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SUCCESSFUL MANAGEMENT OF SEWAGE GAS POISONING

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ABSTRACT

Management of poisoning cases is still a big challenge to the critical care field. These poisons could be accidental, suicidal or homicidal. And in many cases antidotes are not there or they are not rapidly available. And most of the time victims are brought to critical care in mentally obtended state where the history is not even available. And one of the highly fatal type of poisoning is sewage gas poisoning. Victims scum to the toxic substance or due to secondary phenomenon of multi-organ failure.

INTRODUCTION:

Sewage gas is by byproduct of human waste. Primary components include H2S, NH3,CO2. It is not toxic at low levels. However chronic exposure or higher levels of exposure can cause symptoms of gas poisoning and can be rapidly fatal. H2S being main culprit. In high levels of exposure symptoms include: eye- throat irritation,CNS depression (>500ppm) ,seizures, cardio-respi depression, coma and even death immediately (>1000ppm).1,2 We hereby present 2 cases of successful management of sewage gas poisoning in our government setup critical care unit.

CASE REPORT:

For manual scavenging 3 sanitary care workers entered a sewer. Just on opening the lid of sewer they became unconscious and fell down. They were pulled out by their co-workers. One person died on the spot, while other 2 were taken to local hospital. Their GCS was poor < 8 were referred to our hospital. On being received in icu both were in altered mental sensorium with frothing from mouth.

CASE REPORT 1: 28 Yr /M presented in unconscious state and seizure like activity, had episode of hemoptysis in emergency. CVS: tachycardia present PR- 120/mint ,BP- 126/80 mmhg . Respiratory: RR>42/mint, spo2-92%on NRM @15L/min , on auscultation: B/L coarse crepts were present. Pupils: NSNR . GCS was E2V2M3.

Patient was immediately intubated and put on ventilator AC-VCV mode. ABG report showed acidosis PH 7.28 ,Pco2 -36.9, PO2- 147 , HCO3-17.4 , lact- 1.62 with Fio2 -100%. Chest x-ray shows B/L infiltrates. Diagnosis of acute sewage gas poisoning with chemical pneumonitis leading to ARDS was made based upon chest findings, ABG and CXR. ECG and echo was normal on the day of admission to the icu.

Critical illness management promptly started. Initial ventilator settings- FiO2: 100% PEEP:7 RR: 14 TV 6ml/kg. As ARDS is acute inflammatory state and early methylprednisolone is helpful, so infusion was started @ 1mg/kg/day. Dvt prophylaxis was given with LMWH.

On third day pt started desaturating ,on auscultation B/L silent chest and Bronchospasm was diagnosed. Spo2 :91%, infusion aminophy line started @0.5mg/kg/hr with ongoing methyl prednisol one infusion. Patient shifted to prone position for 18 hrs alternatively with inverse ratio ventilation and serial ABGs were done . ABG report

showed PH-7.2,PCO2-88.8,PO2-64,HCO3-40.2, lact-1.34 with FiO2-90%. Sedation started with infusion dexmedetomidine.

After 48 hrs, FiO2 decreased to 60% , repeat ABG showed improvement PH-7.56,PCO2-44.7,PO2-187,HCO3-35.4,lact-0.70. Infusions aminophyline, methylprednisolone and dexmedetomidine were reduced. On auscultation conducted sounds were present, for which chest Physiotherapy and nebulisation was done. Alongside RT feed was started.

Patient started improving clinically maintained spO2 on FiO2-40% ,PEEP requirement decreased put on SIMV-VCV mode and slowly weaned off , extubated and put on Bipap. Supportive management was ongoing- nebulisation, chest physio, spirometry, oral feed. Patient started maintaining on NRM then O2 face mask , finally brought on room air and shifted to ward in satisfactory condition after 15 days.



Day 2:

Day 4:



Day 10:



CASE REPORT 2: 24 Yr/M , presented in altered mental sensorium. On examination CVS: PR-116/min,BP -110/82mmhg ,SpO2-92% on NRM @15 L/min. Respiratory:RR-35/min, on auscultation B/L coarse crests present.Pupils: B/L NSNR , GCS:E2V2M2

Patient intubated immediately put on AC-VCV mode of ventilator. ABG :PH-7.203, PO2-75.1,PO2-90 ,HCO3-29. Infusion methylprednisolone and for sedation dexmedetomidine started. CXR-showed diffuse infiltrates. ECG and echo were normal.

On third day sedation stopped, patient put on SIMV-VCV. Patient responded to vocal commands weaned off from ventilator and extubated ,put on BiPAP. ABG improved PH:7.45, PCO2-42.3,PO2-95,HCO3-29.5, lac- 1.22. Supportive management was given : nebulisation, spirometry, adequate hydration. Patient brought on room air and shifted back to ward in satisfactory condition after 6 days.

DISCUSSION:

Sewage gas poisoning diagnosis can be made based upon history of exposure and clinical examination. In our case, 3 of the workers immediately became unconscious on entering the sewer. One out of them died on the spot. That means they were exposed to higher gas concentration levels. Exposure can lead to cardiac systolic dysfunction, chemical pneumonitis or neurotoxicity.3In our case respiratory system was involved mainly leading to ARDS and neurological symptoms. This occurs because of direct airway exposure to toxic inhalation gases leading to alveolar oedema. Patient recovered from ARDS – radiographic improvement on CXR, ABG and clinical improvement in RR and chest findings .

ARDS supportive management was done- steroid therapy, prone positioning, intravenous fluid balance, antibiotic therapy, aminophyline infusion for reactive airway. Chest Physiotherapy, postural drainage was done. Nutrition maintained through RT feed. Nitrite was procured very late so was not used.3,4,5

CONCLUSION:

Early definitive diagnosis and icu shifting with appropriate pharmacological treatment along with different modes of mechanical ventilation led to the successful management of sewage gas poisoning.

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