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Case Report

Fatal Hair Dye Poisoning

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Abstract

Suicide is the 10th leading cause of death worldwide with an increasing trend and accounted for 187,000 deaths in 2010 in India. Super Vasmol 33™ is a low cost, freely available, emulsion based hair dye used with main components of Paraphenylene Diamine (PPD), resorcinol, propylene glycol and sodium Ethylene Diamine Tetra Acetic Acid (EDTA). We report hair dye poisoning in a 50 year old male patient who consumed around 50 ml of Super Vasmol 33™ and expired on day 3. PPD component of hair dye can cause rhabdomyolysis, laryngeal edema, severe metabolic acidosis, Acute Kidney Injury (AKI) and myocarditis. No known specific antidote is available at present and treatment is supportive. Public awareness of the toxic potential and regulating the PPD concentration in hair dyes is to be done.

Introduction

Suicide (Latin suicidium, from Sui caedere, "to kill oneself") is the act of intentionally causing one's own death. Commonly used methods of suicide vary worldwide and are partly related to availability. Common methods are hanging, pesticide poisoning, and firearms. Suicide is the cause of 842,000 deaths in 2013 compares to 712,000 deaths in 1990 [1]. It is the 10th leading cause of death globally [2,3]. Globally, more than a million deaths occur per year due to suicides of which 20% are Indians [4], even though they form 17% of the world population. Suicide attempts are up to 20 times more frequent than completed suicide [5] and account for an estimated 10 to 20 million every year [6].

Super Vasmol 33™ is a low cost, freely available, emulsion based hair dye used in India. Main components of the dye are Paraphenylene Diamine (PPD), resorcinol, propylene glycol, sodium Ethylene Diamine Tetra Acetic Acid (EDTA), preservatives and perfumes. PPD is the main toxic ingredient and is used worldwide as a key ingredient in hair dye formulations to produce various shades using different concentrations. PPD Concentration in hair dyes ranges from 0.2% to 3.75% that gives color from golden blond to black [7]. PPD appears as white crystals when it is pure and turns rapidly to brown when exposed to air [7]. Application of henna (prepared from leaves of nontoxic herb - labwsonia alba) & PPD called black henna accelerates dyeing process in 1-2 hours compared to 2-12 hours when henna used alone.

In Morocco, a nontoxic herbal extract known as "takast" is extracted from the gallnut of athl-pine (Tamarix aphlya) is used as hair dye. First artificial dye was synthesis in the laboratory was done in 1856 and usage of PPD started in 1883. PPD is mixed with Hydrogen peroxide causing an oxidation hair dye product [8].

Clinical manifestations of PPD poisoning usually progress in three phases. Phase 1: Acute presentation causing edema of neck, airway obstruction, severe vomiting and gastritis. Phase 2: Subacute presentation causing Acute Kidney Injury (AKI), rhabdomyolysis [9] and hemolysis. Phase 3: progression to Multiorgan Dysfunction Syndrome (MODS) and death. Systemic toxicity occurs in a dose dependent manner [10]. Seizures can occur due to effect of toxins in dye or hypocalcaemia. Symptomatic treatment is the only choice as there is no specific antidote. We report hair dye poisoning in a 50 year old male patient who consumed around 50 ml of Super Vasmol 33™ and expired on day 3.

Case Report

A 50 year-old man was bought to emergency room in a state of unresponsiveness to pain stimuli and Glasgow Coma Scale (GCS) was noted to be 3/15. Patient was immediately resuscitated as per ACLS guidelines with Cardio-pulmonary resuscitation for a period of 45 min. and adrenaline injections. Initially patient was given ambu bag ventilation with two handed jaw thrust technique. After revival, Bougie- assisted endotracheal intubation was done with difficulty due to laryngeal edema and spasm. Patient was shifted to Intensive care unit and was given mechanical ventilation.

History from relatives revealed consumption of around 50 ml of Super Vasmol 33™ 2 hours ago

in home. Past history is significant for diabetes mellitus and ischemic heart disease from past 5 years with non-compliance for treatment. Clinical examination showed facial swelling with edematous lips, swollen neck and tongue. Physical examination post resuscitation was remarkable for fever (40°C), hypotension (BP: systolic 70 mm of hg), cyanosis and other signs of Systemic Inflammatory Response Syndrome (SIRS), including no respiratory effort and increased heart (142/min) rates.

Systemic examination revealed bilateral coarse crepitations and abdominal distension. Chest radiography showed extensive alveolointerstitial infiltrate compatible with Acute Respiratory Distress Syndrome (ARDS). Patient was started on prophylactic antibiotics, inotropes and ryle's tube insertion. Patient had decreased urine output (10ml over 6 hours) with dark colored urine. Investigations revealed total white blood cell count of 48,100 /mm^3 (neutrophils 74%, lymphocytes 20%, eosinophils 3%, monocytes 3%) ESR 40mm/hr.), hemoglobin of 14mg/dl and platelet count was 135,000/microliter. The Arterial Blood Gas analysis (ABG) showed pH of 7.31, a PO2 of 50 mmHg, PCO2 of 39.5mmHg and 20.9 mmol/l of bicarbonate. Renal function test was abnormal with creatinine of 6.6mg/dl and blood urea of 140mg/dl and potassium of 7.6 mEq/l. Liver function test was abnormal with elevated transaminases. ECG showed tachycardia and ST –T changes in leads 3 and a VF.

Creatine Phosphokinase (CPK) levels were 3490 U/L, serum calcium 7.1 mg/dl, and urine myoglobin was positive. Patient was subjected to continuous renal replacement therapy (hemodialysis), calcium correction and hydrocortisone in view of respiratory distress and hypotension. Condition of patient deteriorated on 2nd day with oliguria, shock and Multiple Organ Dysfunction Syndrome (MODS). Inspite of the efforts, patient expired on day 3 due to MODS.

Discussion

PPD is derived from paranitroaniline which is highly toxic and used extensively in industrial products, textile or fur dyes, colored cosmetics, tattoos, photography, lithography, photocopying and printing inks, black rubber, oils, greases & gasoline. It is produced through oxidation by mixing with hydrogen peroxide. PPD can be absorbed from skin causing irritation and is used without safety standards in Africa, Arab countries and India. First report of poisoning was described by Nott in 1924 in a hair salon owner [11]. The exact concentration that can cause toxicity is not known. It is reported that 3gms consumption can cause systemic complications. Lethal dose is 7-10 gm and most deaths occur within 24 h of consumption with a mortality rate of 47%.

PPD molecular weight is 108.15 g/mol and lethal dose is 0.028 mg/l. Propylene Glycol lethal dose is 1gm/l. EDTA (Ethylene Diamine Tetra Acetic Acid) lethal dose is 2.0–2.2 g/kg body weight [12]. Renal involvement of PPD can vary from transient proteinuria to Acute Kidney Injury (AKI). Mechanisms of injury are 1) Direct toxic effect due to its aromatic structure leading to easy reabsorption and concentration in tubule and causing ARF [13]. 2) Rhabdomyolysis with deposition of myoglobin in the renal tubules and hemolysis causing hemoglobinuria which in turn result in Acute Tubular Necrosis (ATN) and Acute Renal Failure (ARF) [14]. 3) Propylene glycol results in a high anion gap metabolic acidosis and ARF due to its nephrotoxic potential [8].

Phase I symptoms occur within 4 to 6 hours [14,15]. 5 to 10 ml can result in laryngeal edema due to direct toxic effect on mucous membranes by PPD. Rapid development of face, neck, pharynx & larynx edema along with respiratory distress. Tongue appears dry &wooden - hard & swollen due to edema. Tracheostomy may be required due to difficulty in intubation [15]. Phase II symptoms occur from days to weeks [15]. Chocolate brown colored urine due to methemoglobinemia. Rhabdomyolysis, Acute Tubular Necrosis (ATN), arrhythmias and intra vascular hemolysis can occur. Common cause of death reported is due to acute renal failure in turn because of rhabdomyolysis [16].

There is no known antidote for PPD Poisoning. Supportive treatment includes 1.Water or milk. 2. Tracheostomy. 3. Hemodialysis 4. Oxygen inhalation or mechanical ventilation. 5. Calcium correction in case of hypocalcaemia. Hemodialysis is ineffective in removing the toxin. Diagnosis is done by demonstration of myoglobin in urine and increased serum creatine phosphokinase and aldolase. Myoglob in with a molecular weight of 17 KD a binds only lightly to the plasma proteins and is excreted in urine.

The European Union restricted PPD concentration in hair dye to 6% maximum, while in Asian and African countries, it is available up to a concentration of 97%.

Conclusion

Suicide by hair dye consumption (especially Super Vasmol 33¨) is an emerging mode of self-harm in developing countries due to easy availability and low cost. Early management of rhabdomyolysis and acute kidney injury with supportive treatment may improve outcome in lower dosage hair dye poisoning. Public awareness of the toxic potential and regulating the PPD concentration in hair dyes is to be done.

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