusion are early signs of acute CO monoxide toxicity.^[36] All except two of our patients had a GCS of 15 at the time of presentation, making significant CO toxicity unlikely.

Although inhalational injury in the absence of cutaneous burn injury is associated with lower mortality, it can lead to significant morbidity requiring prolonged ventilator support and hospital stay. Clinical features, X-ray involvement and degree of diffusion defect in the lung as evidenced by blood gas analysis in the first 48 h may not be indicative of the degree of parenchymal injury and can lead to significant worsening subsequently. In fact, the initial picture could lead to a misleading underestimate of the severity and subsequent progression of the injury. Clinical outcomes may be better correlated to the severity of lung injury 72 h after the insult. Airway problems involving the glottic and subglottic regions may occur several weeks after the injury and require repeated endoscopic intervention. Our patients did well long term; all except two were back to work at 8 months post hospital discharge, although two of them had continued problems with communication due to persisting difficulty with phonation.

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