A RETROSPECTIVE STUDY OF HAIR DYE POISONING CASES IN GOVT RAJAJI HOSPITAL, MADURAI

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MADURAI MEDICAL COLLEGE, MADURAI.

CERTIFICATE

This is to certify that this dissertation titled "**A RETROSPECTIVE STUDY OF HAIR DYE POISONING CASES IN GOVT RAJAJI HOSPITAL, MADURAI"** submitted by **DR.T.SUDHARSON** to the faculty of Forensic Medicine, The Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of MD degree branch XIV Forensic Medicine, is a bonafide research work carried out by him under our direct supervision and guidance.

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DECLARATION

I**, DR.T.SUDHARSON,** solemnly declare that the dissertation titled "**A RETROSPECTIVE STUDY OF HAIR DYE POISONING CASES IN GOVT RAJAJI HOSPITAL, MADURAI"** has been prepared by me. This is submitted to **The Tamilnadu Dr. M.G.R. Medical University, Chennai,** in partial fulfillment of the regulations for the award of MD degree (branch XIV) Forensic Medicine.

Place: Madurai **DR.T.SUDHARSON**

Date:

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CONTENTS

1. INTRODUCTION

1.1. POISONING IN INDIA

Poison is a quantitative concept, almost any substance being harmful at some doses but, at the same time, being without harmful effect at some lower dose. Between these two limits there is a range of possible effects, from subtle long-term chronic toxicity to immediate lethality. The definition of a poison, or toxicant, also involves a qualitative biological aspect because a compound, toxic to one species or genetic strain, may be relatively harmless to another. Exposure of humans and other organisms to toxicants may result from many activities: intentional ingestion, occupational exposure, environmental exposure, as well as accidental and intentional (suicidal or homicidal) poisoning.

Poisoning is a major health problem in many countries, including India. In our country, the problem is getting worse with time, as newer drugs and chemicals are developed in vast numbers, and there are no stringent rules and regulations for their dispensing and use. There are more than 9 million natural and synthetic chemicals worldwide, and the list keeps growing inexorably. Pesticides are the commonest cause of poisoning and according to World Health Organization (WHO)

estimates, approximately 3 million pesticide poisonings occur annually worldwide, causing more than 220 000 deaths. India accounts for one-third of pesticide poisoning cases in the third world and the worst affected are farm workers.[1]

The reason could be attributed to the increasing number of toxic chemicals and their large scale use without proper testing of their toxic properties. Banned products also continue to flow into the market. House hold products like medicines, cleaning products and cosmetics have further widened the spectrum of toxic products to which people may be exposed.

As much as we might wish for the end of poverty, ignorance, hunger, and exposure to hazardous chemicals, and as much as we work toward these goals, the challenges are formidable, and the end is not in sight. Chemicals and finished products made from chemicals continue to play an ever-present part in our lives. Although it is not evident that the benefits of chemicals always outweigh their risks, there is little doubt that a wide spectrum of chemicals and drugs has enhanced both the duration and quality of our lives. That said, certain of them, in certain situations, are clearly harmful to certain people. Among the fruits of

toxicologists' labors is information on how best to eliminate, reduce, or prevent such harm.

A retrospective analysis of poisoning calls received by the National Poisons Information Centre showed a total of 2719 calls over a period of three years (April 1999 to March 2002). The queries were made on poisoning management (92%) and information (8%) about various products and functioning of the centre. Hair dye constituted 30(1.1%) of these cases which is an alarmingly high incidence.[1]

Globally suicide rates have increased by 60% in the 50 years. Suicide is now ranked among the three leading causes of death in the age group between 15 and 44 years.[2] The reasons proposed are increased day-to-day mental stress due to industrialization, marital disputes, poverty, and unemployment. In developing countries pesticide poisoning is one of the leading contributors to this preventable tragedy.

Recently, substances which are used for domestic and cosmetic purposes viz., thinners, antiseptics, talcum powder, silica gel, detergents, naphthalene balls and camphor are also used for suicide.

One substance which worth a mention here is the hair dye which is emerging as an important etiological factor.

Though uncommon in the west, both accidental and intentional ingestion of PPD is frequently reported from Africa, the Middle-East, and the Indian subcontinent where PPD is commonly mixed with henna, which is traditionally applied to colour the palms of hands and to dye the hairs. Paraphenylenediamine poisoning was the number one cause of poisoning in Morocco during the 1990s. In India due to popular usage, hair-dye poisoning is quite likely. Numerous case reports have been reported from India. Most reported cases were from adolescents and adults

It is obvious, but often forgotten, that toxicity is always a consequence of exposure and that no matter what the results of hazard assessment, without exposure there cannot be a toxic effect. What has not changed, however, is the need for the toxicological literature to serve many masters. Given the eclectic nature both of the methodological roots and the practical needs served by toxicology, general works are needed more than ever.

2. REVIEW OF LITERATURE

In 1924, Nott described the first case of systemic toxicity with PPD in the owner of a hair saloon.[3] Though uncommon in the west, both accidental and intentional ingestion of PPD is frequently reported from Africa, the Middle-East, and the Indian subcontinent where PPD is commonly mixed with henna, which is traditionally applied to colour the palms of hands and to dye the hairs. It has been reported around the world, more so in the underdeveloped and developing countries. Paraphenylenediamine poisoning was the number one cause of poisoning in Morocco during the 1990s.[4] In two large series from Sudan and Morocco, children constituted 18% and 11.5% of the affected individuals respectively.[5][6] In India due to popular usage, hair-dye poisoning is quite likely. Numerous case reports have been reported from India.[7][8][9][10]

A retrospective analysis of poisoning calls received by the National Poisons Information Centre showed a total of 2719 calls over a period of three years (April 1999 to March 2002). 44.1% of these cases were poisoning with household products which include phenyl, naphthalene, kerosene, detergents, corrosives, hair dye, antiseptics, thinner, silica

gel, pesticides, talcum powder, camphor etc. Hair dye constituted 30(1.1%) of these cases which is an alarmingly high incidence.[1]

Types of poison	**No of cases**	**Percentage**
Household products	1102	44.1%
Pesticides	516	
Phenyl	114	
Thermometer mercury	119	
Naphthalene	44	
Kerosene	25	
Detergents and corrosives	76	
Antiseptics	54	
Hair dye	**30**	
Others(thinner, silica gel, button battery, talcum powder, hydrogen peroxide)	124	

Agricultural products	**320**	**12.8%**
Aluminium phosphide	120	
Organophosphates	60	
Organochlorines	75	
Ethylenedibromide	30	
Herbicides	22	
Pyrethroids	13	

Industrial chemicals	**224**	**8.9%**
Copper sulphate	48	
Hydrocarbons	20	
Nitrobenzenes	26	
Alcohol	24	
Ammonia	6	
Sulphuric acid, Hydrochloric acid	8	
Acetone, gases	12	
Heavy metals	6	
Turpentine oil	11	
Others	63	

Drugs	**470**	**18.8%**
Benzodiazepines	83	
Anticonvulsants	74	
Analgesics	47	
Antihistaminics	35	
Ayurvedic/Homeopathic	19	
Antipsychotics	12	
Mixed drugs	23	
Tricyclic antidepressants	4	
Others(cardiovascular drugs, thyroid hormones, iron, OCPs, antidiarrheals)	173	

Animal bites and stings	**118**	**4.7%**
Snake	62	
Scorpion	24	
Honey bee/Wasp	9	
Spider/Lizard	7	
Others(dog, monkey, mongoose, rat, jelly fish)	16	

Plants	**44**	**1.7%**
Datura	28	
Yellow oleander	5	
Bhang	5	
Others	6	

Unknown	74	2.9%
Miscellaneous	142	5.6%

2.1. Types of hair dyes

Among the Egyptians, there were hairdressers as early as 5000 years BC and the art of dyeing hair with vegetable dyes was known already at that time. The first artificial dye was synthesized in the laboratory in 1856, and permanent hair colourants have been in commercial use for over 100 years.[11] Hair dyeing, a practice of great antiquity, is still widely prevalent today. A variety of materials are used for the purpose of producing variously tinted hair.

2.1.1. Classification I

(1) Those of vegetable origin (henna, chamomile)

(2) Metallic substances (silver, manganese, bismuth)

(3) Synthetic compounds (paraphenylenediamine, paratoluenediamine, pyrogallol).

Because of the ease of application, the relative permanence, and supposed "naturalness" of the resulting tints, the last group has been greatly exploited in recent years. Soon after their introduction, however, as important fixtures in the cosmetic armamentarium, reports of toxic effects began to appear in medical literature.

2.1.2. Classification II

Hair dyes can also be classified into four categories, each with a specific composition and action mechanism:

1. Gradual hair colouring (using metallic dyes such as salts of lead, bismuth or silver)
2. Temporary dyes (water-soluble dyes that withstand only one-time shampooing)
3. Semi-permanent dyes (which can withstand 4-5 times of shampooing)
4. Permanent hair colours

Permanent hair colours are the most popular hair dye products. The permanent agents contain oxidizing agents that interact with the dye to cause colour molecule to fix on to hair shaft and most common being the Hydrogen Peroxide. Common dye intermediates used in 1:1 concentration with Hydrogen Peroxide are Paraphenylenediamine, Resorcinol or Aminophenols. The remaining ingredients are soaps and synthetic detergents. Paraphenylenediamine is a type of permanent hair colour used as a component of permanent waving kits in hair styling.

They may be further divided into **oxidation hair dyes** and **progressive hair dyes**. Oxidation hair dye products consist of (1) a solution of dye intermediates, e.g., para-phenylenediamine, which form hair dyes on chemical reaction, and preformed dyes, e.g., 2-nitro-p-phenylenediamine, which already are dyes and are added to achieve the intended shades, in an aqueous, ammoniacal vehicle containing soap, detergents and conditioning agents; and, (2) a solution of hydrogen peroxide, usually 6%, in water or a cream lotion.

The ammoniacal dye solution and the hydrogen peroxide solution, often called the developer, are mixed shortly before application to the hair. The applied mixture causes the hair to swell and the dye

intermediates (and preformed dyes) penetrate the hair shaft to some extent before they have fully reacted with each other and the hydrogen peroxide and formed the hair dye.

Progressive hair dye products contain lead acetate as the active ingredient. Lead acetate is approved as a colour additive for colouring hair on the scalp at concentrations not exceeding 0.6% w/v, calculated as metallic lead . Bismuth citrate, the other approved colour additive, is used to a much lesser extent. Progressive hair dyes change the colour of hair gradually from light straw colour to almost black by reacting with the sulphur of hair keratin as well as oxidizing on the hair surface.[12]

2.2. POISONOUS INGREDIENTS IN HAIR DYE

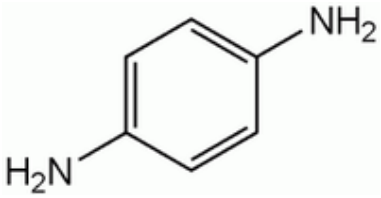
The active ingredients of a commonly used hair dye in India include paraphenylene diamine (PPD), propylene glycol, liquid paraffin, cetostearyl alcohol, sodium lauryl sulphate, EDTA and resorcinol.

2.2.1. PARAPHENYLENE DIAMINE

Paraphenylene diamine (PPD) [C6H4 (NH2)2] a coal tar derivative, is an aromatic amine not found in nature and it is produced commercially by many industrial companies. It is a derivative of paranitroanaline that is available in the form of white crystals when pure and rapidly turns to brown when exposed to air.[13][14][15] The major oxidation product of PPD is Bondrowski's base, which is allergenic, mutagenic and highly toxic.

It is widely used in industrial products such as textile or fur dyes, dark coloured cosmetics, temporary tattoos, photographic development and lithography plates, photocopying and printing inks, black rubber, oils, greases and gasoline.

PPD is the most common constituent of hair dye formulations. It is often the key ingredient but can also be used for colour enhancement. PPD is commonly used in its raw form for cosmetic purposes in Africa, Middle East and Indian subcontinent while it is rarely used in the west.

***p*-Phenylenediamine**

IUPAC name 1,4-diaminobenzene

In Sudan, PPD is mixed with henna, leaves of Lawsonia Alba, which is a non toxic herb used to decorate the hands and feet in special social events, such as wedding ceremonies. Henna needs to be applied to the skin several times in order to achieve the deep brown or black colour. When PPD is added to henna, it reduces the number of skin application to one. More often, PPD is used separately.[5]

In Morocco, a non-toxic herbal extract from the gallnut of athel-pine (Tamarix aphyla) is traditionally used to dye hair. This natural extract is locally known as “takawt”. Its rarity resulted in the use of PPD as a substitute under the misleading name of “occidental takawt”.[6]

The concentration of PPD in different hair dyes range from 0.2 % to 3.75%. The exact concentration that causes toxicity is not known. Interestingly, variable concentrations of PPD are used in different hair dye formulations from different countries depending on the local manufacturer advice. All such recommendations are related to formulations used to dye hair and safe concentrations regarding skin application and decoration are not known. In countries where PPD is produced (Germany, Japan and UK) it is considered an occupational health hazard and the standard for air contamination developed by health authorities is 0.1 mg/m3 in the working atmosphere. In many countries in the Middle East PPD is freely available at low cost on local markets; 10 g of PPD can be bought for less than one US Dollar.

Toxicity

The toxicity of PPD includes skin irritation, contact dermatitis, chemosis, lacrimation, exophthalmos, or even permanent blindness, due to local contact. Ingestion of PPD produces two types of toxic effects. The first consists of rapid development of severe oedema of the face, neck, pharynx, tongue, and larynx with respiratory distress, often requiring tracheostomy. In the later phase, rhabdomyolysis and acute

tubular necrosis supervene. Vomiting, gastritis, hypertension, vertigo, tremors, and convulsions have been reported.[9][10]

The characteristic triad of features encountered is early angioneurotic edema with stridor, rhabdomyolysis with chocolate coloured urine and acute renal failure. Whenever this combination occurs in poisoning, hair dye is a strong suspect.[15][16][17][18][19][20][21]

Respiratory system and upper airway manifestations

One of the most severe clinical manifestations and the main cause of death in PPD poisoning is upper airway obstruction (angio-edema) manifesting with a hard swollen protruding tongue and edematous bull neck. The respiratory syndrome following the ingestion of PPD is represented by asphyxia and respiratory failure secondary to inflammatory edema involving cricopharynx and larynx. Clinical picture may resemble Ludwig's angina.[9][10][15-21]

Rhabdomyolysis

Rhabdomyolysis can be caused by a variety of physical, chemical, metabolic, infective and toxic causes. Rhabdomyolysis can develop due to physical (heat stroke), chemical (drugs, poisons), metabolic

(Hypokalemia) and infective (Leptospirosis, Dengue virus) causes.[23][24]

PPD can bring about rhabdomyolysis by promoting calcium release and leakage of calcium ions from the smooth endoplasmic reticulum, followed by continuous contraction and irreversible change in the muscle's structure.[23][24]

Rhabdomyolysis is the main cause of acute renal failure and the morbidity and mortality are high once renal failure develops.[15-21]

Renal failure

The extent of renal involvement varies between transient proteinuria and oliguric AKI. AKI commonly develops a few days after PPD exposure. The kidney injury is thought to be due to the direct toxic effect of PPD, hypovolemia, hemolysis and rhabdomyolysis with the deposition of myoglobin casts within the renal tubules. The pathogenesis of ATN, independent of rhabdomyolysis appears to be due to the aromatic structure of PPD making it easily reabsorbed and concentrated in tubules. When the toxicity was in small doses like hair dye, membranous nephropathy has been reported owing to in situ formation of immune complex. Proposed mechanisms for the renal

damage include direct tubular toxicity due to induction of active oxygen radicals and reduced renal perfusion resulting from intravascular volume depletion and possibly decreased vasomotor tone.[9][10][15-21]

Myoglobinuric ARF is observed in the tropics after a variety of conditions, such as crush syndrome, burns, heat stroke, electrical injury, eclampsia, prolonged labour, poisoning with mercuric chloride, zinc or aluminum phosphide, status epilepticus, viral myositis and status asthmaticus. The diagnosis is established by the demonstration of myoglobin in urine and elevated levels of creatine phosphokinase and aldolase in the serum. Since myoglobin is a small molecule with a molecular weight of 17 kDa and binds only lightly to the plasma proteins, it escapes easily in the urine. Therefore, the urine may not contain myoglobin if the patient presents late in the course of the disease and the true incidence of myoglobinuric ARF will be underestimated. Severe hypocalcaemia and hyperuricaemia during the oliguric phase and hypercalcaemia during the diuretic phase are characteristic of this condition. The pathogenesis of myoglobinuric ARF is similar to that following intravascular hemolysis.[8]

In this context it is worth mentioning that all these parameters of renal function remain unaltered until almost 50% of reduction in glomerular filtration rate. Urine microscopy may show occasional granular casts and degenerating renal epithelial cells and pus cells, suggestive of possible acute tubular necrosis. However, absence of red blood corpuscles rules out the possibility of hematuria. Positive benzidine test (based on heme-associated peroxidation and consequent nascent oxygen-mediated color development with Benzidine in acidic media) with the urine sample reflects the presence of hemoglobin or myoglobin. Further treatment of sample by addition of potassium chloride in acidic media generates characteristic rhombic crystal (otherwise known as hemin crystal) and indicates presence of hemoglobin.[25]

Skin and eye manifestations

It is well known that PPD cause skin irritation, kerato-conjunctivitis, conjunctival swelling and eczema of the eyelids. Allergic reactions causing dermatitis, urticaria and asthma have also been reported.[15-21]

Other manifestations

Flaccid paraplegia, palato-pharyngeal and laryngeal paralysis were also reported in adults and children. Neurotoxicity causing mental alteration and coma was also observed and was possibly related to brain anoxia and severe metabolic acidosis associated with AKI. Other features include anaemia, leukocytosis, haemoglobinaemia, and haemoglobinuria. Liver necrosis has also been reported. Hypertension is generally seen, but presence of hypotension/shock is a poor prognostic indicator.[9][10][15-21]

A rare case of Nephrogenic Systemic Fibrosis due to hair dye poisoning has also been reported.[26]

Cardiac toxicity causing arrhythmia, heart block and sudden death was also reported in some studies. It is commonly the direct cause of death in children and adults with PPD poisoning. Cardiac toxicity is mainly caused by the direct toxic effect of PPD on the heart, rhabdomyolsis of the cardiac muscle causing severe damage and hyperkalemia.[27][28]

Laboratory features

Raised serum osmolality, ALT, CPK, LDH and urea, and hyperkalemia, methemoglobinemia, hemoglobinemia and metabolic acidosis. High CPK, LDH, GOT and GPT, and leukocytosis, indicates massive skeletal muscle necrosis. Peripheral smear can show anisocytosis and poikilocytosis. Urine analysis can show raised urine osmolality, proteinuria, hematuria, hemoglobinuria, myoglobinuria and albuminuria.[9][10][15-21]

Diagnosis

The diagnosis of PPD intoxication is largely dependent on clinical manifestations. The clinical features are rather unique and in the absence of laboratory facilities in many developing countries the angio-edema of the face and neck together with the hard protruding tongue and the chocolate-brown colour of the urine are used for clinical diagnosis. Organ damage may be assessed by appropriate tests for rhabdomyolysis, and kidney and liver involvement. The urine can be tested for PPD using thin layer chromatography sprayed with 0.2% aqueous potassium permanganate which is essential for medico-legal

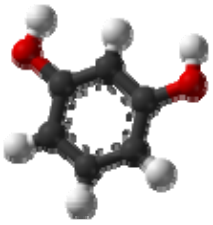
purposes. However, this test is not routinely available and there is a need for a rapid test to demonstrate PPD in blood or urine.[9][10][15-21]

It is worth mentioning that the amount of PPD that can cause systemic poisoning is only three grams, while the lethal dose is 7-10 grams.[15]

2.2.2. RESORCINOL

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. Used externally it is an antiseptic and disinfectant, and is used 5 to 10% in ointments in the treatment of chronic skin diseases such as psoriasis, hidradenitis suppurativa and eczema of a sub-acute character. Topical acne treatments at 2% or less concentration and in prescription treatments at higher concentrations. Weak, watery solutions of resorcinol (25 to 35 g/kg) are useful in allaying the itching in erythematous eczema. [13][14]

Resorcinol is also used as a chemical intermediate for the synthesis of pharmaceuticals and other organic compounds. It is used in the production of diazo dyes and plasticizers and as a UV absorber in resins. Resorcinol is an analytical reagent for the qualitative determinaion of ketoses (Seliwanoff's test).

Resorcinol	
	
IUPAC name Benzene-1,3-diol	

Toxicity

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. Resorcinol is also toxic to the thyroid gland and cause hypothyroidism. But the characteristic features of rhabdomyolysis and laryngeal edema which typify PPD poisoning are absent.[15][29][30]

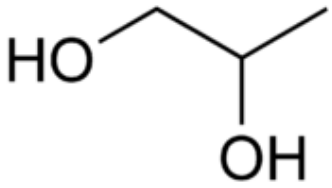
2.2.3. PROPYLENE GLYCOL

Propylene glycol is a viscous, colorless liquid commonly used as a solvent in hair dyes. It is also used as a solvent in many pharmaceuticals, including oral, injectable and topical formulations. Diazepam, which is insoluble in water, uses propylene glycol as its solvent in its clinical, injectable form. It is also used as food additive, a moisturizer in medicines, cosmetics, food, toothpaste, mouth wash, hair care and tobacco products, In hand sanitizers, antibacterial lotions, and saline solutions. As a non-toxic antifreeze for winterizing drinking water systems, and in applications where the used antifreeze eventually will be drained into the soil, water, or a septic system.[13][14]

Toxicity

The acute oral toxicity of propylene glycol is very low, and large quantities are required to cause perceptible health damage in humans; propylene glycol is metabolized in the human body into pyruvic acid (a normal part of the glucose-metabolism process, readily converted to energy), acetic acid (handled by ethanol-metabolism), lactic acid (a

normal acid generally abundant during digestion), and propionaldehyde.

Propylene glycol

<u>IUPAC name</u> propane-1,2-diol

Serious toxicity generally occurs only at plasma concentrations over 1 g/L, which requires extremely high intake over a relatively short period of time. It would be nearly impossible to reach toxic levels by consuming foods or supplements, which contain at most 1 g/kg of PG. Cases of propylene glycol poisoning are usually related to either inappropriate intravenous administration or accidental ingestion of large quantities by children. The potential for long-term oral toxicity is also low.[31][32]

It is associated with hypotension, bradycardia, QRS and T abnormalities in ECG hemolysis, hyperosmolality, raised anion gap metabolic acidosis, central nervous system depression, arrhythmias and renal dysfunction. Acute tubular necrosis has been described. Proximal renal tubular cell swelling and vacuole formation have also been seen in propylene glycol ingestion. But the characteristic features of rhabdomyolysis and laryngeal edema which typify PPD poisoning are absent.[30][31]

2.2.4. SODIUM LAURYL SULFATE

Sodium lauryl sulfate (SLS) or sodium dodecyl sulfate (SDS or NaDS) ($C_{12}H_{25}SO_4Na$) is an anionic surfactant used in many cleaning and hygiene products.[13][14]

SLS is a highly effective surfactant and is used in any task requiring the removal of oily stains and residues. For example, it is found in higher concentrations with industrial products including engine degreasers, floor cleaners, and car wash soaps. It is used in lower concentrations with toothpastes, shampoos, and shaving foams. It is an

important component in bubble bath formulations for its thickening effect and its ability to create a lather.

Sodium lauryl sulfate

IUPAC name Sodium dodecyl sulfate

Toxicity

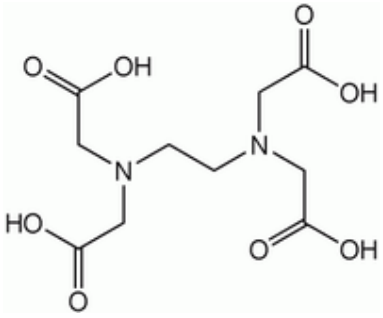
SLS is not carcinogenic, either when applied directly to the skin or consumed. SLS may worsen skin problems in individuals with chronic skin hypersensitivity, with some people being affected more than others. SLS has also been shown to irritate the skin of the face with prolonged and constant exposure (more than an hour) in young adults. A preliminary study suggested SLS in toothpaste caused the recurrence of aphthous ulcers, commonly referred to in some countries as canker sores or white sores. May cause hair loss by attacking the follicle. Is potentially harmful to skin and hair. Cleans by corrosion. Dries skin by stripping the protective lipids from the surface so it can't effectively regulate moisture. Another extremely serious problem is the

connection of SLS with nitrate contamination. SLS reacts with many types of ingredients used in skin products and forms nitrosomines (nitrates). Nitrates are potential carcinogens.[13][14]

2.2.5. ETHYLENE DIAMINE TETRA-ACETIC ACID(EDTA)

Ethylenediaminetetraacetic acid is a polyamino carboxylic acid and a colourless, water-soluble solid.

In industry, EDTA is mainly used to sequester metal ions in aqueous solution. In the textile industry, it prevents metal ion impurities from modifying colours of dyed products. In the pulp and paper industry, EDTA inhibits the ability of metal ions, especially Mn^{2+}, from catalyzing the disproportionation of hydrogen peroxide, which is used in "chlorine-free bleaching." In similar manner, EDTA is added to some food as a preservative or stabilizer to prevent catalytic oxidative discolouration, which is catalyzed by metal ions. In personal care products, it is added to cosmetics to improve their stability toward air. In soft drinks containing ascorbic acid and sodium benzoate, EDTA mitigates formation of benzene (a carcinogen).[13][14]

Ethylenediaminetetraacetic acid

<u>IUPAC name</u> 2,2',2",2"'-(ethane-1,2-diyldinitrilo)tetra acetic acid

In medicine EDTA is used to bind metal ions in chelation therapy, e.g., for mercury and lead poisoning. It is used in a similar manner to remove excess iron from the body. This therapy is used to treat the complication of repeated blood transfusions, as would be applied to treat thalassaemia. EDTA acts as a powerful antioxidant to prevent free radicals from injuring blood vessel walls.

Dentists use EDTA solutions to remove inorganic debris (smear layer) and prepare root canals for obturation. It serves as a preservative (usually to enhance the action of another preservative such

asbenzalkonium chloride or thiomersal) in ocular preparations and eyedrops. In evaluating kidney function, the complex $[Cr(edta)]^-$ is administered intravenously and its filtration into the urine is monitored. This method is useful for evaluating glomerular filtration rate.

EDTA is used extensively in the analysis of blood. It is an anticoagulant for blood samples for CBC/FBEs (complete blood count also known as full blood examination). Laboratory studies also suggest that EDTA chelation may prevent collection of platelets on the lining of the vessel [such as arteries] (which can otherwise lead to formation of blood clots, which itself is associated with atheromatous plaque formation or rupture, and thereby ultimately disrupts blood flow).

2.2.6. CETOSTEARYL ALCOHOL

Cetostearyl alcohol, cetearyl alcohol or cetylstearyl alcohol is a mixture of fatty alcohols, consisting predominantly of cetyl and stearyl alcohols and is classified as a fatty alcohol. It is used as an emulsion stabilizer, opacifying agent, and foam boosting surfactant, as well as an aqueous and

nonaqueous viscosity-increasing agent. It imparts an emollient feel to the skin and can be used in water-in-oil emulsions, oil-in-water emulsions, and anhydrous formulations. It is commonly used in **hair conditioners and other hair products.**[13][14]

2.3. MANAGEMENT OF HAIR DYE POISONING

Hair dye ingestion is a medical emergency and has high mortality. There is no specific antidote. The most important aspect of management is early recognition of poisoning by this compound.

Specific therapy involves prompt removal of unabsorbed poison from the gastrointestinal tract, and elimination of the unreacted Paraphenylenediamine from the circulation. Immediate gastric lavage should be performed, preferentially with 2% sodium bicarbonate. The hydrophilic nature and low molecular weight of PPD would suggest a low adsorbability. On the other hand, charcoal therapy is simple, inexpensive, well tolerated and non-toxic.[15-21]

Early intervention with half normal saline and soda bicarbonate infusion has been shown to be beneficial in Rhabdomyolysis.[24]

For patients presenting with asphyxia, Tracheostomy and intensive medical treatment with hydrocortisone and chlorpheniramine maleate

(antihistaminic drug) and penicillin cover are life saving measures. The mild cases were successfully treated with hydrocortisone and chlorpheniramine maleate and penicillin without tracheostomy.[22]

Renal support in the form of dialysis is required in ARF. Treatment is mainly supportive depending on clinical features at presentation.[33][34]

2.4. POSTMORTEM FINDINGS

External findings are nonspecific. Internally the respiratory tract showed laryngeal edema, mucus bronchial secretions, pulmonary edema and congestion. Hyperemia and edema of laryngeal mucosa which occludes almost the entire upper airway is seen in anaphylactic deaths.[17]

Lungs show features of ARDS. Lungs are heavy, firm, red and boggy. They exhibit congestion, interstitial and alveolar edema, inflammation, fibrin deposition and diffuse alveolar damage. The alveolar walls become lined with waxy hyaline membranes. Alveolar hyaline membranes consist of fibrin rich edema fluid.[35][36] Scattered coagulative necrosis of skeletal muscles are seen associated with inflammatory cell infiltration.[24]

Hyperemia and congestion of all abdominal organs are seen in cases which die within 3 days of consumption.

Histopathology of the cadaveric kidney shows changes of acute tubular necrosis. Renal collecting ductules and distal tubules occluded by dark brownish myoglobin casts and its epithelium massively necrotized. An eosinophilic substance in renal cortical tubular lumen may be seen.[35][36]

2.5. CHEMICAL ANALYSIS

PPD is detected qualitatively by diazotization and coupling reaction. The viscera are soaked in hexane. Decap the hexane layer. To the filtrate add 10ml of acid alcohol mixture, 1ml of con. HCl and zinc dust(0.2gm). Heat in a water bath to reduce the solution. After half an hour cool and add paraffin wax (to absorb proteins). To the filtrate add con. HCl and 5ml of 0.25% $NaNO_2$ for diazotization. Cool to 0 degrees. The excess NO_2 is removed by the addition of 2ml of 7.5% ammonium sulphamate. After 10minutes it is coupled with N1-Naphthyle-Ethylenediamine dihydrochloride. Leave it for 30 minutes. Appearance of Magenta color indicates the presence of paraphynelene diamine. PPD can be detected quantitatively by thin layer chromatography.[37]

3. AIMS AND OBJECTIVES:

1. To study the epidemiology and prevalence of suicidal hair dye poisoning
2. To analyze the presenting features of Hair dye poisoning.
3. To analyze the treatment modalities
4. To study the postmortem findings

4. MATERIALS AND METHODS

Duration of study: 14 months

Period of study: July 2009 to August 2010

Selection of study subjects: Patients admitted with history of Hair dye poisoning at Government Rajaji Hospital, Madurai

Number of cases studied: 36 patients

Data Collection: Clinical, Biochemical, Postmortem, Histpathological and Toxicological analysis

Methods: Standard Clinical, Autopsy and Laboratory methods.

Ethical clearance: Obtained

Inclusion Criteria: Patients with history of hair dye poisoning

Design of Study: Retrospective study

Statistical Tools

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using **Epidemiological Information Package (EPI 2008)** developed by Center for Disease Control, Atlanta.

5. RESULTS

36 cases of hair dye poisoning admitted in Government Rajaji Hospital, Madurai during July 2009 to August 2010 were studied. Various data like the background history, clinical features, investigations, treatment, outcome, postmortem findings and toxicological analysis had been analyzed in detail and the results are plotted as below.

Table 1: Incidence of hair dye poisoning

Year	Total number of cases
2008	11
2009	17
2010(upto August)	25

Table 2: Age distribution

Age group (in years)	Cases	
	No	**%**
< 15 years	-	-
16 – 30	30	83.3
31 – 40	3	8.4
41 – 50	2	5.5
Above 50yrs	1	2.8
Total	36	100
Range	16 – 65 years	
Mean	25.9 years	
SD	9.9 years	

83.3% of the hair dye poisoning cases are in the 16 – 30 age groups.

Table 3: Sex distribution

Sex	Cases	
	No	**%**
Male	5	13.9
Female	31	86.1
Total	36	100

86% of the hair dye poisoning cases were females

Table 4: Clinical features

Clinical feature	Yes		No	
	No.	**%**	**No.**	**%**
Chocolate brown urine	16	44.4	20	55.6
Shock	14	38.9	22	61.1
Angio Edema	19	52.8	17	47.2
Rhabdomyolysis	23	63.9	13	36.1
Hemolysis	20	55.5	16	44.4
Renal failure	11	30.6	25	69.4
ECG changes	14	38.9	22	61.1
Sepsis	2	5.6	34	94.4

Rhabdomyolysis is the most common clinical feature.

Table 5: Outcome

Outcome	Cases	
	No	**%**
Alive	22	61.1
Dead	14	38.9
Total	36	100

Mortality rate was 38.9%

Table 6: Cause of death

Cause of death	Cases	
	No	**%**
Laryngeal Edema	9	64.3
Renal failure	2	14.3
Septicemia	1	7.1
ARDS	2	14.3
Total	14	100

Laryngeal edema is the most common cause of death

Table 7: Day of death and cause of death

Day of death	Cause of death							
	Laryngeal edema		Renal failure		Septicemia		ARDS	
	No.	%	No.	%	No.	%	No.	%
Same day (4)	4	100	-	-	-	-	-	-
1 – 2 days (5)	5	100	-	-	-	-	-	-
3 – 4 days (2)	-	-	-	-	-	-	2	100
> 4 days (3)	-	-	2	66.7	1	33.3	-	-
Total	9	64.3	2	14.3	1	7.1	2	14.3

Laryngeal edema is the most common cause of death in the first 2 days. Renal failure is the most common cause after 4 days.

Table 8: Chemical analysis

Chemical analysis	Cases	
	No	**%**
Positive	3	21.4
Negative	10	71.4
Pending	1	7.1
Total	14	100

Chemical analysis was negative in 71.4% of cases

Table 9: HPE of kidney

HPE of kidney	Cases	
	No	**%**
Normal	6	42.9
Myoglobin casts	5	35.7
A T N	3	21.4
Total	14	100

HPE of kidney was normal in 42.5% of the cases

Table 10: Quantity consumed and outcome

Outcome	**Quantity consumed**	
	Mean	**S.D.**
Alive	36.6	15.1
Dead	66.8	17.8
'p'	**0.0001** **Significant**	

Table 11: Clinical features and outcome

Clinical features	Outcome				'p'
	Alive		Dead		
	No.	%	No.	%	
Chocolate brown urine					
Yes	10	62.5	6	37.5	0.8484
No	12	60	8	40	Not significant
Shock					
Yes	5	35.7	9	64.3	**0.0321**
No	17	77.3	5	22.7	**Significant**
Angio Edema					
Yes	7	36.8	12	63.2	**0.0049**
No	15	88.2	2	11.8	**Significant**
Rhabdomyolysis					
Yes	16	69.6	7	30.4	0.3039
No	6	46.2	7	53.8	Not significant

Renal failure					
Yes	8	72.7	3	27.3	0.2853
No	14	56	11	44	Not significant
ECG changes					
Yes	8	57.1	6	42.9	0.9689
No	14	63.6	8	36.4	Not significant
Sepsis					
Yes	1	50	1	50	0.6333
No	21	61.8	13	38.2	Not significant

Cases that developed shock and/or angioedema have a significantly higher rate of mortality.

Table 12: Treatment given and outcome

Treatment given	Outcome				'p'
	Alive		Dead		
	No.	%	No.	%	
Timely G L					
Yes	17	73.9	6	26.1	**0.0388**
No	5	38.5	8	61.5	**Significant**
Tracheostomy					
Yes	7	53.8	6	46.2	0.7517
No	15	65.2	8	34.8	Not significant
Mechanical Ventilation					
Yes	4	100	-	-	0.1242
No	18	56.3	14	43.8	Not significant
Haemodialysis					
Yes	8	72.7	3	27.3	0.2953
No	14	56	11	44	Not significant

6. DISCUSSION

During a period of 14 months, **36** cases of hair dye poisoning admitted in Government Rajaji Hospital, Madurai were studied. Various data like the background history, clinical features, investigations, treatment, outcome, postmortem findings and toxicological analysis had been analyzed in detail using EPI INFO 2002, designed by CDC, Atlanta.

1. There are 25 cases of hair dye poisoning upto the month of August 2010 which is more than 60% increase in incidence.

A retrospective analysis of poisoning calls received by the National Poisons Information Centre revealed 30 cases of hair dye poisoning. In a three and half year study(January 2006 to July 2009) of hair dye poisoning by Anurag Chrispal et al (TROPICAL DOCTOR > VOLUME 40, NUMBER 2 > PP. 100-103) there were 13.

In an early report from Sudan, 31 children with PPD intoxication were described over a 5-year period In Sudan, over a 10-year period (1995–2005), 3159 patients were reported to suffer from PPD poisoning**(**Abderlraheem MB, El-Tigani MA, Hassan EG, Ali MA,

Mohamed IA, Nazik AE. Acute renal failure owing to paraphenylene diamine hair dye poisoning in Sudanese children. Ann Trop Paediatr. 2009 Sep;29(3):191-6**)**

A study from Morocco described 374 cases of PPD poisoning in adults and children over a 10-year period(Filali A, Semlali I, Ottaviano V, Fumari C, Corradini D, Soulaymani R. A retrospective study of acute systemic poisoning of paraphenylenediamine (occidental takawt) in Morocco. Afr J Trad. 2006;3(1):142-9.)

Hair dye poisoning was once unknown in India and common in East African countries. But various studies in India indicate that the incidence is increasing at an alarming rate.

2. 83.3% of the hair dye poisoning cases in our study come under the age group of 16 – 30 years. There was not even a single case in the age group less than 15 years. All the cases were due to intentional self harm and there was no case of accidental or homicidal poisoning.

A study conducted by Mohamed Abdelraheem, Mohamed Hamdoukb, Eduard E Zijlstrac in Sudan over a 10-year period

(1995–2005), 3159 patients were reported to suffer from PPD poisoning; among these were 568 (18%) children below the age of 14 years. In a study from Morocco, 11.5% of affected patients were children less than 15 years of age.

A study from Morocco described 374 cases of PPD poisoning in adults and children over a 10-year period. The majority of patients (54%) were in the 15-24 years age group and children contributed significantly to the study population (11.5%). The majority of poisoning was intentional (78.1%).

The mean age in our study is 25.9 years.

A three and half year study of hair dye poisoning by Anurag Chrispal et al (TROPICAL DOCTOR > VOLUME 40, NUMBER 2 > PP. 100-103) there were 13 cases in a 3 years study and the mean age was 27.2 years.

3. 31 out of 36(86.1%) cases were females in our study.

Majority of the studies indicate a higher incidence of hair dye poisoning in females.

In a four year (2002 to 2006) study of hair dye poisoning by Ram et al, out of the 10 cases 8 were males and 2 were females. (Ram R, Swarnalatha G, Prasad N, Dakshinamurty KV. Paraphenylene

diamine ingestion: An uncommon cause of acute renal failure J Postgrad Med 2007;53:181-2)

In the study by Krishnaswamy Sampathkumar (Indian Journal Of Critical Care Medicine-2007; Volume 11, 212-214) both the cases were females.

A three and half year study of hair dye poisoning by Anurag Chrispal et al (TROPICAL DOCTOR > VOLUME 40, NUMBER 2 > PP. 100-103) there were 13 cases and 11 out of 13 cases were females.

Reddy et al studied 10 cases of hair dye poisoning between January 2006 and February 2007. 6 were males and 4 were females (IS Reddy, VK Somani, G Swarnalata, Sanjay Maitra; Indian Academy Of Dermatology, Venereology And Leprology 2010; 76; 4 ; 400-403)

A total of 150 adult patients who had PPD poisoning were admitted to Khartoum Teaching Hospital (KTH) Sudan. 120 of these were females (80%) (Abderlraheem MB, El-Tigani MA, Hassan EG, Ali MA, Mohamed IA, Nazik AE. Acute renal failure owing to paraphenylene diamine hair dye poisoning in Sudanese children. Ann Trop Paediatr. 2009 Sep;29(3):191-6)

Generally suicidal tendency is more in women and suicidal hair dye poisoning is not an exception to this.

4. Clinical features in our study are rhabdomyolysis(64%) angioedema(53%) chocolate brown urine(44%) shock(39%) ECG changes(39%) renal failure(30%) and sepsis(5%)

Kallel *et al* studied 19 patients with PPD intoxication in Tunisia over a six-year period. 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%). Rhabdomyolysis was seen in all the patients. ARF was seen in 47.4% and hyperkalemia in 26.3%(Kallel H, Chelly H, Dammak H, Bahloul M, Ksibi H, Hamida CB, *et al*. Clinical Manifestations of systemic paraphenylene diamine intoxication. J Nephrol 2005;18:308-11)

Suliman *et al.*, studied 150 patients who presented with PPD poisoning in Sudan over a 10-year period. 60% had ARF. Angioneurotic edema was encountered in 68%. (Suliman SM, Fadlalla M, Nasr ME, Beliela MH, Fesseha S, Babiker M, et al. Poisoning with hair-dye containing paraphenylene diamine: Ten years experience. *Saudi J Kidney Dis Transpl.*1995;6:286–9.)

In an observational study of hair dye poisoning in Hyderabad, India, acute renal failure developed in 100% of patients due to rhabdomyolysis. (Soni S, Nagarik A, Gopalkishan A, Anuradha Supervasmol 33 poisoning- Abstract presented at the 38th Annual conference of Indian society of Nephrology; 2007)

The frequency of Rhabomyolysis, chocolate brown urine (Myoglobinuria, renal failure in our study is much lower than the other studies. The reason may be the higher percentage of deaths(65%) in our study occurred in the first 2 days while the above mentioned symptoms usually appear on the third or fourth day.

5. The mortality was 38.9% of the cases.

A three and half year study of hair dye poisoning by Anurag Chrispal et al (TROPICAL DOCTOR > VOLUME 40, NUMBER 2 > PP. 100-103) there were 13 cases and 3 of them died(mortality 23%)

In an observational study of hair dye poisoning in Hyderabad, India, the mortality was high. Six out of the ten patients died (60%)(Soni S, Nagarik A, Gopalkishan A, Anuradha Supervasmol 33

poisoning- Abstract presented at the 38th Annual conference of Indian society of Nephrology; 2007)

In a 10-year retrospective study of PPD poisoning reported to the Poison control centre of Morocco, 374 cases were analysed. There was 21% mortality. (Filali A, Semlali I, Ottaviano V, Fumari C, Corradini D, Soulaymani R. A retrospective study of acute systemic poisoning of paraphenylenediamine (occidental takawt) in Morocco. Afr J Trad. 2006;3(1):142-9)

The average mortality rate over 10 years 10.6 % peaking up to 27% in % in 1995 and declining to 5.5% in 2005 which reflects better care.

(Abderlraheem MB, El-Tigani MA, Hassan EG, Ali MA, Mohamed IA, Nazik AE. Acute renal failure owing to paraphenylene diamine hair dye poisoning in Sudanese children. Ann Trop Paediatr. 2009 Sep;29(3):191-6)

Reddy et al studied 10 cases of hair dye poisoning between January 2006 and February 2007. 6 out of the 10 cases died(Mortality 60%) (IS Reddy, VK Somani, G Swarnalata, Sanjay Maitra; Indian Academy Of Dermatology, Venereology And Leprology 2010; 76; 4 ; 400-403)

Mortality in our study is comparatively high when compared to other studies.

6. Out of the 14 cases 9 cases died of laryngeal edema.

In a 10 year retrospective study of PPD poisoning reported to the Poison control centre of Morocco, 374 cases were analysed. Rhabdomyolysis and acute renal failure were the main contributing factors for the 21% mortality. (Filali A, Semlali I, Ottaviano V, Fumari C, Corradini D, Soulaymani R. A retrospective study of acute systemic poisoning of paraphenylenediamine (occidental takawt) in Morocco. Afr J Trad. 2006;3(1):142-9)

Reddy et al studied 10 cases of hair dye poisoning between January 2006 and February 2007. Out of the 6 cases that died, 2 were due to cardiac arrhythmia; 3 due to renal failure and 1 due to sepsis (IS Reddy, VK Somani, G Swarnalata, Sanjay Maitra; Indian Academy Of Dermatology, Venereology And Leprology 2010; 76; 4 ; 400-403)

In a four year (2002 to 2006) study of hair dye poisoning by Ram et al, out of the 10 cases, one patient died of ARDS (Ram R, Swarnalatha G, Prasad N, Dakshinamurty KV. Paraphenylene

diamine ingestion: An uncommon cause of acute renal failure J Postgrad Med 2007;53:181-2)

7. Out of the 14 deaths, 9 cases died of laryngeal edema in the first 2 days; 2 cases died of ARDS on the 4th day; one case died of renal failure on the 5th day and another case on 7th day; one case died of sepsis on the 28th day.

Data regarding the day of death and cause of death is not widely available. From out study it is obvious that laryngeal edema occurs between 1-2 days, which is due to hypersensitivty reaction(anaphylaxis). One should be with vigil about angioedema in hair dye poisoning, if it does not occur on the first day it may occur on the second day. Everything should be kept ready for emergency tracheostomy to secure the airway.

ARDS occurs between 3rd to 7th day. So patient must have an easy accessibility to the ventilator.

Rhabdomyolysis, hemolysis and their complication like renal failure are subacute and present later between 3rd to 7th day. Renal parameters and CPK levels should be monitored throughout the hospital stay.

Sepsis may not be the complication of PPD poisoning per se. It may be nosocomial due to the invasive management which asserts the importance of aseptic precautions and antibiotic prophylaxis.

8. Chemical analysis was negative in 10 out of 14 cases(71.4%). PPD is detected qualitatively by diazotisation and coupling reaction. Quantitative detection is by thin layer chromatography. The reason may be due to the quick metabolism of PPD.

9. HPE kidney was done for the 14 cases died of hair dye poisoning. 6 were normal; 5 showed myoglobin casts and 3 showed changes of ATN.

In cases died of laryngeal edema which occurs in first 2 days, the HPE was normal, because myoglobinuria and ATN usually occurs after the 3rd day.

Cases died of ARDS had changes of ATN and myoglobin casts, because both occur after the 3rd day.

Renal biopsy was done for the cases which developed renal failure. Biopsy was done after the recovery. 7 biopsies were performed. All were normal.

Suliman *et al.*, studied 150 patients with PPD poisoning in Sudan over a 10-year period. Sixty percent had ARF requiring dialysis whereas 30% had ARF which recovered with conservative measures. All patients recovered renal function after a mean period of 15 days of dialysis. Interestingly renal biopsy was undertaken after recovery of renal function in 20 patients. Not surprisingly, the histology was normal in almost all cases(Suliman SM, Fadlalla M, Nasr ME, Beliela MH, Fesseha S, Babiker M, et al. Poisoning with hair-dye containing paraphenylene diamine: Ten years experience. *Saudi J Kidney Dis Transpl.*1995;6:286–9.)

10. In our study there is a significant association between the quantity consumed and the outcome. The mean quantity consumed by the cases who were alive is 36ml and those cases who succumbed is 66ml. But much importance may not be given to this because most of the effects of the poison is due to the hypersensitivity and the inflammatory reaction evoked which is not dose dependent. Also when the question about the quantity was asked, the answer has a considerable degree of subjective variation.

11. Cases that developed shock and angioedema had a significantly higher rate of mortality than those cases where these features were absent. Out of the 14 cases which died, 9 cases presented with shock, whereas only 5 out of 22 cases which were alive had features of shock. Only 7 out of 22 alive cases developed angioedema, where 12 out of 14 died cases developed angioedema. Thus our study indicates the grave prognosis if a case presents with angioedema and/or shock.

In a study over 3 and half years (January 2006 to July 2009) 13 cases were studied, out of which 5 cases died. 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 tracheostomy/intubation at the primary centre; seizures; established renal failure; and those who subsequently require dialysis, mechanical ventilation or intensive care A three and half year study of hair dye poisoning by (Anugrah Chrispal, Anisa Begum , I Ramya and Zachariah **,** TROPICAL DOCTOR > VOLUME 40, NUMBER 2 > PP. 100-103)

In a four year (2002 to 2006) study of hair dye poisoning by Ram et al, studied 10 cases of hair dye poisoning and states that

hypotensive shock is associated with poor prognosis(Ram R, Swarnalatha G, Prasad N, Dakshinamurty KV. Paraphenylene diamine ingestion: An uncommon cause of acute renal failure J Postgrad Med 2007;53:181-2)

12. In our study gastric lavage is the only significant treatment modality that is associated with better outcome. 17 out of the 22 live cases had been given timely gastric lavage whereas only 6 out of 14 dead cases had been given timely gastric lavage. Tracheostomy is the live saving measure once angioedema develops. Out of the 14 dead cases tracheostomy was done for 6 cases. After tracheostomy, 2 cases died of renal failure, 3 cases died of ARDS and 1 case died of sepsis. Mechanical ventilation was given for 4 out of the 22 live cases which developed ARDS. Hemodialysis is given for 8 out of 22 live cases and 3 out of 12 dead cases. After HD one case died of sepsis. And during HD 2 cases died of renal failure.

Suliman *et al* studied 150 patients who presented with PPD poisoning in Sudan over a 10-year period. Sixty percent had ARF requiring dialysis whereas 30% had ARF which recovered with conservative measures. Angioneurotic edema was encountered in

68% and emergency tracheostomy had to be done in 15.8%. All patients recovered renal function after a mean period of 15 days of dialysis (Suliman SM, Fadlalla M, Nasr ME, Beliela MH, Fesseha S, Babiker M, et al. Poisoning with hair-dye containing paraphenylene diamine: Ten years experience. *Saudi J Kidney Dis Transpl.*1995;6:286–9)

Anurag chrispal et al state that early therapy with tracheostomy and aggressive forced diuresis are essential in order to prevent the high mortality associated with this toxin after he studied 13 cases over 3 and half years (January 2006 to July 2009) (Anugrah Chrispal, Anisa Begum , I Ramya and Zachariah , TROPICAL DOCTOR > VOLUME 40, NUMBER 2 > PP. 100-103)

7. SUMMARY AND CONCLUSION

- From our study we learn that the incidence of hair dye poisoning is increasing. Health authorities should call for the prevention of the use and trade of PPD in the market. PPD containing hair dyes should be classified under SCHEDULE H substance so as to restrict its supply and sale. There is a strong case for the regulation and restriction of sale of para-phenylenediamine.

- More than 80% of the cases are in the adolescent age group and more than 80% of the cases were females. Efforts should be directed to create awareness among people by undertaking education of parents and caretakers, especially those taking care of children and adolescent age groups, by distributing educational leaflets, brochures and handouts on proper storage, use and disposal of various chemicals used at home. Campaigns in the media and educational programmes could also help in highlighting the preventive measures.

- Rhabdomyolyis is the most common feature followed by angioedema. Laryngeal edema is the most common cause of death in first 2 days and Acute renal failure is the most common cause

after 5 days. Whenever the characteristic triad of stridor due to upper airway edema, rhabdomyolysis and acute renal failure develops in a poisoning, hair dye should be considered. Early airway protection, alkaline diuresis and dialysis are the three management strategies helpful in this situation. There is no specific antidote available. Awareness about this condition is helpful in early intervention to reduce mortality.

- There is a significant association between quantity consumed and outcome. Cases that developed angioedema and shock have poor prognosis. Timely Gastric lavage is associated with better outcome. There is a National Poisons Information centre functioning at the AIIMS, New Delhi, which functions round the clock, to direct medicos, paramedical staff and care takers in case of emergency. Such a Poison Information centre should be established in each district Headquarter hospitals and qualified personel should be appointed for early detection and management of various poisons to save precious lives. Also, it is important that the medical fraternity be aware of this devastating poisoning because the toxin involved is available quite freely and used extensively

- Mortality rate is 38.9%. The scale of the problem is enormous due to the increased incidence of morbidity and mortality. Improvement in the preventive and management programme can be brought about by identification of high risk circumstances, susceptible groups within the population, chemical substances and commercial products involved in poisoning cases in the community

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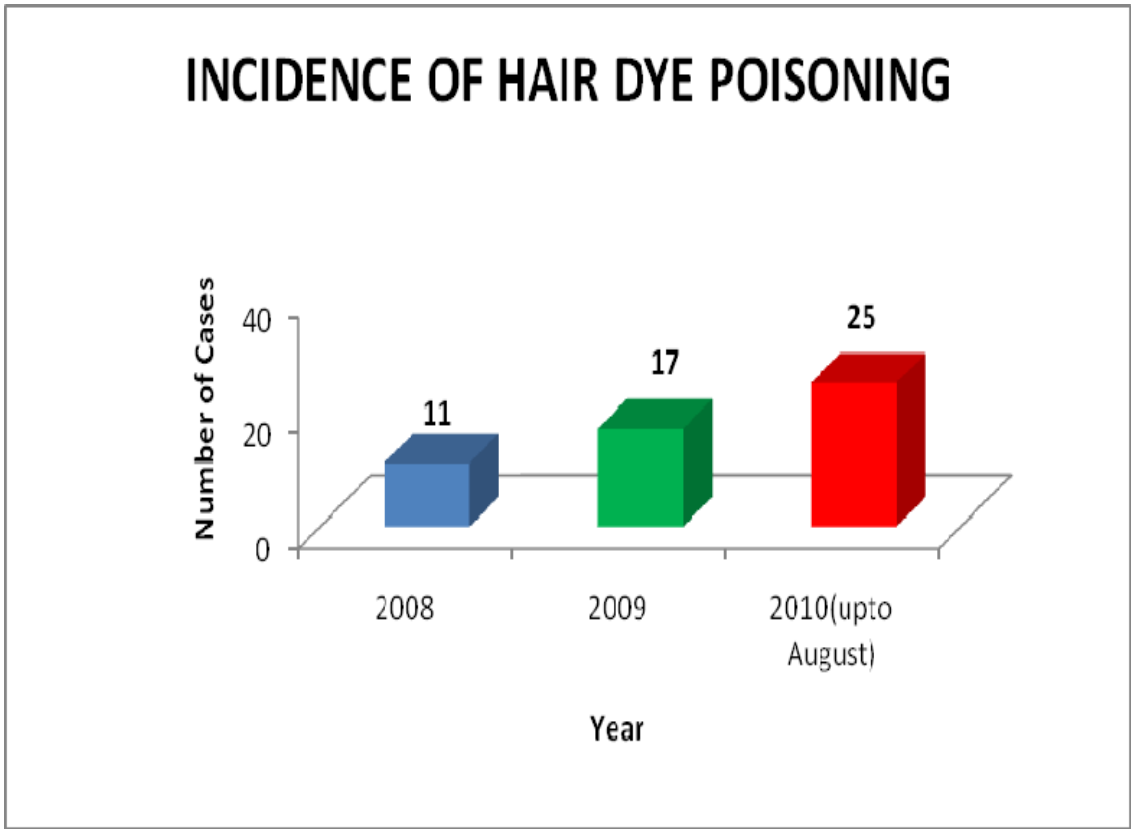
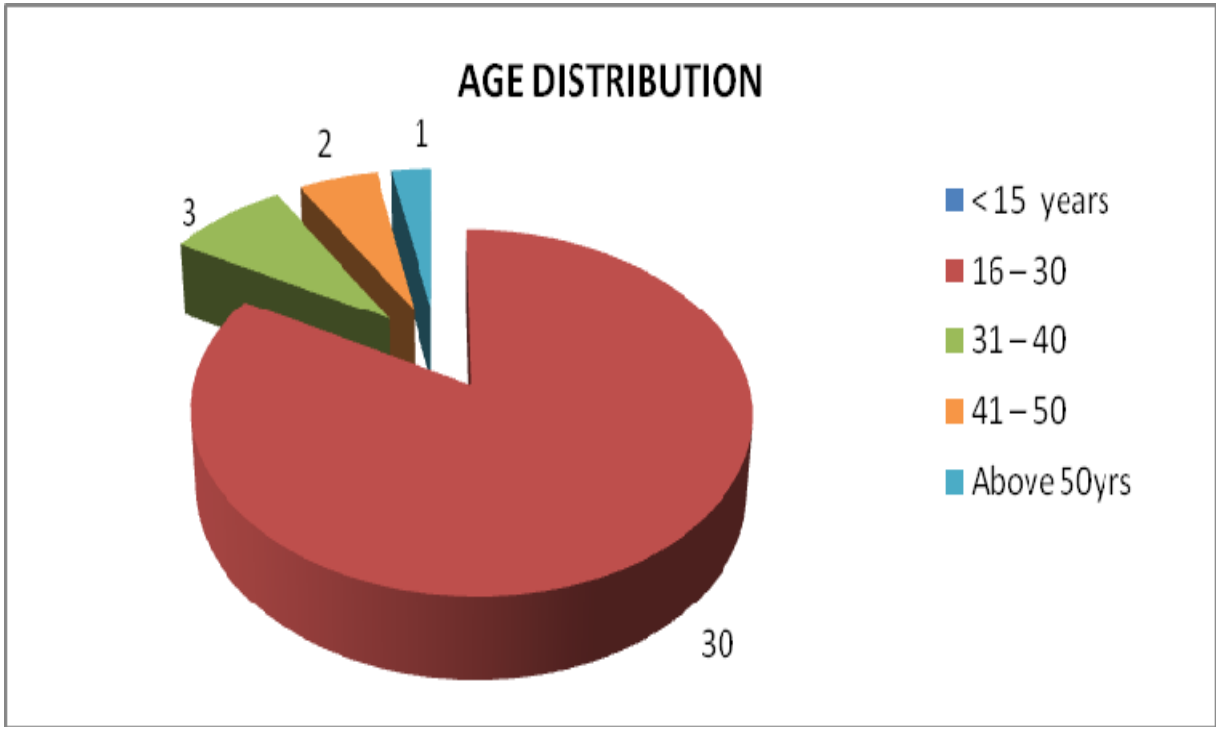
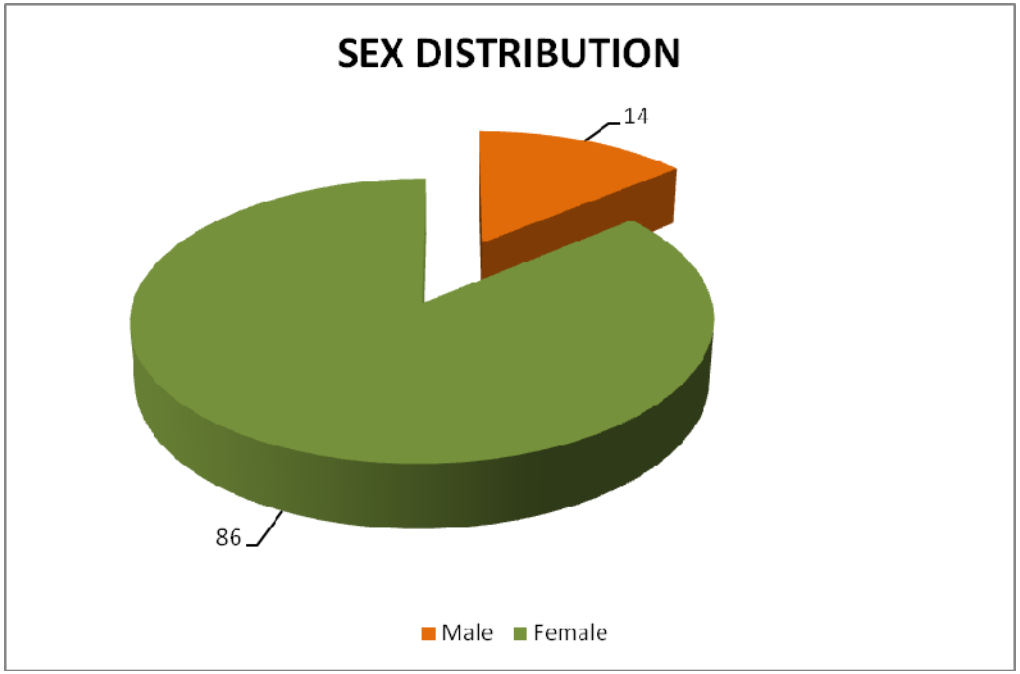
GRAPH 1: INCIDENCE OF HAIR DYE POISONING



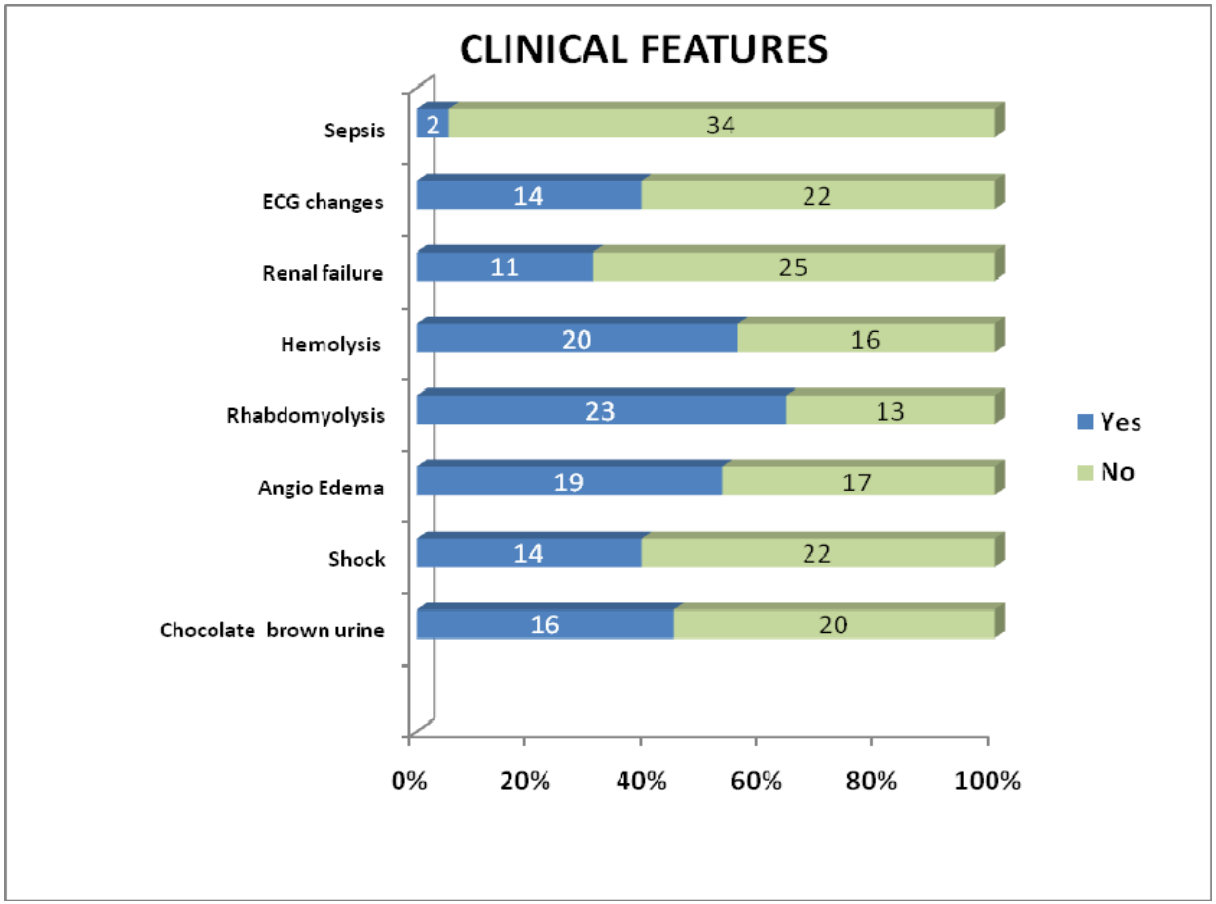
GRAPH 2: AGE DISTRIBUTION



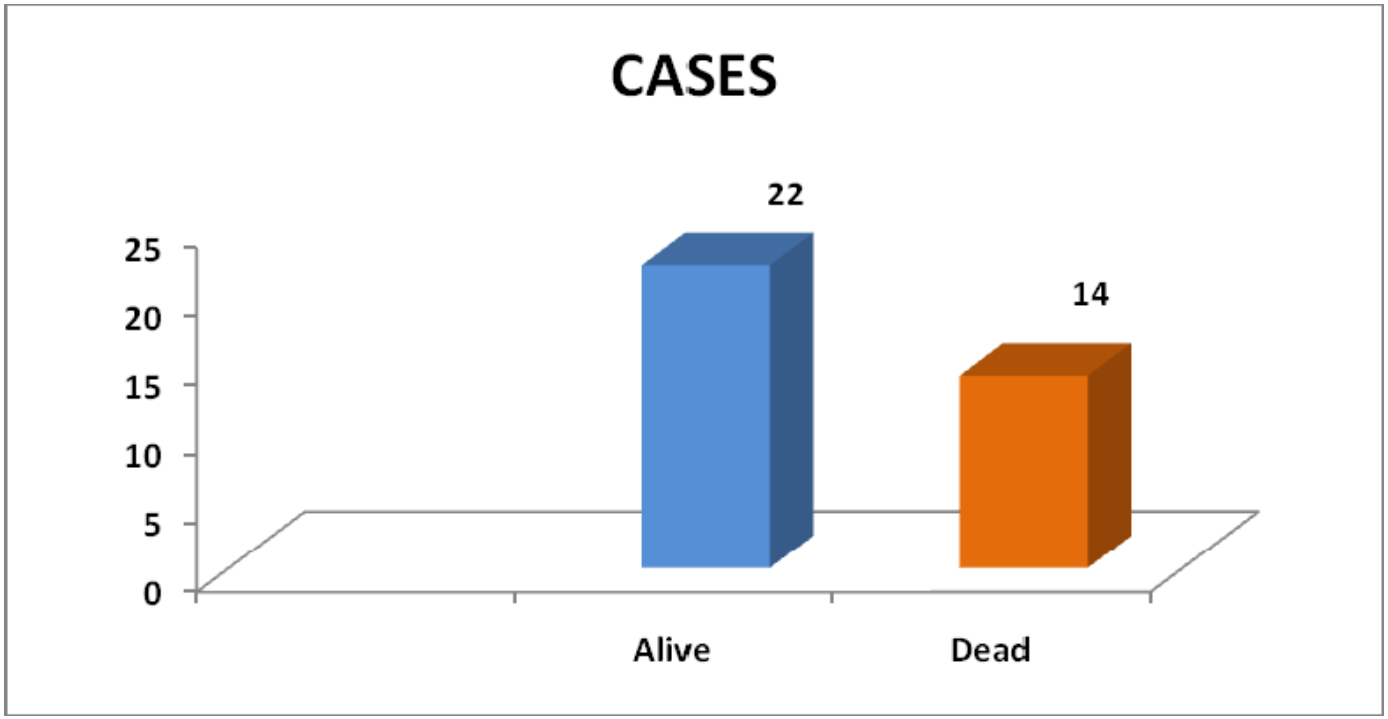
Graph 3: SEX DISTRIBUTION



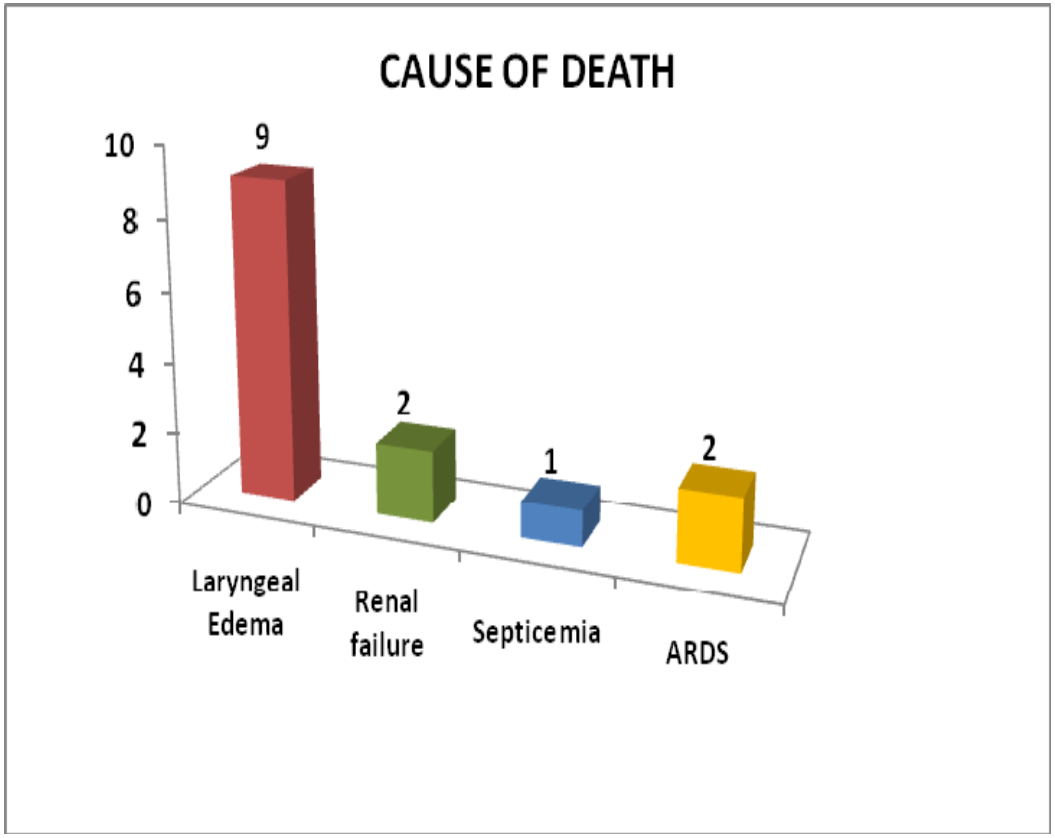
GRAPH 4: CLINICAL FEATURES



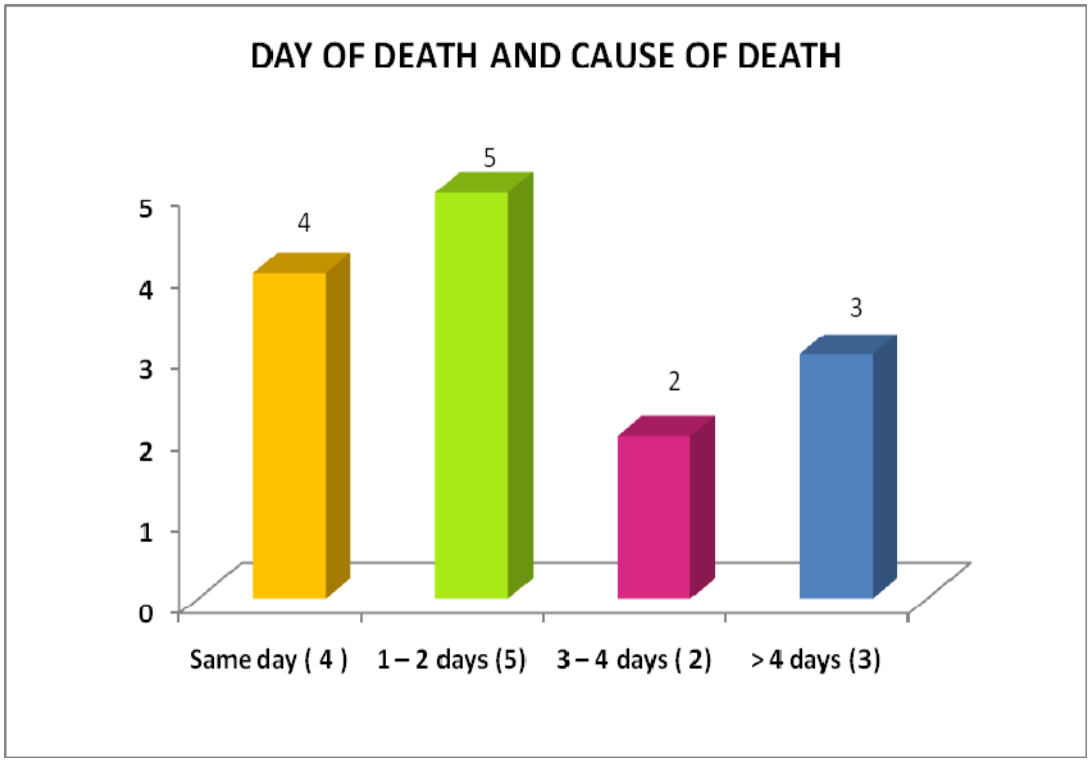
GRAPH 5: MORTALITY



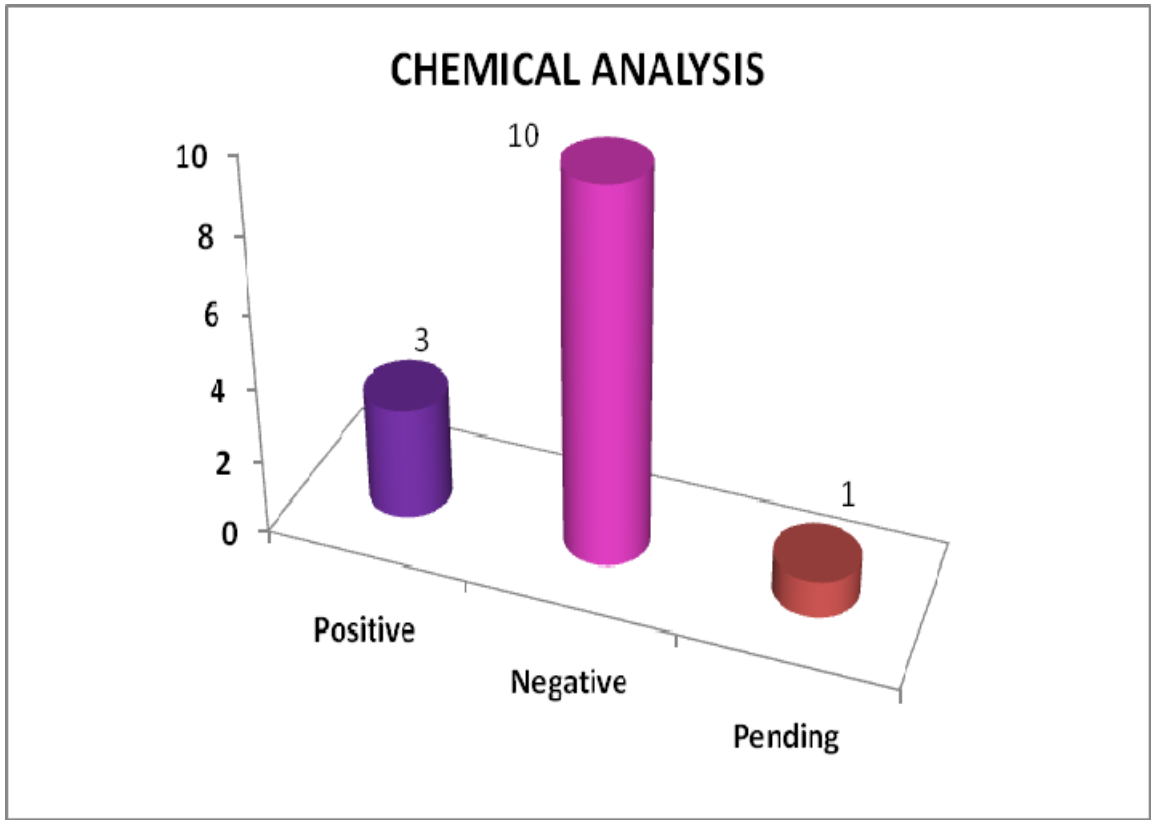
Graph 6: CAUSE OF DEATH



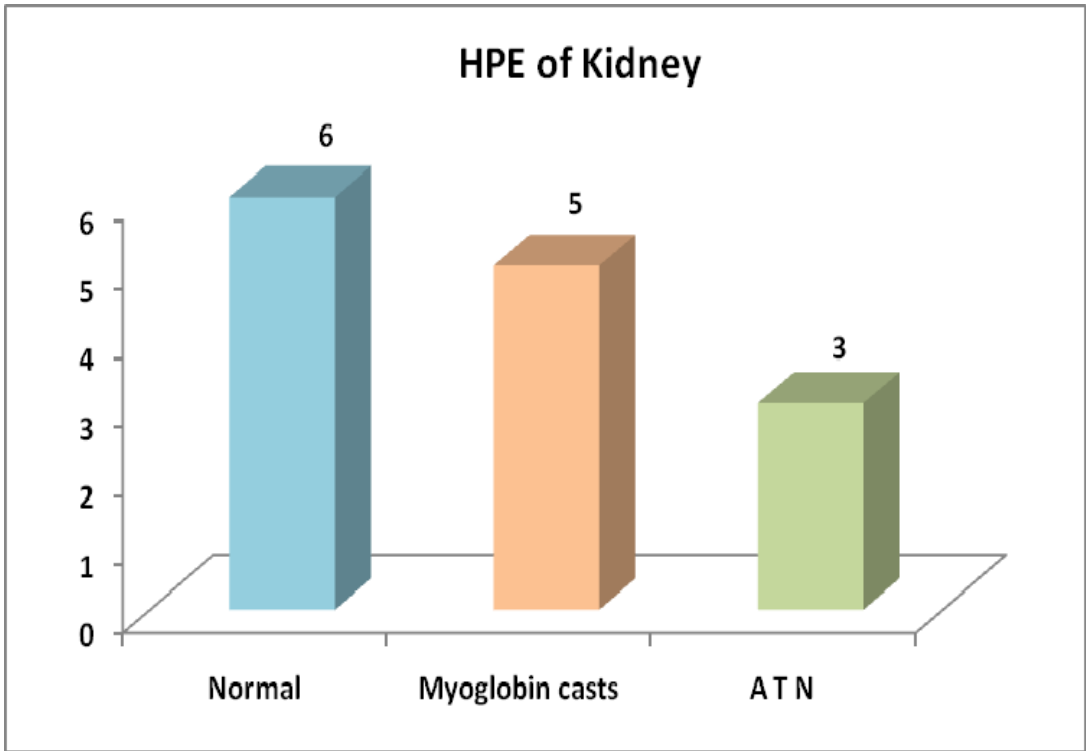
GRAPH 7: DAY OF DEATH AND CAUSE OF DEATH



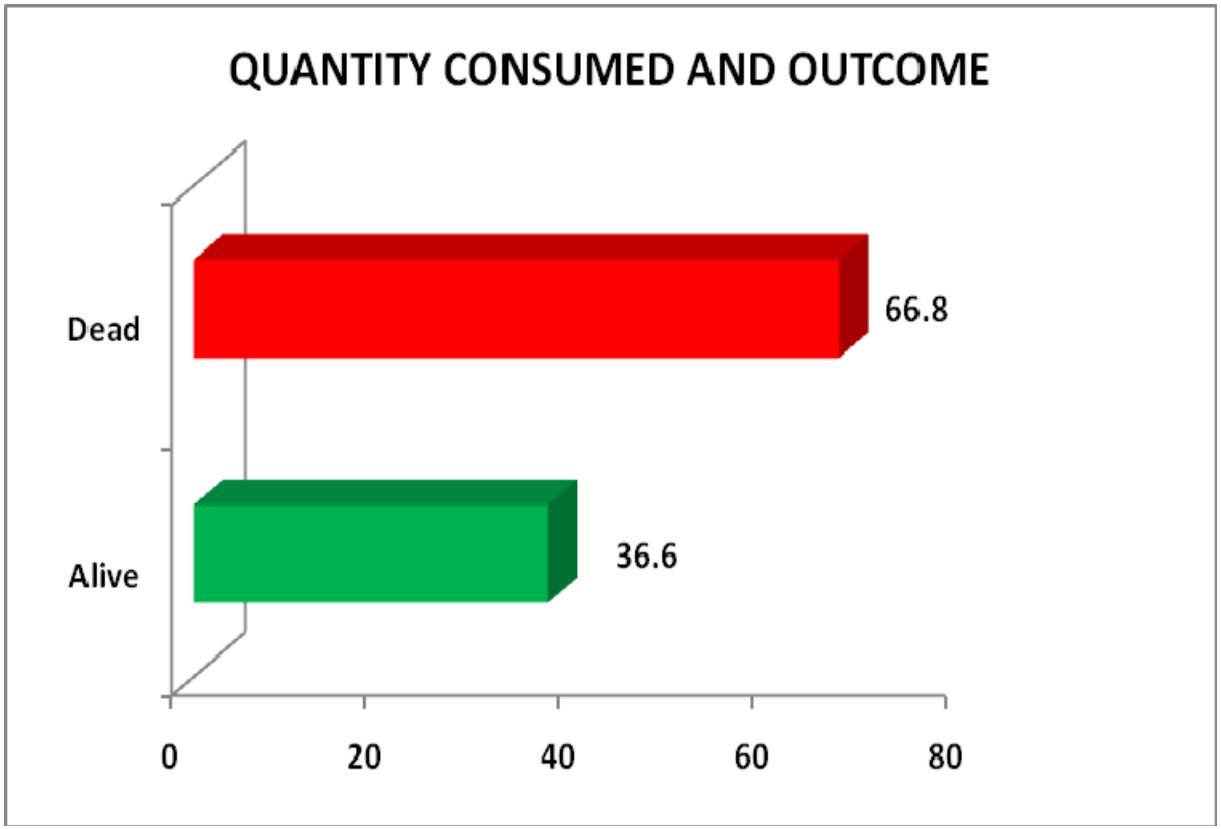
GRAPH 8: CHEMICAL ANALYSIS



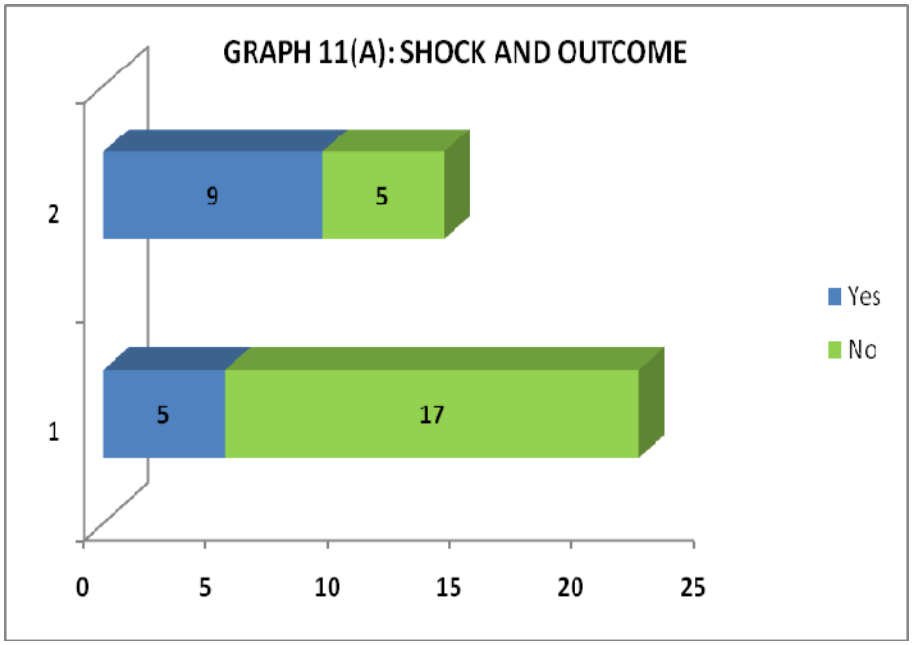
GRAPH 9: HPE OF KIDNEY



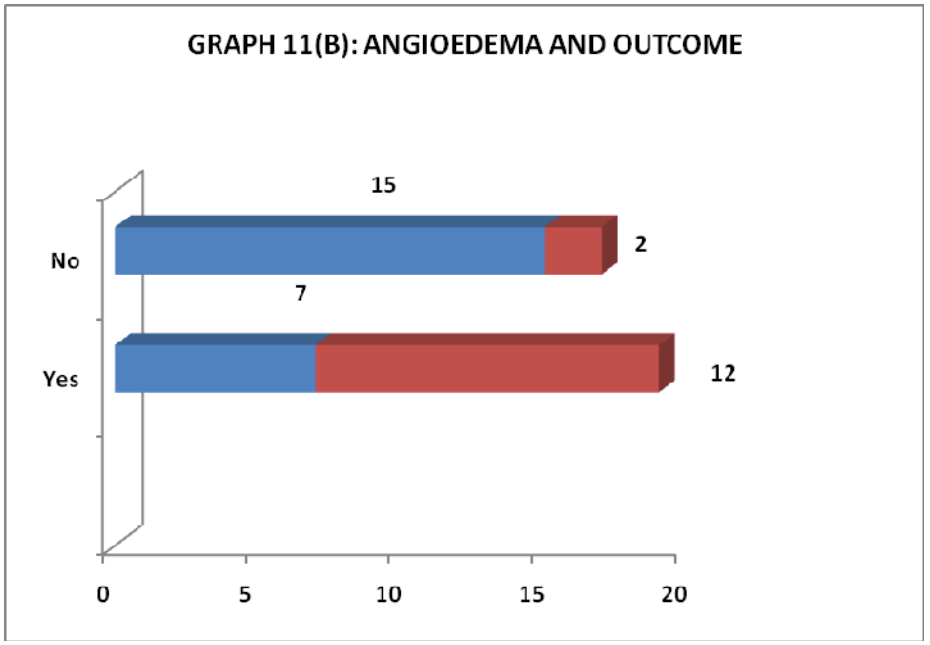
GRAPH 10: QUANTITY CONSUMED AND OUTCOME



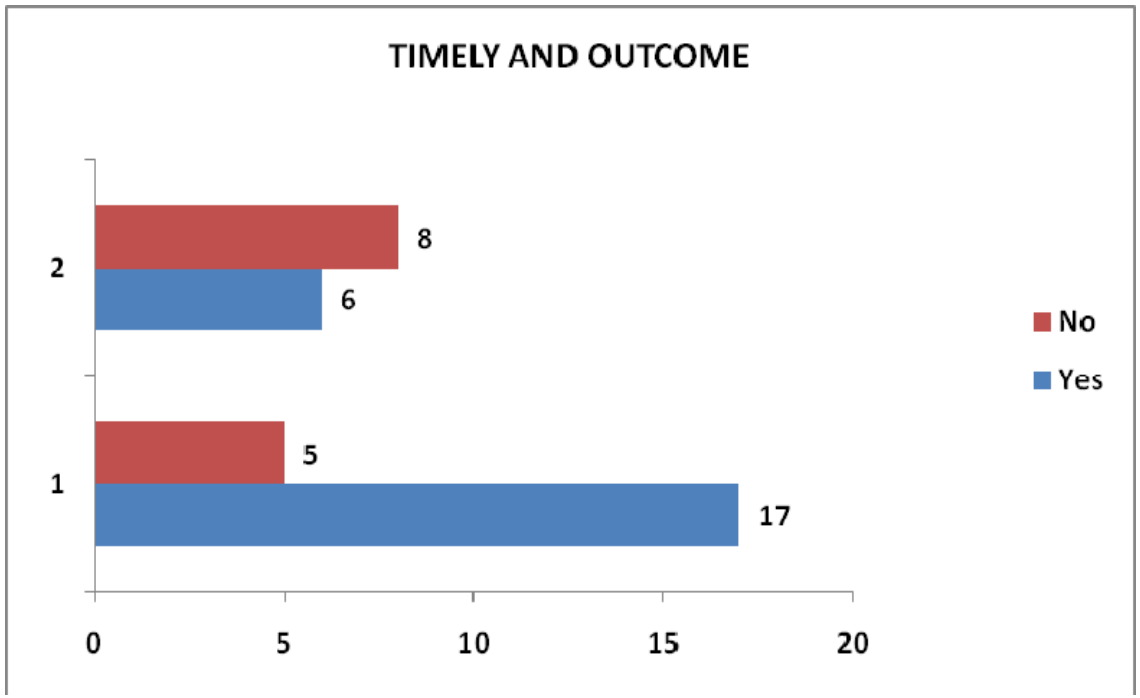
GRAPH 11(A): SHOCK AND OUTCOME



GRAPH 11(B): ANGIOEDEMA AND OUTCOME



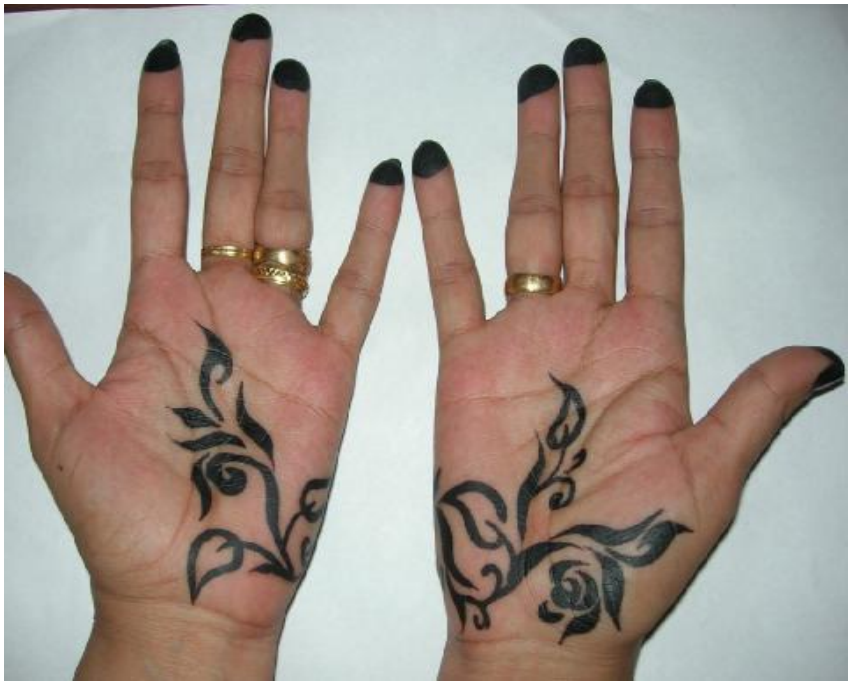
GRAPH 12: TIMELY GL AND OUTCOME



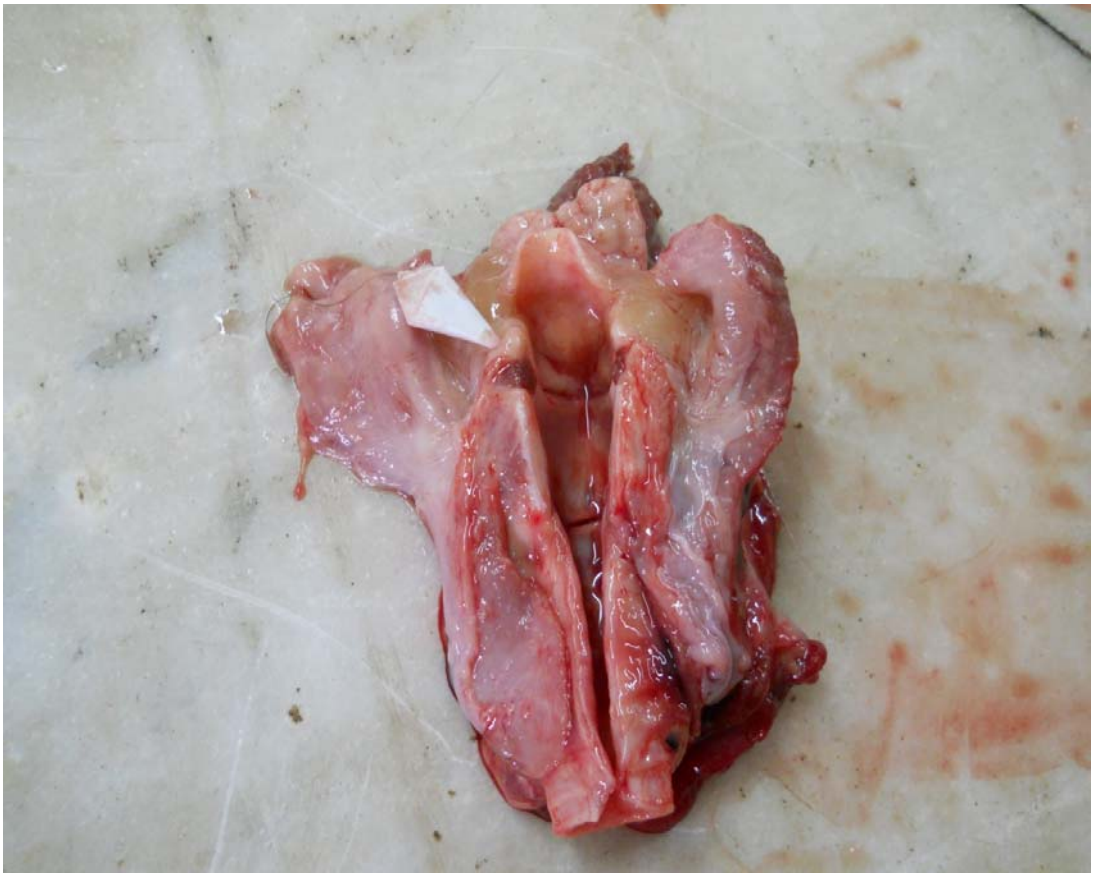
PARAPHENYLENE DIAMINE CRYSTALS

PARAPHENYLENE DIAMINE – TURNS BLACK ON EXPOSURE TO AIR

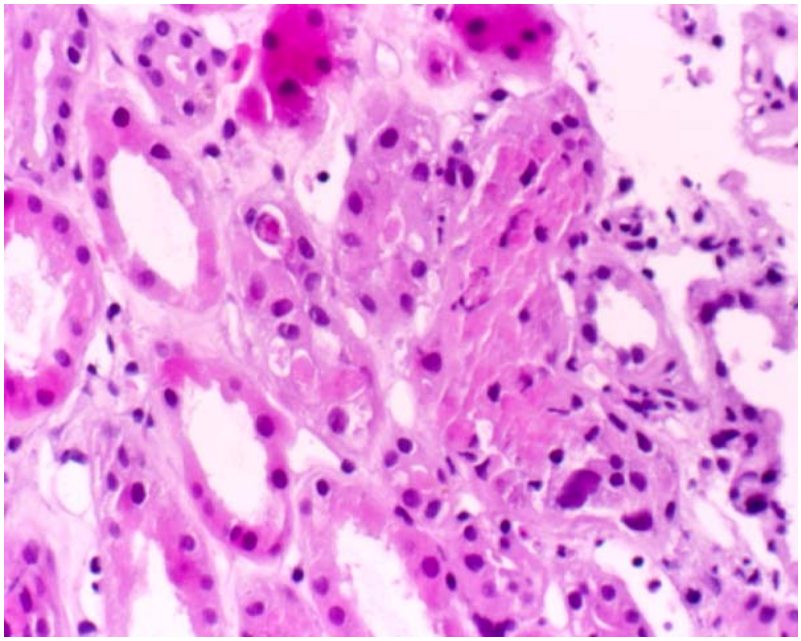
USE IN SUDAN AND ARAB COUNTRIES



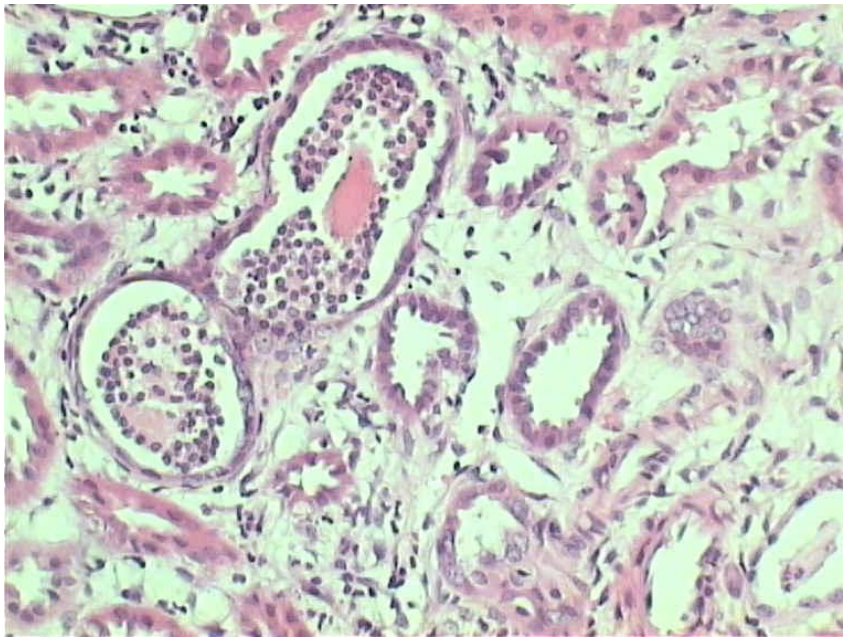
LARYNGEAL EDEMA



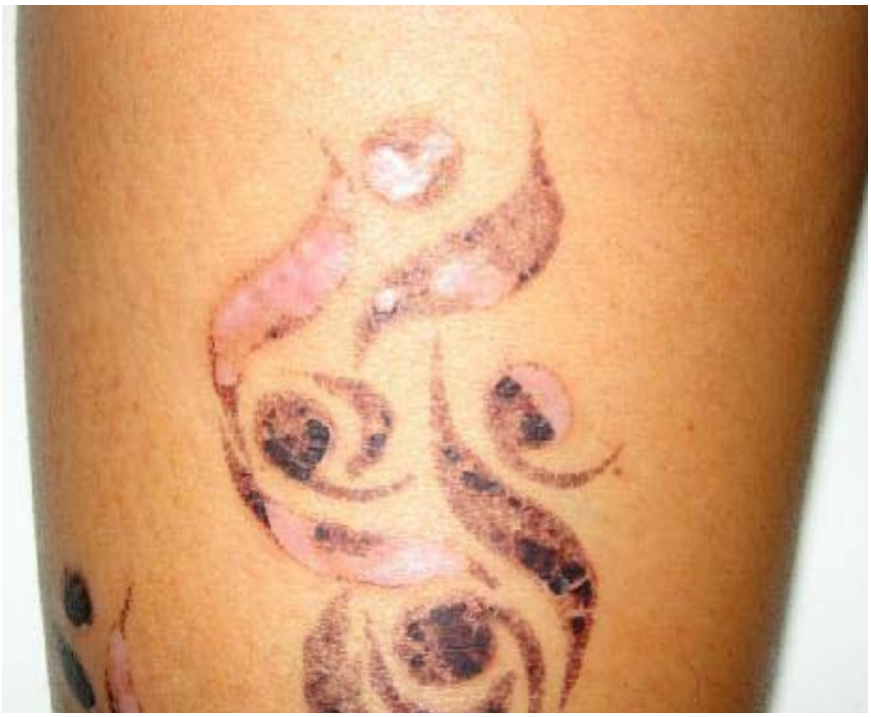
ACUTE TUBULAR NECROSIS



MYOGLOBIN CASTS



DERMATITIS



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HAIR DYE POISONING - ALIVE CASES (1)

S.NO	NAME	SEX	AGE	DOA	DOD	SOCIO ECONOMIC STATUS	MARITAL STATUS	NATIVITY	H/O PSYCHIATRIC ILLNESS	MANNER OF CONSUMPTION	QUANTITY	CHOCOLATE BROWN URINE	SHOCK
1	SELVI	F	25	14/7/2009	19/7/2009	L	MARRIED	TAMIL	NO	INTENTIONAL	20ML	NO	NO
2	VINOTHINI	F	17	12/8/2009	30/8/2009	M	SINGLE	SRILANKAN	NO	INTENTIONAL	50ML	YES	NO
3	VADIVUKKARASI	F	20	24/11/2009	30/11/2009	L	SINGLE	TAMIL	NO	INTENTIONAL	50ML	YES	NO
4	IYNGARAN	M	28	29/11/2009	17/12/2009	L	SINGLE	TAMIL	NO	INTENTIONAL	50ML	YES	YES
5	NATHIYA	F	20	1/12/2009	8/12/2009	M	SINGLE	TAMIL	NO	INTENTIONAL	50ML	NO	NO
6	SINDHUJA	F	19	5/1/2010	9/1/2010	M	SINGLE	TAMIL	NO	INTENTIONAL	25ML	NO	NO
7	KALIAMMAL	F	25	10/1/2010	19/1/2010	L	MARRIED	TAMIL	NO	INTENTIONAL	20ML	NO	NO
8	SUJATHA	F	33	12/2/2010	3/3/2010	M	MARRIED	TAMIL	NO	INTENTIONAL	50ML	YES	YES
9	BANUMATHY	F	24	28/2/2010	1/3/2010	M	MARRIED	TAMIL	NO	INTENTIONAL	20ML	NO	NO
10	JANARTHANAN	M	24	3/3/2010	23/3/2010	L	SINGLE	TAMIL	NO	INTENTIONAL	50ML	YES	NO
11	MYTHILI	F	22	11/3/2010	17/3/2010	M	MARRIED	TAMIL	NO	INTENTIONAL	20ML	YES	NO
12	GANESH	M	21	12/3/2010	19/3/2010	M	SINGLE	TAMIL	NO	INTENTIONAL	50ML	NO	NO
13	ANNALAKSHMI	F	25	2/4/2010	7/4/2010	L	MARRIED	TAMIL	NO	INTENTIONAL	20ML	NO	NO
14	POORNAPANDI	F	22	8/4/2010	29/4/2010	L	MARRIED	TAMIL	NO	INTENTIONAL	50ML	YES	YES
15	PANDISELVI	F	18	23/4/2010	16/5/2010	L	SINGLE	TAMIL	NO	INTENTIONAL	50ML	YES	NO
16	MYTHILI	F	23	7/5/2010	2/6/2010	M	MARRIED	TAMIL	NO	INTENTIONAL	50ML	YES	YES
17	SELVAKUMARI	F	20	6/7/2010	29/7/2010	L	MARRIED	TAMIL	NO	INTENTIONAL	50ML	YES	YES
18	SELVAKUMARI	F	31	19/7/2010	23/7/2010	L	MARRIED	SRILANKAN	YES	INTENTIONAL	20ML	NO	NO
19	THAYAMMAL	F	30	20/7/2010	24/7/2010	L	MARRIED	TAMIL	NO	INTENTIONAL	20ML	NO	NO
20	RAJESWARI	F	30	27/7/2010	31/7/2010	M	MARRIED	TAMIL	NO	INTENTIONAL	50ML	NO	NO
21	SIVAGAMI	F	16	11/8/2010	18/8/10	M	SINGLE	TAMIL	NO	INTENTIONAL	20ML	NO	NO
22	JEEVA	F	22	10/8/2010	15/8/2010	M	MARRIED	TAMIL	NO	INTENTIONAL	20ML	NO	NO

HAIR DYE POISONING - DEAD CASES (1)

S. NO	NAME	SEX	AGE	DOA	SOCIO ECONOMIC STATUS	MARITAL STATUS	NATIVITY	H/O PSYCHIATRIC ILLNESS	MANNER OF CONSUMPTION	QUANTITY	CHOCOLATE BROWN URINE	SHOCK	ANGIO - EDEMA	RHABDO-MYOLYSIS	RENAL FAILURE
1	RANI	F	30	12/7/2009	L	MARRIED	TAMIL	NO	INTENTIONAL	50ML	NO	YES	YES	YES	NO
2	SARAVANAKUMARI	F	22	21/7/2009	L	MARRIED	TAMIL	NO	INTENTIONAL	50ML	YES	NO	YES	YES	YES
3	VIJAYALAKSHMI	F	23	1/8/2009	M	MARRIED	TAMIL	NO	INTENTIONAL	70ML	NO	YES	YES	NO	NO
4	IRULAN	M	65	4/10/2009	L	MARRIED	TAMIL	NO	INTENTIONAL	50ML	YES	NO	NO	YES	NO
5	GOPAL	M	48	26/10/2009	L	MARRIED	TAMIL	NO	INTENTIONAL	70ML	NO	YES	YES	NO	NO
6	VIMALA	F	18	9/11/2009	M	SINGLE	TAMIL	NO	INTENTIONAL	50ML	YES	NO	YES	YES	YES
7	MAHALAKSHMI	F	20	27/1/2010	M	SINGLE	TAMIL	NO	INTENTIONAL	70ML	NO	YES	YES	NO	NO
8	BOOMI DEVI	F	30	7/3/2010	L	MARRIED	SRILANKA	NO	INTENTIONAL	50ML	YES	YES	YES	YES	NO
9	SANTHANA MARI	F	16	22/03/2010	L	SINGLE	TAMIL	NO	INTENTIONAL	50ML	YES	NO	NO	YES	YES
10	KILDA	F	35	15/4/2010	L	MARRIED	SRILANKA	YES	INTENTIONAL	75ML	NO	YES	YES	NO	NO
11	LAKSHMI	F	45	16/4/2010	M	MARRIED	TAMIL	NO	INTENTIONAL	75ML	NO	YES	YES	NO	NO
12	ANUSHA STEFFI GRAFF	F	18	9/6/2010	M	SINGLE	SRILANKA	NO	INTENTIONAL	75ML	YES	NO	YES	YES	NO
13	CHELLATHAI	F	19	21/6/2010	M	SINGLE	TAMIL	NO	INTENTIONAL	100ML	NO	YES	YES	NO	NO
14	DHANALAKSHMI	F	30	19/7/2010	M	MARRIED	TAMIL	NO	INTENTIONAL	100ML	NO	YES	YES	NO	NO