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Paraphenylenediamine Poisoning Causes by Herbal Extracts, Preservatives, and Perfumes User as Daily Cosmetics: A Comprehensive Study

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Any substance dangerous to living organisms when either applied internally or externally, destroy the action of vital functions or prevent the continuance of life is known as poison. Cosmetics, medicines, and other household items could lead to a serious poisoning if accidently consumed by children. The home environment is the primary location of most poisonings, with over 90% of reported poisonings occurring at home. Most non-fatal poisonings occur in children younger than six years of age. Paraphenylenediamine (< 4%), resorcinol, propylene glycol, liquid paraffin, cetostearyl alcohol, sodium lauryl sulfate, EDTA sodium, herbal extracts, preservatives, and perfumes. Paraphenylene diamine and resorcinol are known toxicants with multi-organ effects, while the toxicity profiles of others are not known. It causes systemic toxicity, manifested by severe edema of neck and face and laryngeal edema with respiratory distress frequently requiring emergency tracheostomy and mechanical ventilation. It also causes rhabdomyolysis and acute renal failure, culminating in death if not treated aggressively. The controlled supervision over trading of hair dye is necessary to stop Paraphenylenediamine poisoning. We recommend that the selling of hair dye containing PPD should be controlled and public education programme should be initiated in this regard.
Keywords: *Cosmetics, Paraphenylenediamine, poisoning*

I. Introduction

Intentional application of poison has used as a method of assassination, murder, suicide, and execution [1]. The history of poison stretches from before 4500 BC to the present day. Poisons have been used for many purposes across the span of human existence, most commonly as weapons, anti-venoms, and medicines [2]. Poison has allowed much progress in branches, toxicology, and technology, among other sciences [3]. It was discovered in ancient times, and was used by ancient tribes and civilizations as a hunting tool to quicken and ensure the death of their prey or enemies. This use of poison grew more advanced, and many of these ancient peoples began forging weapons designed specifically for poison enhancement. Later in history, particularly at the time of the Roman Empire, one of the more prevalent uses was assassination. Any substance dangerous to living organisms when either appliedinternally or externally, destroy the action of vital functions or prevent the continuance of life is known as poison. As per law, any substance, irrespective of its quality or quantity, when given with an intention to endanger, injure, or kill a person is called a poison[4]. Biologically speaking, any substance if given in large enough amounts is poisonous and can cause death. The study of the symptoms, mechanisms, treatment and diagnosis of biological poisoning is known as toxicology [5].

The epidemiology of poisoning was studied from various perspectives. It includes overall mortality, hospital admission rates, and enquiries to poisons Information Services. Accidental poisoning is most common in children, but deliberate self-harm becomes predominant in teenage years and early adulthood.Nearly one million people die each year because of suicide, and chemicals account for a significant number of these deaths[6]. The dangers of poison exposure exist even in our own home. Cosmetics, medicines, and other household items could lead to a serious poisoning if accidently consumed by children. The home environment is the primary location of most poisonings, with over 90% of reported poisonings occurring at home. Most non-fatal poisonings occur in children younger than six years of age[7].

General Management of Poisoning:

1. Removal of unabsorbed poison - cleaning, suction, stomach wash;
2. Antidote for neutralizing absorbed poison - activated charcoal, chelates;
3. Removal of absorbed poison and its intermediary substances - catharsis, enema;
4. Symptomatic relief[8].

It is an emulsion based hair dye commonly used in India.Paraphenylenediamine(< 4%), resorcinol, propylene glycol, liquid paraffin, cetostearyl alcohol, sodium lauryl sulfate, Ethylenediaminetetraacetic acid (EDTA) sodium, herbal extracts, preservatives, and perfumes.Paraphenylenediamine(PPD) and resorcinol are

known toxicants with multi-organ effects, while the toxicity profiles of others are not known.Accidental or suicidal ingestion of PPD causes systemic toxicity, manifested by severe edema of neck and face and laryngeal edema with respiratory distress frequently requiring emergency tracheostomy and mechanical ventilation. It also causes rhabdomyolysis and acute renal failure, culminating in death if not treated aggressively. Resorcinol is a Phenolic chemical used in photography, tanning and cosmetics (hair dye) industry. It is also a pharmaceutical agent used topically in skin diseases. Resorcinol is a moderately toxic and corrosive chemical. After oral administration, resorcinol is readily absorbed from the gastrointestinal tract, metabolized, and excreted by male and female rats, indicating little potential for bioaccumulation in animal tissues. It is known to cause eye, skin, oral and gastrointestinal injuries. Systemic toxicity is manifested as vomiting, dyspnea, methemoglobinemia, hypothermia, tachypnea, pallor, profuse sweating, hypotension and tachycardia.Rhabdomyolysis means destruction or disintegration of striated muscle. This syndrome is characterized by muscle breakdown and necrosis resulting in the leakage of the intracellular muscle constituents into the circulation and extracellular fluid. Rhabdomyolysis ranges from an asymptomatic illness with elevation in the creatine kinase (CK) level to a life threatening condition associated with extreme elevations in CK, electrolyte imbalances, acute renal failure and disseminated intravascular coagulation. The cause of rhabdomyolysis is usually easily identified; however, in some instances the etiology is elusive. Muscular trauma is the most common cause of rhabdomyolysis. Less common causes include muscle enzyme deficiencies, electrolyte abnormalities, infectious causes, drugs, toxins and endocrinopathies. Rhabdomyolysis is commonly associated with Myoglobinuria, and if this is sufficiently severe, it can result in acute renal failure. Weakness, myalgia and tea-colored urine are the main clinical manifestations.The most sensitive laboratory finding of muscle injury is an elevated CK level. In the absence of myocardial or brain infarction, CK > 5000 U/l indicates serious muscle injury.

The management of patients with rhabdomyolysis includes advanced life support (airway, breathing and circulation) followed by measures to preserve renal function and the latter includes vigorous hydration. The use of alkalizing agents and osmotic diuretics, while commonly used remains of unproven benefit[9].

Bourquia et al.[10]conducted a review on Systemic toxicity of Paraphenylenediamine. 4-cases, seen in the nephrology department, concerning women aged from 18 to 35 years who had tried to commit suicide by drinking varying amounts of that hair dye. The initial symptom was acute asphyxia, which required emergency tracheostomy in three cases. Thereafter, the most important visceral damage was acute renal failure, usually with oliguria or anuria, for which hemodialysis was performed in two cases. In these patients treated at an early stage, the mid- and long-term prognosis was satisfactory. Rhabdomyolysis is the principal mechanism underlying PPD systemic toxicity; it is in particular, responsible for the renal failure observed.Averbukh et al.[11]conducted a study on rhabdomyolysis and acute renal failure induced by Paraphenylenediamine and concluded that paraphenylenediamine may cause rhabdomyolysis resulting in acute renal failure in humans.Saito et al. [12]presented Rhabdomyolysis due to Paraphenylenediamine (hair dye) reported an autopsy case, a 44 years old, previously healthy male, drinking a beverage containing PPD, prepared for a homicidal use. Total intake of PPD was about 3 g (63 mg/kg). Principal clinical manifestation of the patient was muscle rigor with tenderness, initially developed at the lower extremities and subsequently extending to all over the skeletal muscles. Laboratory examinations disclosed high CPK (137,600), LDH (3895), GOT (3400) and GPT (545), and leukocytosis (26600), indicating massive skeletal muscle necrosis. ECG revealed mild depression of ST junction in the II and aVF leads. Urine showed dark brownish discoloration and diminished in volume subsequently. Scattered necrosis of muscular fibers was observed in a biopsy of the femoral muscles. The consciousness was rather clear during the course. The patient collapsed and soon died in the course of about 30 h. Clinically, the cause of death was thought to be acute renal failure due to rhabdomyolysis. Afterwards PPD was detected in the urine obtained in the hospital. Autopsy confirmed the clinical diagnosis; Renal collecting ducts and distal tubules were occluded by dark brownish myoglobin casts and its epithelium massively necrotized; skeletal muscles showed scattered coagulation necrosis and were partially associated with inflammatory cell infiltration. Lie et al.[13] conducted a study on Rhabdomyolysis in self-induced poisoning. A prospective study of 103 patients hospitalized for self-poisoning the incidence of rhabdomyolysis (creatine kinase > 1000 U/l) was nearly 7%. A further 9.7% had elevated creatine kinase activity, but lower than 1000 U/l. Two patients showed clinical symptoms of rhabdomyolysis at time of admission; one after heroin and the other after salicylate intoxication. Both developed renal failure, and one of them underwent peritoneal dialysis. The high incidence of rhabdomyolysis found in the study suggests that creatine kinase activity should be considered in all cases of intoxication admitted to hospital. Rhabdomyolysis may often present no symptoms even in conscious patients, and serious complications can be limited by preventive measures if rhabdomyolysis is recognized early. Shemesh et al. [14] conducted a study on Rhabdomyolysis in Paraphenylenediamine intoxication. They noticed edema of the upper airways and rhabdomyolysis developed in a young patient because of Paraphenylenediamine poisoning. Treatment with adrenaline, steroids and enforced diuresis prevented tracheostomy and renal failure. Zeggwagh et al. [15] reported a case on left ventricular thrombus and

Myocarditis induced by Paraphenylenediamine poisoning. They showed that the Paraphenylenediamine (PPD) poisoning caused Myocarditis but there is no data on the echocardiographic features. They report a case of Myocarditis induced by PPD poisoning with echographic data. After ingestion of 5 g of PPD, an 18-year-old woman was admitted to the hospital with asphyxia and rhabdomyolysis. An electrocardiogram showed ventricular extra systoles and negative T waves. The serum concentration of CK was 28,020 UI l (-1) (iso-enzyme MB = 840 UI l (-1)). A transthoracic echocardiography showed significant left and right ventricular hypokinesis (shortening fraction = 20% and left ventricular ejection fraction = 35%) and a left ventricular apical thrombus. Anticoagulation treatment with heparin was initiated. A follow-up echocardiogram performed on the 15th day showed normalization of ventricular function and disappearance of the thrombus. No embolic event was noted. Echocardiography is indicated in the Myocarditis induced by PPD poisoning to prove the ventricular function as well as the presence of a thrombus. Case report on acute renal failure following PPD poisoning: a case report and review, systemic poisoning with PPD by Anuradha et al., [16] presenting with characteristic features of severe angioneurotic edema, rhabdomyolysis and intravascular hemolysis with hemoglobinuria culminating in acute renal failure. Though rare in western countries, such poisoning is common in East Africa, Indian subcontinent and Middle East countries. They discussed the clinical features and key management issues of systemic PPD poisoning. Kallel et al. [17]conducted a retrospective study on Clinical manifestations of systemic paraphenylenediamine intoxication over six years (1994–2000) in the medical intensive care unit (ICU) of a University hospital and it concerned 19 patients hospitalized for systemic PPD intoxication. The mean age (± SD) was 27.9 ± 16.8 yrs, the sex ratio was about 0.58 and the Simplified Acute Physiology Score (SAPS II) was 30 ± 27. At admission, clinical symptoms were dominated by cervicofacial edema (79%), chocolate brown colored urine (74%), upper airway tract edema (68.4%), oliguria (36.8%), muscular edema (26.3%) and shock (26.3%). The biological results were dominated by rhabdomyolysis (100%), metabolic acidosis (100%), acute renal failure (ARF) (47.4%) and hyperkalaemia (26.3%) (Biological disturbances were more pronounced in patients with ARF). The therapies used were gastric lavage (100%), fluid infusion (100%), mechanical ventilation (84.2%), alkalinization (80%), corticosteroids (84.2%), vasopressors (26.3%) and renal replacement therapy (26.3%). The intoxication evolution was marked by the death of six patients (31.6%); five of them had developed ARF. The mechanical ventilation duration and the ARF occurrence testify to the severity of the intoxication. Ayoub Filali et al. [18] conducted aretrospective Study of Acute Systemic Poisoning of Paraphenylenediamine (Occidental Takawt) in Morocco. In this study they revealed 374 cases with a female predominance (77%). The majority of poisoning was intentional (78.1%) and the group most prone to PPD poisoning were the young population (15.1–25 and 25.1–35 years-old-age groups) accounting for 54.3 and 15.2%, respectively and 21.1% of poisoning cases were fatal, and the source/route of poisoning was by ingestion in the largest number of cases (93%). 50% of poisoning were from the south of Morocco, where photo-therapy knowledge is very developed. The largest number of cases was recorded in 2001 (20.1%). Motaouakkil et al. [19]reported a study on rhabdomyolysis and paraphenylene-diamine poisoning a cohort study was spread over six years (1999–2004). 315 patients were admitted over this period. They noticed a clear female predominance. The intoxication was voluntarily aiming at autolysis in 93.3% of the cases. The patients were admitted at about 5±5.3 hours after the intoxication. The clinical chart was at first dominated by the respiratory and renal symptoms. The mean of CPK was 132,351.8±164,978 UI/l. The treatment was especially symptomatic. The mortality was 47%. The multivariate analysis concluded that acid urinary pH, hyperglycemia, hard muscles, beta mimetic drugs and MPM II>0.14 were associated with a poor prognosis. The PPD poisoning represents the first cause of toxic rhabdomyolysis in their context and responsible of high mortality. For that, it's necessary to control PPD trade, to inform the medical persons and a rapid management.

Ram et al. [20]conducted a study on Paraphenylenediamine ingestion: an uncommon cause of acute renal failure. During a four-year period from 2002 to February 2006, 10 persons were admitted to their Institute after consuming a hair dye in a suicidal bid. The percentage of ARF due to PPD was 0.95%. Seven patients out of 10 (70%) who consumed PPD developed ARF. All 10 patients, including the patients who had normal renal function had features of rhabdomyolysis. Two patients required ventilator support for respiratory distress and two more required tracheostomy due to upper airway tract edema. One patient has expired after two sessions of dialysis. Renal biopsy in two patients (one postmortem) showed acute tubular necrosis along with presence of casts in tubules due to myoglobin. Bhargava et al., [21]case study on Hair Dye Poisoning had shown that, Hair dye ingestion is an uncommon form of poisoning in the west; however, in some parts of the world such as East Africa and Indian Sub-continent it is common. The main component of hair dye causing toxicity is Paraphenylenediamine (PPD), has been found to cause angio-neurotic edema, rhabdomyolysis and renal failure. They presented a case of hair dye poisoning who presented with respiratory distress due to laryngeal edema and later developed trismus and carpo-pedal spasm. This case report highlights the combined toxicities of sodium EDTA and PPD [22]. Out of 10 cases six were males and four females. The mean age was 20.9 yrs. At admission, clinical symptoms were dominated by paresis, angioedema, asphyxia, oliguria and itching. The biological results were dominated by rhabdomyolysis (100%), metabolic acidosis (100%), acute renal failure

(ARF) (80%), hyperphosphatemia (70%) hyperkalaemia (50%) and hypocalcemia (50%). The therapies used were gastric lavage (100%), fluid infusion (100%), mechanical ventilation (80%), alkalinization (40%), corticosteroids (50%), and renal replacement therapy (80%). Six patients died. The amount of toxin, higher level of azotemia, potassium and phosphorous and lower calcium at the time of admission were significantly associated with higher mortality. Levels of CPK, LDH, AST, ALT and delay in access to tertiary care centre were higher in those who expired; the difference was statistically not significant. Age and serum bicarbonate level did not influence mortality. Clinical manifestations of systemic PPD intoxication include respiratory, muscular, renal and hemodynamic syndromes. Treatment is largely supportive. Mortality rate is high and ARF affects survival adversely.

Abidi et al.[23] reported a case on myocardial lysis in a fetus induced by maternal Paraphenylenediamine poisoning following an intentional ingestion to induce abortion. The acute toxicity of Paraphenylenediamine (PPD) has been associated with several histopathological changes. However, its toxicity for the fetus has never been reported in the literature. They report a case of myocardial lysis in a fetus expelled by a 22yearold mother after apparent ingestion of an unknown amount of PPD. The patient was admitted to intensive care unit with acute onset of respiratory distress and rhabdomyolysis. The pelvic ultrasonography on admission showed a normally progressing pregnancy of 23–24 weeks. On day 9 post-ingestion, the patient spontaneously expelled a non-viable fetus. The fetal examination did not show any external or macroscopic abnormalities. However, the histopathological exam showed an important heart and lung congestion. There was also some interstitial edema and inflammation at the base of the lingua, in addition to a chorionic villus thrombosis and abruption placentae. The histopathology of the myocardium showed lysis of the cardiac muscle. This observation suggests that the PPD was most likely responsible for the myocardial injury in the fetus.

Abdelraheem et al., [24] studied on acute renal failure owing to paraphenylenediamine poisoning in Sudanese children. A three year period (2006–2008) were extracted from the medical records of the Pediatric Nephrology Unit, 17 children (16 female) were admitted to the Pediatric Nephrology Unit with PPD intoxication. Mean age was 13.8 yrs (range 2–18). Thirteen (76.4%) had attempted suicide, three (17.6%) were poisoned because of attempted murder and one poisoning (5.8%) was accidental. Eight children (47%) required tracheostomy for severe angioneurotic edema. Of 12, (71%) who developed acute renal failure (ARF), nine required dialysis and three were managed conservatively. Two children (12%) died and the other 15 recovered with normal renal function. PPD intoxication is a life-threatening condition with significant morbidity and mortality in children. Clinical manifestations and outcome are similar to those in adults. Mortality can be reduced by early recognition, prompt referral and aggressive supportive treatment. Sahay et al. [25]studied on Hair dye ingestion--an uncommon cause of acute kidney injury. 30 consecutive cases (24 males and 6 females) of hair dye induced renal failure seen. All the patients were aged between 18 to 40 years (26.9 ±4.95 years). The quantity of dye consumed ranged between 50–100 ml (79.5 ±22.45). All patients presented with oliguria and fluid overload. Dyspnoea was seen in 24 (80%) while 10 (33.33%) had hypertension. Encephalopathy and seizures were seen in 10 (33.3%). None of the patients had evidence of hemolysis, hematological abnormalities or skin rash. Three patients had elevated SGPT (340 IU/l) which returned to base line after two weeks while creatinine phosphokinase (CPK) was elevated in 6(20%) patients. The oliguric phase lasted from one to three weeks and serum creatinine normalized in 21 (70%) patients. Renal biopsy done in 15 patients (done ante mortem in 10 and postmortem in 5) showed evidence of acute tubular necrosis (ATN) in eight, acute interstitial nephritis (AIN) in seven patients. All patients received dialytic support. Eight (26.6%) patients succumbed. Hair dye is an unusual but important cause of acute kidney injury. The commonest renal lesions are acute tubulointerstitial damage. Respiratory and hemodynamic supportive therapy is essential for recovery. Yadavendra Reddy et al. [26]conducted a study on Paraphenylenediamine Poisoning. There was ten-fold rise in the number of Paraphenylenediamine Poisoning cases in the year upto May 2008. They are replacing the other modes of suicidal attempts such as pesticide poisoning and burn. It is more common in females (65%) and more in the age group of 15 to 35 years. There is high incidence and rapid rise of Paraphenylenediamine poisoning cases in this area. Many of them developing severe laryngeal edema within hour of consumption; which is not responding to medical treatment. So, timely reaching the hospital and emergency tracheostomy had major impact on mortality. A number of patients also developed renal failures. These alarming facts suggest the necessity to impose the regulation on the sale of Paraphenylenediamine as hair dye. Mohamed Abdelraheema et al., [27] study on Paraphenylene Diamine (Hair Dye) Poisoning in Children; in this article they reviewed the literature for PPD intoxication in children. PPD intoxication is a major health problem in eastern Africa, particularly Sudan, and in Morocco. It is also common in the Indian subcontinent. In two large series from Morocco and Sudan, Children constituted 11.5 and 18% of affected individuals, respectively. Acute poisoning by PPD causes characteristic severe angio-edema of the upper airway, often requiring tracheotomy, accompanied by a swollen, dry, hard and protruding tongue. PPD intoxication results in multisystem involvement and can cause rhabdomyolysis and acute kidney injury (AKI), flaccid paralysis, severe gastro-intestinal manifestations, cardiotoxicity and arrhythmias. This form of severe intoxication is fatal if not treated

aggressively. There is no specific antidote and treatment is mainly supportive with renal replacement therapy commonly used in cases with AKI. Mortality rate ranges between 12–42% are reported. PPD intoxication is a life threatening condition. Clinical outcomes rely on early recognition, prompt referral, and aggressive supportive treatment in collaboration with different specialties.

Dr. Sushil Kumar [28] conducted a review on Suicide by Para-phenylenediamine Poisoning has shown that 23-cases of acute Para-phenylenediamine poisoning were examined clinically at emergency ward of Rama Medical College Hospital, Kanpur (Uttar Pradesh) irrespective of age, sex and socio-economic strata along with route and manner of administration of the poison. Albuminuria, anaemia, hypocalcemia, leucocytosis, thrombocytopenia, increased serum bilirubin, prolonged bleeding and clotting time along with increased levels of liver enzymes and serum creatinine were observed as significant bio-chemical parameters; on investigation in respect to complete haemogram, liver function test, renal function test, serum electrolytes, serum CPK levels and arterial blood gas analysis. This study showed that PPD poisoning was fatal in about 18.75 percent cases. The poisoning of PPD was more common in this region previously. Poisoning of hair dye (PPD) is more common these days. The controlled supervision over selling of hair dye is necessary to stop PPD poisoning. They recommend that the selling of hair dye containing PPD should be banned and public education programmers should be initiated in this regard so that mortality from PPD may be prevented, because availability of PPD in home causes easy accessibility of this poison. Anugrah Chrispal et al. [29] conducted a study on Hair dye poisoning- an emerging problem in the tropics: an experience from a tertiary care hospital in South India. A chart review including records of clinical presentations, laboratory findings and treatment details was carried out. Eleven of the patients were women and the mean age was 27.2 years. The predominant clinical features were cervico-facial edema and pain, cola-coloured urine and oliguria. Laboratory investigations revealed elevated hepatic transaminases (100%), leucocytosis (92.3%), elevated creatinine phosphokinase (92.3%), metabolic acidosis (84.6%), hypocalcaemia (61.5%), hyperphosphataemia (46.2%) and renal failure (38.5%). Trends towards a poor outcome were evident among the following patients: late presentation at our centre; when no gastric lavage was done at the primary-care centre; those requiring tracheotomy/intubation at the primary centre; presentation with a low Glasgow Coma Score or seizures; established renal failure; and those who subsequently require dialysis, mechanical ventilation or intensive care. They concluded that hair dye poisoning classically presents with cervico-facial edema, severe rhabdomyolysis and renal failure. Early therapy with tracheostomy and aggressive forced dieresis are essential in order to prevent the high mortality associated with this toxin. It is imperative to raise public awareness of the potential toxicity of the dye as well as to educate physicians about the need for aggressive and early treatment.

Shalaby et al. [30]conducted a retrospective study on Clinical profile of acute Paraphenylenediamine intoxication in Egypt over seven years (2001–2008) on 25 cases with acute PPD intoxication admitted to the Poison Control Center Ain Shams University Hospitals, Cairo, Egypt. The mean age of the cases was 35.34 +/- 10.5 years; the male to female ratio was 18:7. Cervicofacial and laryngeal edema was the dominating presenting manifestation in 72% of the cases, 100% of the cases developed rhabdomyolysis, 80% had impaired renal functions, elevated liver transaminases were detected in 76% of cases, 75% showed hyperkalaemia and 16% died due to ventricular arrhythmia. In conclusion, PPD causes serious multisystem toxicity and its selling to the public should be officially restricted. Gurusamy et al. [31]reported a case study on Hair Dye Poisoning in a Tertiary Care Hospital at Madras Medical College & Government General Hospital, Chennai-03. This study analyzed the profile of five hair dye-poisoning cases. Out of them, four were males. The only female patient was six months amenorrhea. As she consumed only few ml. she was asymptomatic. The fetal heart sounds and movements were good. Among the male patients, two patients developed angio-neurotic edema. One was in need of emergency tracheostomy and the other one was treated comfortably with epinephrine injection. Both renal as well as respiratory failure occurred in two patients who died in the hospital itself. A male patient with respiratory failure presented with metabolic acidosis and hypotension died within 20 h of admission to the hospital. The one who expired after 20 days of hospital admission had pancreatitis the cause of which was enigmatic as had binge alcohol intake with hair dye. Rhabdomyolysis occurred in two patients one of whom had very high CPK, i.e., > 60,000 units/dl. Alkaline diuresis was done for the patient who had rhabdomyolysis, which prevented the development of renal failure in those patients. Nephrotic range of protenuria was present in only two cases though albuminuria was found in four out of five cases. All the patients who developed acute renal failure died in spite of dialysis. Acute renal failure increases duration of hospital stay and mortality. They conclude that hair dye ingestion was associated with respiratory, muscular, renal and metabolic complications. As there is no specific antidote for paraphenelynediamine, early referral and prompt supportive therapy for the complications will minimize the morbidity and mortality.

Paraphenylenediamine Hair dye intoxication is a life threatening condition. Clinical outcomes rely on early recognition, prompt referral, and supportive therapy in collaboration with different specialties.PPD at various concentrations, i.e., 0.3–7% can be fatal to the humans if consumed orally. The other toxic component of the hair dye was resorcinol,which is a corrosive and causes renal toxicity. The three main reasonsfor occurrence of deaths were; cardio respiratory failure, Myocarditis and Acute renal failure.

II. Conclusion

The controlled supervision over trading of hair dye is necessary to stop super Paraphenylenediamine poisoning. We recommend that selling of Paraphenylenediamine hair dye containing PPD should be controlled and public education programme should be initiated in this regard, so that mortality from Paraphenylenediamine poisoning may be prevented, because availability of Paraphenylenediamine hair dye containing PPD in home causes easy accessibility of this poison. The community should be educated not to do such activities, which endanger their lives.

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