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Nitrogen pneumonitis - A case of chemical pneumonitis with complications caused by inhalation of nitrogen gas

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ABSTRACT

Nitrogen oxides are low water soluble and can reach till distal airways and cause toxic effects when exposed to high concentration. It stimulates free radical generation, results in protein oxidation, lipid peroxidation and epithelial damage; and it reduces resistance to infection by altering macrophage and defence mechanism. Farmers who exposed to silos, fire fighters, and coal miners after firing of explosives, air conditioning workers are more prone to develop inhalation injury. Here we report a case of 17 year old male diagnosed as nitrogen gas related chemical pneumonitis presented with moderate ARDS and progressive hemorrahagic effusion. Oxygen therapy, Intercostal drainage and steroids were administered resulting remission of symptoms and radiological improvement. Patient was discharged and followed up.

Keywords: Pneumonitis, Nitrogen, Hemorrhagic Effusion, Corticosteroids.

1. INTRODUCTION

Nitrogen gas is a colorless, odourless inert gas contains two molecules of nitrogen. In atmosphere, N_2 occupies maximum percentage of composition. Nitrogen gas is used for production of fertilizers, explosives and to purge air conditioning system. Adequate protection is mandatory to use nitrogen gas since it has potential to explode when high concentration of nitrogen-nitrogen triple bond forms. It is a low water soluble and can reach till distal airways and cause toxic effects (Amaducci and Downs, 2021). Beyond the threshold limit value, Nitrogen gas can damage the mucus membrane and cause severe pulmonary disease like ARDS, bronchiolitis obliterans for which early corticosteroid treatment is recommend to reduce inflammation (Bur et al., 1997). Hereby, we report a case of chemical pneumonitis caused by inhalation of high concentration nitrogen gas and its management challenges.

2. CASE REPORT

A 17 year old male referred from outside hospital to our casualty with complaints of shortness of breath, cough with Blood tinged sputum and sustained injuries as nitrogen gas cylinder exploded in the work place (Air-Conditioner service centre). Explosion of nitrogen gas cylinder, while he performing AC leakage test using nitrogen gas without proper safety measures. Upon arrival to the emergency room he had dyspnea with laboured breathing, two episodes of hemoptysis and multiple injuries over right forearm, right sided chest wall, face and neck with no significant comorbidities. Oxygen therapy initiated immediately with Non-Rebreather mask, and his oxygen saturation was 93% with 10L/min of 02, BP-90/60 mmHg, Respiratory Rate – 28 breath/min. Heart rate- 122 beats/min. Temperature=98.5 F.

On clinical examination, patient was conscious, oriented to time, place and person. Abrasions over right sided chest wall, face and neck. Laceration over right forearm and right sided forehead and lateral angle of lip. On auscultation, Inspiratory crackles were heard over the right hemithorax and the left mammary, inter, and infrascapular areas. Cardiovascular and abdomen examination were normal and no specific neurological impairment. Patient was intubated and placed on mechanical ventilation in view of tachypnea, hemodynamic instability and facial injury where NIV cannot be administered. After suturing the wound and dressing he was shifted to ICU.

Arterial blood gas on arrival showed PH-7.42, $PaCO_2$-30mmHg, PaO_2-72 mmHg, HCO_3-21mmol/L. Chest X-ray on arrival showed alveolar opacities over right hemithorax and left perihilar region and left sided scoliosis present (congenital) (Figure 1). CT chest showed centrally distributed Ground glass opacity involving right upper, middle and lower lobe and left perihilar and lower lobe (Figure 2 & 3). This case was categorized as moderate ARDS by the berlin definition of 2012.TLC showed 17000 cells/ul, Serum LDH -380IU/L, CRP- 1.5mg/dl, serum procalcitonin- 0.15ng/ml; ECG, Liver function and renal function tests are within normal limits.

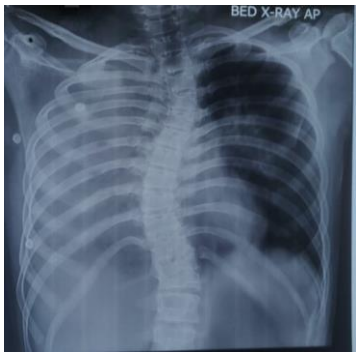

Figure 1 Chest X ray – Alveolar opacities over Right hemithorax and Left Perihilar region with scoliosis to left.

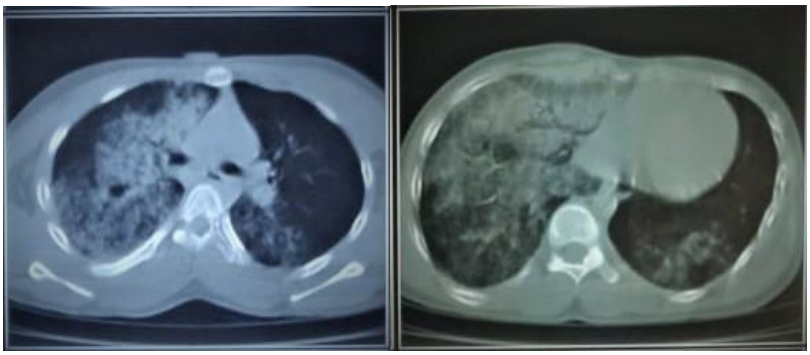

Figure 2 & 3 CT Chest shows – Centrally distributed Ground glass opacities involving Right upper, middle and lower lobe, and left Perihilar & Lower lobe.

Intravenous methyl prednisolone pulse therapy (250mg twice daily for 2 days) was initiated with empirical broad spectrum antibiotic coverage. On day 3, Patient physical and hemodynamic status improved drastically hence extubation done. On day 4, Inspite of clinical improvement, patient had developed progressive right sided pleural effusion which is observed through serial chest X-rays for which 28F intercostal drainage placed on right sided pleural space through right 5^{th} ICS (Figure 4, 5, 6 & 7). Around 900ml of hemorrhagic fluid was drained. On day 5, successful oxygen wean off achieved and IV Steroid were slowly tapered. On day 7, ICD removal was done after complete lung expansion with nil drain. Patient improved on both clinical and radiological basis

on day 10 hence, he was discharged and advised to continue oral prednisolone for a week. During follow-up, he had normal pulmonary function.

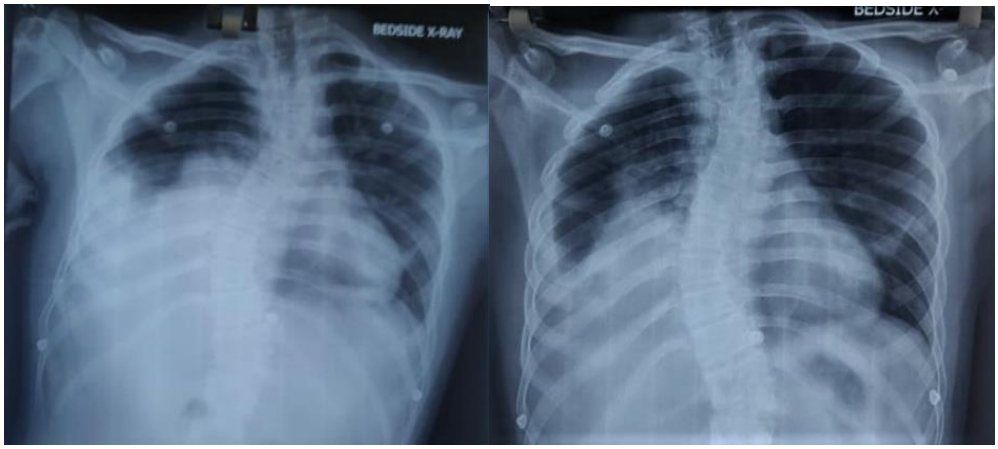

Figure 4 & 5 Chest X ray - Right sided Pleural Effusion

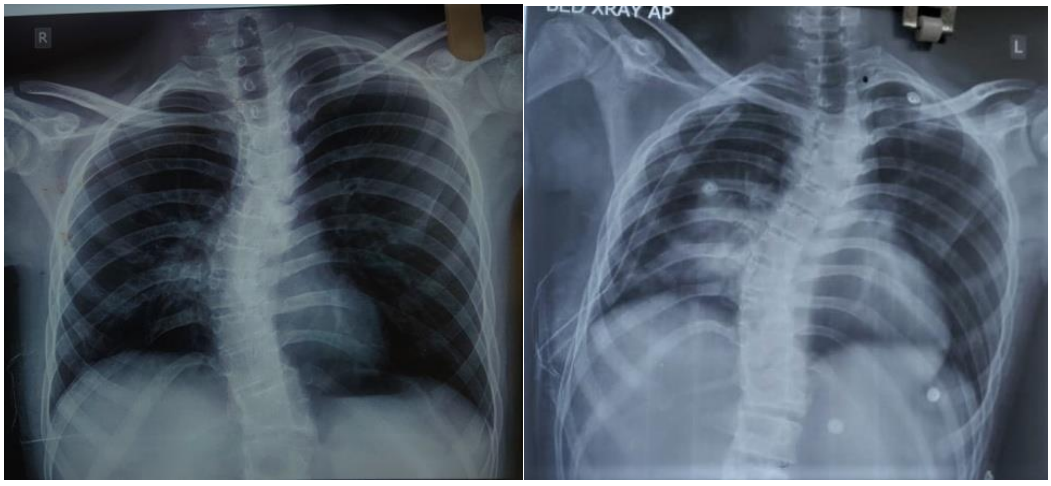

Figure 6 and 7 Chest X ray – ICD insitu, Right Perihilar opacity, Resolved pleural Effusion. Radiological resolution noted.

3. DISCUSSION

Chemical pneumonitis refers to inflammation of respiratory tract due to inhalation of various irritant substances, gases, metallic compounds and organic compounds (Murphy et al., 2010). Nitrogen combines with oxygen produce various nitrogen oxides which can enter into distal bronchioles and cause severe inflammation if exposed to higher concentration (more than 25 ppm -100ppm) Exposure more than 150ppm is usually fatal (Douglas et al., 1989). Chief exposure from inhalation of toxic NO_2 gas over silage called 'Silo fillers disease' commonly occurs among farmers. Other potential exposures from motor vehicle exhaust, air conditioning system (Jayalakshmi et al., 2009).

The reaction ($3NO_2+H_20=2HNO_3+NO$) occurs after inhalation. Through reactive nitrogen-derived free radicals, nitrogen dioxide and other oxides of nitrogen harm lung tissue (Masuda et al., 2000). The nitric acid has corrosive elements often responsible for inflammation causing acute lung injury in the form of pulmonary edema or hemorrhagical veolitis, airway denudation, subsequent proliferation followed by fibrosis (Amaducci and Downs, 2021). In our patient, hemorrhagicalveolitis resulted as hemorrhagic pleural effusion occurred after 24 hours of exposure.

In some patients, it can enhance its inflammatory response and cause hemodynamic instability also. The onset of signs and symptoms after exposure varies from few minutes to months based on concentration and duration of exposure (Lee et al., 2014). It is categorized as acute, subacute and delayed phase. Sudden death can occur due to severe laryngospasm and bronchospasm if exposed to very high concentrations. Exposure to lower concentration can often lead to bronchiolitis obliterans (Horvath et al., 1978). In our patient high concentration of nitrogen gas cause diffuses alveolar damage. Rarely, this may also be absorbed, results systemic toxicity.

The clinical manifestations of Nitrogen oxide-chemical pneumonitis are non-specific and include shortness of breath, cough, chest pain and hemoptysis. Diagnosis is usually suspected from the proper history and imaging. Characteristic CT findings are diffuse air-space consolidations/ground glass opacities (Bur et al., 1997). Treatment is usually symptomatic. Corticosteroids have a major role to reduce inflammation and limit fibrosis. In case of complications such as pleural effusion may need drainage of fluid or severe ARDS may require mechanical ventilation where lung protective ventilation strategies to be followed (Tanaka et al., 2017). In our case, even after administering steroid pulse therapy, patient had developed progressive pleural effusion after 24 hours drained by tube thoracocentesis but further accumulation of fluid or pulmonary fibrosis are prevented.

4. CONCLUSION

We conclude that, proper safety measures like facemask/face shield/PPEs are to be taken to avoid toxic gases inhalation. Affected individuals has to be treated with corticosteroids as early and effectively to prevent further complications.

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Authors' Contributions

All authors contributed equally to the manuscript.

Informed consent

Written & Oral informed consent was obtained from all individual participants included in thestudy. Additional informed consent was obtained from all individual participants for whom identifying information is included in this manuscript.

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Conflicts of interest

The authors declare that there are no conflicts of interests.

Data and materials availability

All data associated with this study are present in the paper.

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