La Prensa Medica Argentina

Case Report

Acute Necrotising Pneumonia with Bronchiectatic Changes and Transaminitis following Kerosene Ingestion: An Unusual Acute Presentation

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Rec date: June 02, 2014 **Acc date:** Sep 29, 2014 **Pub date:** Oct 03, 2014

Abstract

Hydrocarbon exposures are frequent and account for an inordinate number of health care visits and hospital admissions. There are very few case reports of bronchiectasis due to kerosene ingestion in literature. A 16 year old girl with history of kerosene ingestion presented with features of acute onset respiratory distress which resulted from bilateral pneumonia progressing to bilateral pleural effusion. It eventually leads to necrotising pneumonitis with mostly right lung bronchiectasis. It was controlled successfully with antibiotics and pulmonary physiotherapy. She had Transaminitis which resolved spontaneously.

After a hospital stay of 24 days the patient was discharged home and she was declared completely fit for daily activities 4 months later.

Key Words:

Kerosene poisoning, Necrotising pneumonitis, Bronchiectasis, Transaminitis

Introduction

Kerosene ingestion is the commonest form of acute childhood poisoning in most developing countries. Nearly 40% occur in children younger than 6 years. In Saudi Arabia, it accounts for 25% of accidental home poisoning [1], largely confined to the age group 1-5 years. [2,3,4,5,6] In one study from Riyadh [3], 80% of cases were from outskirts where kerosene is used for cooking and lighting purposes, and the kerosene kept in bottles is within easy reach of curious children.

Apart from central nervous system (CNS), gastrointestinal (GI), cardiovascular, hepatic, renal, hematologic and cutaneous toxicities, fatal chemical pneumonitis deserves special mention. But necrotising pneumonitis with bronchiectasis along with hepatic transaminitis has not been reported till date in literature.

Case report

A 16 years old girl, presented with the chief complaint of acute onset respiratory distress following accidental ingestion of Kerosene oil as told by the relatives. Further enquiry revealed that she had inhalational abuse as well. A perineal scald was also present.

On general survey patient was conscious, febrile, and tachypneic with respiratory rate 40/ min, tachycardia with pulse rate of 126/ min, normotensive and accessory muscles of respiration working. Her oxygen saturation of peripheral blood (SpO2) value was in the range of 92%-94% in room air.

Chest examination revealed bilaterally coarse crackles suggestive of chemical pneumonitis which was evident in Chest skiagram (PA view) with affection of right lung > left lung.

Investigations showed a total leukocyte count of 26,400/cu.mm (83% neutrophils), with toxic granules present and leftward shift of neutrophil series. Arterial blood gas analysis revealed mild hypoxemia (Partial pressure of oxygen in arterial blood/PaO_2 → 70mm of mercury, partial pressure of carbon dioxide in arterial blood/ $PaCO_2$→30mm of mercury) respiratory alkalosis with partially compensated metabolic acidosis and hypokalemia.

While cultures of Blood, urine and sputum were awaited, she was started empirically on clindamycin (600mg) in intravenous (IV) route thrice daily (TDS), piperacillin + tazobactum (4.5gm) IV TDS, amikacin (1gm) IV once daily (OD) and supportive care.

Associated there was transaminitis with raised AST, ALT (AST→180 IU/ L, ALT→220 IU/ L) and fever peaked to 106^0F even after 3 days of the above mentioned antimicrobials. Thereafter, clindamycin was replaced by vancomycin (dose -1 gm. 12 hourly in 100ml normal saline over 90 minutes). Though this regimen showed response evident by a decreasing TLC to 20,000/ cum but fever persisted at same degree.

Now antibiotic was stepped up to meropenem (1gm) IV 8hourly , linezolid (600mg) IV 12 hourly, netilmicin 300mg stat and 150mg OD and metronidazole 500mg/ 100 ml TDS after 3 days though all the culture reports came out negative.

Sputum was again sent for gram stain, fungal smear, aerobic and anaerobic culture and sensitivity. Dual malaria antigen and peripheral smear for malaria parasite were also sent.

A high resolution CT scan (HRCT) of thorax interestingly revealed bronchiectatic cavity with superadded infective changes in right lung. Extensive scattered fluid attenuation lesion in alveolar space was seen in right lower lobe and segment of right upper lobe. Cavitary lesion was found in the anterior segment of left upper lobe. Mediastinum was within normal limits. Mild pleural effusion was noted in right mid and lower zone and also in left lower zone. With the changed antibiotic regimen patient was clinically better but high grade fever persisted. Again urine was reviewed for culture sensitivity and fungal smear. However as evident by HRCT, it was necrotising pneumonitis with bronchiectasis mostly involving right lung.

Now we decided to do a fibreoptic bronchoscopy and obtaining bronchoalveolar lavage (BAL) fluid. We did so under topical airway anesthesia, sedation with propofol and analgesia with fentanyl. BAL fluid was sent for gram stain, fungal stain, *Ziehl–Neelsen* (ZN) stain, aerobic, anaerobic and fungal culture.

Now she was maintained on an Antibiotic regimen of meropenem, linezolid, metronidazole and aztreonam (2gm) IV 8 hourly. However the fever which was persistently rising to 106^0F- 104^0F returned to an average of 100^0F with a single spike per day after 10 days of admission. Associated chest care by nebulisation with salbutamol, ipratropium bromide and budesonide continued along with oxygen therapy. Perineal scald was healing after postural care, betadine and antiseptic therapy. Liver enzymes came to base line after 13 days. Patient was started on soft nutritious diet while we awaited the BAL fluid reports. Finally the BAL fluid report revealed huge pseudomonas and anaerobic organism growth sensitive to the drugs we conventionally administered.

Ultimately we could discharge the patient from intensive care in afebrile condition with mild cough on 17th day. 7 days later she was discharged home and on weekly review she was getting much better as far as functional capacity is considered. HRCT thorax done 4 months later was absolutely normal.

Discussion

Vast majority of Kerosene poisonings are accidental. In adolescents and adults, poisoning generally results from inhalational abuse, occupational exposure, intentional ingestion or accidental aspiration during the siphoning of fuels. Our case was intentional oral ingestion following inhalational abuse. 33% of cases develop no symptoms. Cough is the most common symptom in 36% and central nervous system toxicity manifest in 10%, abdominal symptoms in 41% [3] .Death may develop in the seriously ill children in 1-3%. [3,4,5,6]

Pulmonary toxicity results directly from aspiration of even small amounts rather than from haematogenous spread after GI absorption. The potential for aspiration is determined by physical properties of viscosity surface tension and volatility.

Substance with Saybolt Seconds Universal (SSU) value less than 60 indicating a low viscous hydrocarbon which is kerosene oil has a high aspiration potential. [7] Reduced surface tension and high volatility enhances toxic effects.

Signs and symptoms

Aspirated petroleum distillates, inhibit surfactant, resulting in alveolar collapse, ventilation-perfusion (V/Q) mismatch, and subsequent hypoxemia. In addition, bronchospasm and direct capillary damage lead to a chemical pneumonitis and haemorrhagic bronchitis-alveolitis [7,8,9].

Hepatic toxicities depend on the specific toxic substance involved and may present with transaminitis as in our case. Deaths associated with inhalational abuse may result from coma with respiratory depression, aspiration or injuries incurred while intoxicated as well as from cardiac arrhythmias. [10] Patients who aspirate generally demonstrate symptoms within 30 minutes as was our case, those who do not have symptoms within 6 hours of exposure remain asymptomatic. [11]

Initial coughing , gasping and choking may progress and peak during the first 24 to 48 hours to tachypnea with grunting respirations, nasal flaring, retractions and cyanosis.[11,12] Wheezing, rhonchi and rales may be heard on auscultation as was our case. In severe cases, pulmonary edema and haemoptysis occur. Arterial blood gases may demonstrate hypoxemia from V/Q mismatch and early hypocarbia which was found in our case; but it progresses ultimately to hypercarbia and acidosis.

Chest film abnormalities occur in up to 75% of hospitalized patients, appearing within 2 hours in 88% of patients and by 12 hours in 98%. [12,13,14] Radiographic abnormalities and symptoms may be delayed several hours after ingestion and typically peak by 72 hours.

In other few studies chest X-ray changes were reported in 30-60% of children [2,6,13] , pleural effusion in 4% , pneumatocele in 1-8%. [15,16,17,18]

Early radiographic abnormalities include unilateral but more commonly bilateral basilar infiltrates and fine punctate perihilar densities. Localised areas of atelectasis are often present, whereas pleural effusion, pneumatocele and pneumothorax occur infrequently. [13, 14, 19,20]

Pneumatocele is an uncommon complication of kerosene ingestion and may occur in 8% of cases. [15,16,17,18] The pneumatocele usually involve the medial segments of left lower lobe followed by right lower lobe and right middle lobe [18] and usually bilateral and subpleural in position. Pneumatoceles generally occur 3-15 days after ingestion and resolve over 15 days-21 months .[8,17] Radiographic findings correlate poorly with clinical symptoms, lag behind clinical improvement and may persist for several days to weeks after symptoms have resolved [13,17,19] as was our case in follow up phase.

Asymptomatic patients may have abnormal chest film, whereas symptomatic patients may have minimal or no radiographic abnormalities early in the course. [12, 21] Within the first 24-48 hours, fever (100-1040F) and leukocytosis is common [11], which was in our case.

The persistence of fever beyond 48 hours suggests bacterial superinfection as was in our case.

Nausea, vomiting, abdominal pain though are common but hematemesis, melena are rare [21] and transaminitis is not that common.

Dizziness, lethargy (91%), somnolence, may occur with rare occurrence of coma and convulsions. [12,22] Aliphatic hydrocarbons are highly flammable and accidental thermal burns may occur during recreational use. [23] Therefore patients with unexplained burns should be questioned regarding possible inhalation abuse as was our case.

Gastric decontamination is not recommended because absorption is minimal and induced vomiting increases risk of aspiration and pneumonitis. [22,24,25]

Treatment

After basic resuscitation protocols, and attaching all the routine monitors, the specific treatments are done. Acid-base, fluid-electrolyte balance, serial arterial blood gases or pulse oxymetry, chest radiograph evaluation, complete blood counts, liver function tests, sugar/urea/creatinine, serial tracheal aspirate culture/sensitivity are to be followed strictly.

Antibiotics (e.g. penicillin or clindamycin) should be given to patients with documented bacterial pneumonias or worsening chest radiograph, leucocytosis and fever after first 40 hours. [12,21] Hence our patient was kept under broad spectrum antibiotics with anaerobic coverage.

Animal and clinical investigations have failed to demonstrate any beneficial effect of steroid treatment [26,27,28], rather they may be harmful. [29,30,31] Hence steroid was not administered in our case.

Singh et al [32] reported in a randomised trial in 100 children with accidental kerosene ingestion that ampicillin and metronidazole is superior to treatment with either drug alone.

Mahdi et al [1] used ampicillin or penicillin in all cases with bilateral infiltrate in chest X-ray and had good results. Banjar [33] suggests addition of an antibiotic against anaerobic bacteria like clindamycin or metronidazole in cases with severe pneumonic infiltrate and history of vomiting with suspected aspiration. Successful use of high frequency oscillatory ventilation (HFOV) and extra corporeal membrane oxygenation (ECMO) for treatment of respiratory failure has been reported. [34,35,36] Partial or full cardiopulmonary bypass procedures and partial liquid fluorocarbon ventilation should also be considered in patients with intractable respiratory failure.

Apart from all, postural drainage and pulmonary physiotherapy are of extreme help for bronchiectasis management. In our case, antibiotic regimen was institutionalized according to patient's symptoms, signs and investigations but unremitting prolonged fever with persistent crackles and chest symptoms was the reason why BAL fluid was ultimately sent and judiciously antibiotic therapy was used as per protocol.

Conclusion

Though Kerosene ingestion is still a common accidental poisoning in children less than 5 years of age, vast majority recover fully with supportive care. However follow up care with pulmonary function testing should be considered. When appropriate the patient should receive psychiatric evaluation and poison prevention education before final disposition.

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