HOLI COLOUR INHALATION CAUSING ARDS: A LIFE THREATENING ASTHMA EXACERBATION

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ABSTRACT Acute Lung Injury (ALI) and the Acute Respiratory Distress Syndrome (ARDS) are severe respiratory diseases that have a very poor prognosis and have numerous causes. Here we are reporting a very unusual case of chemical inhalation induced ARDS due to exposure to holi colours. A 24-year-old female patient, known case of Bronchial Asthma (poorly compliant to inhaler therapy) presented with sudden onset respiratory distress After extensively excluding other plausible causes, diagnosis of exclusion was made as Bronchial Asthma with Inhalational Injury due to colour dust. Patient responded on steroid therapy, there was an improvement in oxygenation and NIV support was gradually tapered off.

KEYWORDS respiratory distress, lung injury, bronchial asthma

Introduction

Acute Lung Injury (ALI) and Acute Respiratory Distress Syndrome (ARDS) are severe respiratory diseases with a very poor prognosis and numerous causes. In 1967 Ashbaugh and colleagues described a distinctive form of severe, acute respiratory failure pathologically similar to infantile respiratory distress syndrome as having a hyaline membrane, atelectasis, and alveolar haemorrhage. Patients had symptoms of dyspnoea, tachypnoea, hypoxemia (refractory to oxygen therapy), decreased thoracic compliance and radiographic diffuse pulmonary infiltrates. Subsequently, this pattern of respiratory abnormalities became known as Adult Respiratory Distress Syndrome (ARDS). (1,2) Despite applying various empirical approaches to treatment, the mortality rate for ARDS remains between 60 to 75%. (3)

Numerous recognized causes of ALI (hence ARDS) fall into two broad categories; direct injury to lung tissue and indirect lung injury. Common direct injuries include toxic inhalation, aspiration, near drowning, pneumonia, and lung contusion. Common indirect injuries are systemic inflammatory response syndrome (SIRS), drug/ chemical ingestion, severe non-thoracic trauma, and other organ system diseases such as pancreatitis. Chemical inhalational ARDS have very few documented incidents, such as due to high 02 (60%), ZnCI, NH, NO, CI, phosgene, acrolein, toluene diisocyanate, and paraquat. (4)

Here we are reporting a very unusual case of chemical inhalation-induced ARDS due to exposure to holi colours. Holi is an Indian festival of the summer season in which coloured powders (gulal) are used for playing in the fashion of throwing and spraying dry and wet colours on each other and in the air. Holi colours consist of more than 40 – 75% PM10 particles (< 10 µm), which can reach up to the lower respiratory tract. Holi colours have been studied to induce the production of pro-inflammatory cytokines TNFα, IL-6, and IL-1β. They stimulated increased production of PMNs in blood and increased monocytes. Higher concentrations can have cytotoxic effects by inducing oxidative bursts in human granulocytes and monocytes. (5)

Case report

A 24-year-old female patient known case of Bronchial Asthma (poorly compliant to inhaler therapy) presented with sudden onset respiratory distress. She had symptoms of excessive coughing with serous blood-tinged sputum, breathlessness and chest tightness. Upon examination, she was found tachypnoeic, actively using accessory muscles. She was severely hypoxemic, with initial saturation was 88% on room air. She was initiated on oxygen therapy, nebulised with salbutamol, ipratropium and budesonide, and administered injectable steroid hydrocortisone.

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Radiography revealed bilateral diffuse pulmonary infiltrates and Arterial blood gas showed metabolic acidemia (pH 7.195, pc02 33mmHg, Hco3 – 13.4) with hypoxemic respiratory failure (pao2 – 66.3mmHg, fio2 – 60%). She was diagnosed with ARDS based on the radiographic features and P/F ratio. Therefore, she was admitted to Intensive Care Unit. HRCT chest revealed bilateral ground glass haziness in the middle and lower lobes. Routine blood investigations revealed leucocytosis with predominant lymphocytes. Blood and sputum cultures were negative; RTPCR for COVID-19 and H1N1 PCR were negative. The patient was treated with β-lactam IV antibiotic, inj methylprednisolone 40 g once a day, nebuliser, and rest symptomatic. Negative culture reports for pyogenic and fungal organisms and normal CRP, PCT, and βD glucan ruled out differentials of infection. CBNAAT for MTb and AFB smear were negative. Cardiogenic pulmonary oedema was ruled out by normal 2dEcho and NTproBNP levels. Another differential for alveolar haemorrhage was considered CTD, autoimmune disease, and serological markers were done ENA profile, RA factor, Anti CCP, Anti-GBM antibody, Anti phospholipid antibody, Anti Cardiolipin antibody was done which were all negative. Upon further enquiry, it was found that the patient had a history of playing holi the same day 4-6 hrs before the onset of symptoms and had a history of allergy to holi colours. Hence after extensively excluding other plausible causes, the diagnosis of exclusion was made as Bronchial Asthma with Inhalational Injury due to colour dust.

The patient responded to steroid therapy, there was an improvement in oxygenation, and NIV support was gradually tapered off. The patient's leucocytosis was reduced, and the bilateral lung infiltrates were reduced radiographically. Spirometry showed obstructive changes with reversibility. After 5 days of ICU stay, the patient was discharged in a stable condition at room air, on tapered oral steroids and inhaler medications. A Follow-up CT chest was done after 2 weeks after the initial day of insult, which showed normal bilateral lungs and complete resolution without residual fibrosis.

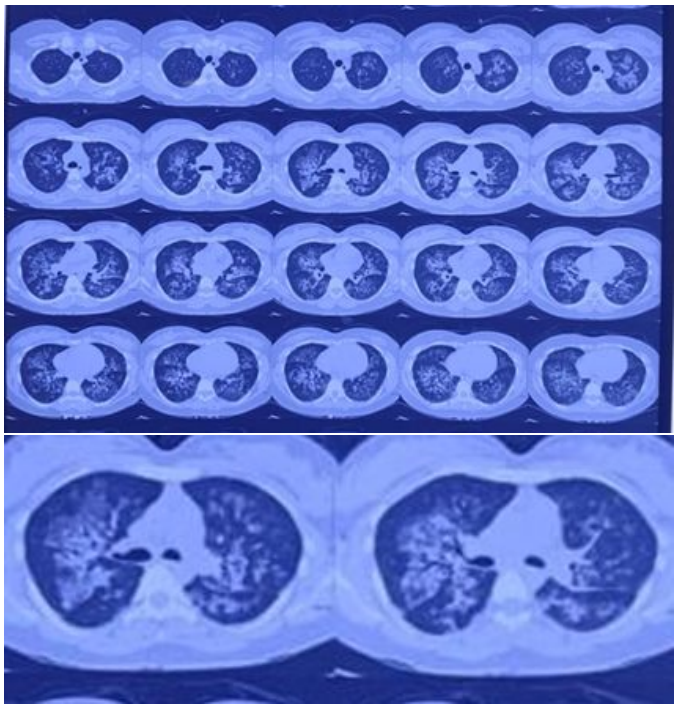

Figure 1 HRCT Chest showing diffuse bilateral diffuse ground glass opacities, central distribution, involving both upper, middle and lower lobes.

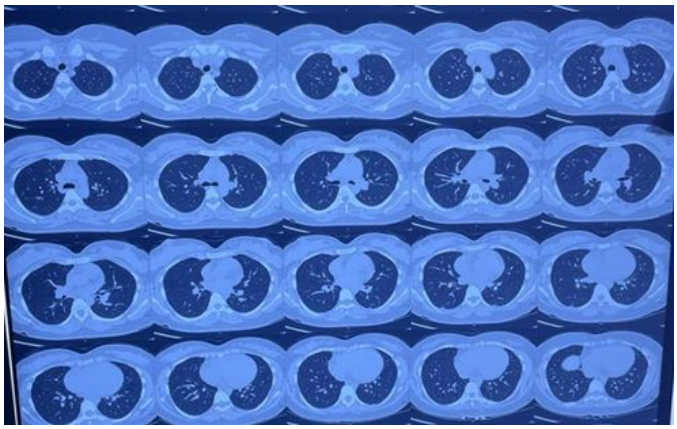

Figure 2 Follow-up HRCT after 2 weeks showing complete resolution of the ground glass shadows, normal lung parenchyma and no residual fibrosis.

Discussion

Inhalational lung injuries are not very commonly encountered in the general population. The occurrence of inhalational injury has mostly been reported due to accidental exposure to fire smoke, inhalation of industrial chemicals known to cause lung damage like chlorine gas, which occurred accidentally due to exposure without protective equipment, and another report was in soldiers due to exposure to hexit smoke (zinc chloride) from ammunition bombs at military training. (6,7,8) In all these settings, the causative agent was known to cause acute lung injury and respiratory distress, and immediate medical care and appropriate treatment could be initiated accordingly. Hence, diagnosing the cause of ARDS becomes challenging when an inhalational injury occurs due to substances that are not known risk factors for respiratory illness.

In the case reported here, a vast population commonly uses the causative agent holi powder (gulal) to play colours. However, such severe respiratory illness has not yet been reported. This highlights that such conditions could be easily missed or delayed, leading to life-threatening death. The patient reported here is a healthcare worker and was present on hospital premises at the time of the attack, leading to prompt access and early initiation of treatment. Our patient presented typically as an acute exacerbation of bronchial asthma but was promptly diagnosed and treated for ards. We here point out the important red flag signs in this patient: unproportionate hypoxemia, not responding to oxygen support, excessive cough with productive frothy reddish sputum, which indicated alveolar damage and bilateral crepitation with rhonchi. All these findings suggested some other pathology along with asthma exacerbation, which could prove detrimental if misdiagnosed and not treated promptly. Extensive workup is important to rule out other possibilities of acute illness. However, the history of inhalant exposure should always be sought after and proved to be the crux for establishing the diagnosis.

Inhalational injuries respond well to treatment, systemic steroids, nebulised bronchodilators and symptomatic management. However, there have been cases where deaths have oc-

curred despite all intensive care measures; hence prognostication should be done cautiously. Follow-up is critical for any residual lung damage of fibrosis or airway remodelling. It can also be noted that asthmatics are at greater risk of the intense inflammatory response to inhalants which are otherwise not harmful to the general population. Thus the probability of inhalational injury can be sought for in asthma exacerbations if no other cause is identifiable and the patient presents in severe distress.

Conflict of Interest

The authors declare no conflict or competing interests.

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References

1. Ashbaugh D.G., Biglow D.B., Petty D.L. and Levine B.E., Acute respiratory distress in adults, Lancet, 2:319-32, 1967.

2. Petty T.L. and Ashbaugh D.G., The adult respiratory distress syndrome-clinical features, factors influencing prognosis and principles of management, Chest 70:233- 239, 1971.

3. Sloane P.J., Gee M.H., Gottleib J.E., Albertine K.H., Peters S.P., Bums J.R., Machiedo G. and Fish J.E., A multicentre registry of patients with acute respiratory distress syndrome: Physiology and Outcome, Am. Rev. Respir. Dis., I46:4 19-426, 1992.

4. Kimmel EC, Still KR. Acute lung injury, acute respiratory distress syndrome and inhalation injury: an overview. Drug and chemical toxicology. 1999 Jan 1;22(1):91-128.

5. Bossmann K, Bach S, Höflich C, Valtanen K, Heinze R, Neumann A, Straff W, Süring K. Holi colours contain PM10 and can induce pro-inflammatory responses. Journal of Occupational Medicine and Toxicology. 2016 Dec;11(1):1-1

6. Gill P, Martin RV. Smoke inhalation injury. BJA Education. 2015 Jun 1;15(3):143-8.

7. Uemura K, Isono M, Kagohashi K, Hasegawa R, Satoh H. Bronchial damage and diffuse alveolar hemorrhage following chlorine gas inhalation: A case report. Experimental and therapeutic medicine. 2017 Nov 1;14(5):5126-8.

8. Hjortsø E, Ovist J, Bud MI, Thomsen JL, Andersen JB, Wiberg-Jørgensen F, Jensen NK, Jones R, Reid LM, Zapol WM. ARDS after accidental inhalation of zinc chloride smoke. Intensive care medicine. 1988 Jan;14(1):17-24.