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Case Report

Two cases of Acute Lung Injury following closed space smoke inhalation

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Introduction

Smoke inhalation is a major cause of mortality and morbidity in closed space fire accidents.¹ Pulmonary effects of smoke inhalation vary from cough and dyspnea within minutes to hours of exposure² to Acute Lung Injury (ALI), Acute Respiratory Distress Syndrome (ARDS)³ and long term airway obstruction.² We report two cases of ALI who presented after smoke inhalation during a fire accident onboard an Indian Naval Submarine.

Case report

Case 1

A 24 year old male patient presented with complaints of breathing difficulty, cough with blackish expectoration and chest tightness after 15 min exposure to smoke during fire onboard a submarine. His examination revealed tachycardia, tachypnea, Oxygen saturation (SpO₂) of 92% on room air and bilateral crackles and diffuse wheeze on chest examination. Rest of systemic examination was within normal limits. His initial chest radiograph was within normal limits, repeat chest radiograph done after 12 h revealed bilateral lower zone air space opacities (Fig. 1). Arterial Blood Gas analysis showed a PaO₂/FiO₂ ratio of 274 (<300). Computerized Tomographic (CT) scan chest revealed areas of ground glass opacities and patchy consolidation in both lower lobes (Fig. 2). He was managed with humidified oxygen to maintain SpO₂ more than 90% and PaO₂ >60 mm Hg, intravenous fluids, nebulized bronchodilators, mucolytics, steroids and antibiotics. The patient was asymptomatic and had bilateral normal breath sound after 07 days of treatment. CT chest repeated after 04 weeks (Fig. 3) revealed complete resolution of radiological opacities. Spirometry and diffusion capacity of lung for carbon monoxide (DLCO) done at time of discharge and at follow up after 3 months were normal.

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Fig. 1 – Chest radiograph showing bilateral non homogenous opacities in lower zones.

Case 2

A 21 years old male patient presented with history of breathlessness, cough with blackish expectoration, wheezing and throat pain after 20 min exposure to smoke. His examination revealed tachycardia, tachypnea, Oxygen saturation of 90% on room air and bilateral wheeze and crackles on chest examination. Rest of systemic examination was within normal limits. His initial chest radiograph was within normal limits, repeat chest radiograph done after 12 h revealed bilateral mid and lower zone air space opacities (Fig. 4). Arterial Blood Gas analysis showed a $\text{PaO}_2/\text{FiO}_2$ ratio of 218 (<300). Computerized Tomographic (CT) scan chest revealed areas of ground glass opacities and patchy consolidation in both lower lobes (Fig. 5). ENT evaluation revealed mild erythema of arytenoids. He was managed with humidified oxygen to maintain SpO_2 more than 90% and $\text{PaO}_2 >60$ mm Hg, intravenous fluids, nebulized bronchodilators, mucolytics, steroids and antibiotics. The patient was asymptomatic and had bilateral normal breath sound after 10 days of treatment. He had complete radiological resolution (Fig. 6) on CT repeated after 4 weeks. Spirometry done at

Fig. 2 – CT Chest showing patchy ground glass opacity and air space consolidation in both lower lobes.

Fig. 3 – Complete resolution of radiological opacities after treatment.

Fig. 4 – Chest radiograph showing bilateral non homogenous opacities in mid and lower zones.

discharge revealed mild obstruction, DLCO was normal. He was managed with inhaled corticosteroids and long acting beta 2 agonist. Spirometry at 03 months showed persistence of mild obstruction, DLCO was normal. Spirometry and DLCO at 06 months follow up visit were normal.

Discussion

Smoke inhalation may involve the upper airway causing thermal injury, lower airways and lung parenchyma may be

involved due to various toxins and chemicals in smoke and metabolic asphyxiation may occur due to impaired delivery or consumption of oxygen.⁴ Exact mechanism of Acute Lung Injury in smoke inhalation is unclear but various factors implicated include Nitric Oxide (NO), poly (ADP-ribose) polymerase (PARP) and airway obstruction.³ Diagnosis of smoke inhalation injury is usually obvious based on history and clinical examination. Usually chest radiograph have a very little value in diagnosis of smoke inhalation injury.⁵ Computerized Tomography (CT) scan have higher sensitivity in diagnosis of pulmonary injury after smoke inhalation, specially during early hours.⁶ In our patients also initial chest radiographs were normal and repeat radiographs only revealed abnormalities which were confirmed on CT scan. Treatment for smoke inhalation injuries remains supportive.⁵ Various strategies to improve oxygenation in these patients may vary from humidified oxygen to non invasive ventilation or invasive ventilation based on the clinical condition of the patient.⁷ Medical adjuncts include beta agonist, aerosolised heparin with N acetylcysteine and anti inflammatory agents.⁷ Our patients were given humidified oxygen, beta agonist and N acetylcysteine and showed appropriate clinical and radiological recovery. The role of steroids in inhalation injury remains inadequately studied and uncertain but same have been used by other authors in similar setting.⁸ In our cases we used Injection Hydrocortisone 100 mg thrice daily followed by oral prednisolone in dose of 0.5 mg/kg for a total duration of five days. No harm from short term use of steroids in this setting has been reported.⁸ Measurement of Carbon Monoxide and Cyanide levels is recommended however same could not be carried out in our patients. Inflammatory changes in airway may persist for 6 month after smoke inhalation² and association of smoke inhalation resulting in injury to small airways has also been demonstrated.¹ In our patients, Case 2 also showed mild obstruction at discharge and initial follow up. In conclusion patients with smoke inhalation are prone to develop both early and delayed pulmonary complications and management remains primarily supportive.

Fig. 5 – CT Chest showing patchy air space consolidation involving both lower lobes.

Fig. 6 – Complete resolution of radiological opacities after treatment.

Conflicts of interest

All authors have none to declare.

REFERENCES

1. Fogarty PW, George PJ, Solomon M, et al. Long term effects of smoke inhalation in survivors of the King's Cross underground station fire. *Thorax*. 1991;46:914–918.
2. Park GY, Park JW, Jeong DH, Jeong SW. Prolonged airway and systemic inflammatory reactions after smoke inhalation. *Chest*. 2003;123:475–480.
3. Enkhbaatar P, Traber DL. Pathophysiology of acute lung injury in combined burn and smoke inhalation injury. *Clin Sci*. 2004;107:137–143.
4. Cancio LC. Airway management and smoke inhalation injury in the burn patient. *Clin Plast Surg*. 2009;36:555–567.
5. Antonio AP, Castro PS, Freire LO. Smoke inhalation injury during enclosed-space fires: an update. *J Bras Pneumol*. 2013;39:373–381.
6. Reske A, Bak Z, Samuelsson A, et al. Computed tomography – a possible aid in the diagnosis of smoke inhalation injury? *Acta Anaesthesiol Scand*. 2005;49:257–260.
7. Dries DJ, Endorf FW. Inhalation injury: epidemiology, pathology, treatment strategies. *Scand J Trauma Resusc Emerg Med*. 2013;21:31–46.
8. Chacko J, Jahan N, Brar G, Moorthy R. Isolated inhalational injury: clinical course and outcomes in a multidisciplinary intensive care unit. *Indian J Crit Care Med*. 2012;16:93–99.