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SUPERVASMOL POISONING- STUDY IN RIMS, KADAPA

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ABSTRACT

Supervasmol 33, a hair dye which is an emerging mode of suicidal poisoning in India, especially few districts in Andhra Pradesh. The major life threatening compound in this hair dye is para-phenylene- diamine (PPD). A study period of 12months with total cases attending casualty from January 2014-December2015 is being conducted by Department of ENT, RIMS Govt. Hospital, Kadapa. Majority of the patients consuming are females in their 2nd & 3rd decade and the majority presenting with angioneurotic edema with stridor, rhabdomyolysis with Acute kidney disease after consumption of more than 50ml. No specific antidote for Para phenylene diamine(PPD), hence management is only symptomatic &supportive with immediate tracheostomy.

1. INTRODUCTION

Super vasmol 33 is an emulsion based hair dye commonly used in India. The main ingredient of the dye is PPD. and a mixture of other chemicals that can damage the respiratory, muscular, renal, and hepatic systems and leads to death .The purpose of the study is to share our experience on clinical presentation, and outcomes of hair dye poisoning in a rural part of Andhra Pradesh.

Ingredients of Super vasmol 33:

Para phenylene diamine (<4%), Propylene glycol, Light liquid paraffin, Ceto stearyl alcohol, Sodium lauryl sulphate liquid, EDTA disodium, Resorcinol, Herbal extracts, Preservative and perfumes, Water

Propylene glycol - is a phenol derivative and commonly used as a solvent

Resorcinol - being a phenol is corrosive and also causes methamoglobinemia & renal toxicity. EDTA causes hypocalcaemia.

2. MATERIALS AND METHODS

A hospital based prospective study has been conducted in the year of 2014 i.e. from January 2014 to December 2014. Patients attending to casualty with consumption of supervasmol 33 keshkala were included in the study. The incidence of patients who developed severe neck, tongue base edema and finally stridor requiring emergency tracheostomy and comparing the incidence of males and females who consumed supervasmol 33, their age group and who have more incidence of developing neck edema is being observed in this study.

3. RESULTS-

Total numbers of cases admitted in causality in a 12 months period of January 2014 to December 2014 were 386. Out of them male were 97 and female were289 with a female preponderance of 74 %(Figure-1). Out of 386 admissions 10 patients died within 24 to 48 hours (Figure-3).

Majority of the patients observed were females who have undergone tracheostomy frequently with commonest age group of 16-30years (Table-1). Most common age group of males presenting with poisoning belongs to the above age group (Table-2). Patients present with severe angioneurotic edema with swelling presenting mainly at the base of tongue extending to neck, face leading to severe respiratory difficulty requiring emergency tracheostomy. Out of the 386 patients a total of 33 patients undergone tracheostomy, in which 84% were females and 15% were males (Figure-2). 15 patients were managed by endotracheal intubation.

4. DISCUSSION

Supervasmol-33 is emerging as a poison in this area. The constituents of this hair dye include PPD (4%), resorcinol, propylene glycol, ethylenediaminetetraacetic acid (EDTA), sodium, liquid paraffin, cetostearyl alcohol, sodium lauryl sulphate, herbal extracts, preservatives, and perfumes. Some of these ingredients are known toxins with multiorgan effects, while the toxicity profiles of others are not known. The combined effect of the individual compounds may be responsible for its significant morbidity and mortality.

Around the world suicide rates have increased by 60% in the past 50 years. Suicide is one among the three leading causes of death in the age group between 15 to 44 years. Recently hair dye poison becomes one of the important etiological factor5. PPD poisoning is a common health problem in the Middle East, especially Sudan and Morocco. It is also common in India but rare in the west.

An eleven year 1992 to 2002 retrospective study from Morocco described 374 cases of PPD poisoning, majority of patients (54%) were 15-24 years age group and children contributed 11.5%. In Sudan, over a 10 year period (1995 to 2005) 3159 patients were reported PPD poisoning, among 18% of children below the age of 14 years. So most reported cases were from adolescents & adults, but a significant number of cases occur among children[3].

A retrospective study of 25 cases over period of 7 years (2001 to 2008) in Egypt [6,12]. In the present twelve month prospective study 386 cases analyzed. Maximum incidence observed in 2nd &3rd decades which is correlating with PK Jain et al and other studies. There were female preponderance of 77% as per Ayoub Filali et al14, 80.7% as per M. Hamdouk[2] and 74.86% PK Jain et al15. It was 74% in the present study. Suicidal in 78.1% as per Ayoub Filali et al [14] and were 87% as per M.Hamdouk. Ingestion of PPD results in Acute Poisoning with major systemic problem. It results in multisystem involvement of CNS, CVS, Renal & Musculoskeletal [3]. The onset of effects is usually within four to six hours after ingestion. The more severe the poisoning the earlier the onset of effects [16].

Within 4 to6 hours [10,13] 25 to 50ml consumption of Super vasmol 33 can cause laryngeal edema due to direct toxic effect of PPD on mucous membranes8.Rapid development of severe edema of the face, neck, pharynx & larynx with respiratory distress. Tongue is dry &wooden - hard & swollen due to edema, Often requires tracheostomy [1,4,13]. Most common cause of death is acute renal failure and also due to rhabdomyolysis8.

Direct trauma to the tissues by chemicals and causes dyspnoea and asphyxia Intense inspiratory effort secondary to laryngeal edema which might have contributed to over distension of the alveoli. Development of pneumothorax reported following laryngeal edema [17].

Clinical features and presentation mainly depends on the quantity of consumption and the time when treatment is started. Patients who consume less than 25ml may not present with any symptom. Female incidence is more than males. Patients consuming 25to 50ml and more than 50ml may present early with neck edema and other laryngeal symptoms requiring emergency tracheostomy with in 6-12hrs of consumption.

Some may not present with classic symptoms but directly present with acute kidney disease with elevated blood urea and serum creatine profiles requiring forced alkaline diuresis even leading to dialysis.

There is no antidote for PPD Poisoning [13,6,4.] Followed symptomatic approach of treatment [4,6,11].

Those patients were started with early treatment such as injectable steroids, antihistamines and diuretics may not develop symptoms even after consuming high doses.

Rapid acting steroids are injected in bolus doses along with antihistamines and forced alkaline diuresis is done to excrete the toxin via renal pathway.

As the patients may aspirate if gastric lavage is done leading to pulmonary edema, the main route of excretion is via renal.

Mortality rate has been decreased in recent years due to early presentation to hospital and early supportive therapy, endotracheal intubation and emergency tracheostomy.

5. CONCLUSION

Some of the countries considered PPD to be great hazard and its use in hair dyes were banned. Germany banned in early 1900's, subsequently in France and Sweden. However in Japan & India it is still common component in hair dyes. In view of high incidence of poisoning in some parts of India it requires awareness in the rural parts about the complications caused by PPD and also make the manufacturing companies to reduce the concentration of PPD and try to use alternate formulas in manufacturing these products. Aware to be created even in local public health centers regarding the early mode of treatment rather than wait and watch policy. Practicing of gastric lavage to be stopped as it may cause aspiration. Finally banning of these products to be considered if all these measures fail.

Patient presenting with severe neck edema with hard woody protruding tongue who undergone tracheostomy

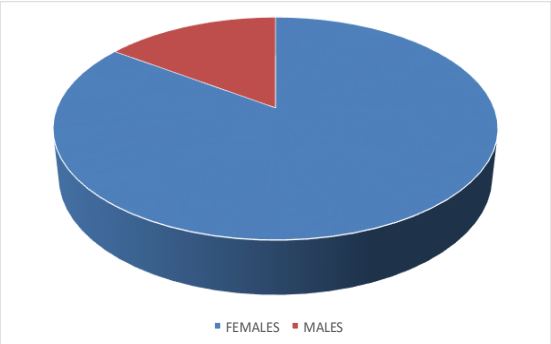
AGE GROUP	NUMBER
0-15	19
16-30	176
31-45	77
46-60	10
60 & above	7
TOTAL	289

AGEWISE DISTRIBUTION IN FEMALES

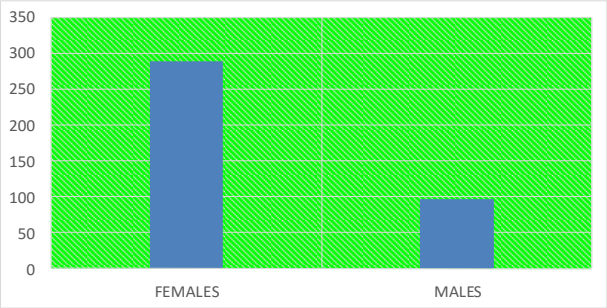
(TABLE-1)

AGE GROUP	NUMBER
0-15	4
16-30	54
31-45	32
46-60	4
60 & above	3
TOTAL	97

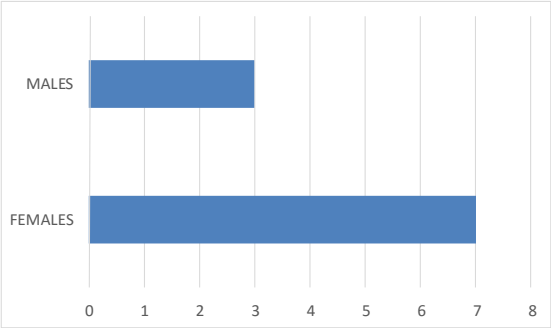
AGE WISE DISTRIBUTION IN MALES (TABLE-2)



INCIDENCE OF TRACHEOSTOMY(FIGURE-2)



INCIDENCE OF MALES AND FEMALES WHO CON-SUMED SUPERVASMOL33 (FIGURE-1)



NO.OF DEATHS(FIGURE-3)

[1]. R. Verma, N. Tewari, S. Jaiswal, V. Rastogi, D.K. Singh and A. Tiwari. Fatal poisoning caused by oral ingestion of a hair dye. The Internet Journal of Emergency and Intensive Care Medicine 2008;11 (1):1092-4051.

[2]. Mohamed I. Hamdouk, Mohamed B. Abdelraheem, Ahbab A. Taha, Mohamed Benghanem and Marc E. De Broe. Paraphenylene diamine hair dye poisoning. Clinical Nephrotoxins 2008;3:871-879.

[3]. Mohamed Abdelraheem, Mohamed Hamdouk, Eduard E Zijlstra. Paraphenylene Diamine (Hair Dye) Poisoning in Children. Arab Journal of Nephrology and Transplantation 2010;3(1): 39-43.

[4]. Sumeet Singla, Sanjeev Miglani, A K Lal, Pulin Gupta, A K Agarwal. Para-phenylenediamine (PPD) Poisoning. JIACM 2005;6(3): 236-8.

[5]. Krishnaswamy Sampathkumar, Sooraj Yesudas. Hair dye poisoning and the developing world. Emerg Trauma Shock 2009 May- Aug;2 (2):129-131.

[6]. Soni SachinS, Nagarik AmitP, Dinaker Manjunath, Adikey GopalKishan, Raman Anuradha. Systemic toxicity of paraphenylenediamine. Indian Journal of Medical Sciences April 2009;63(4):164-166.

[7]. Falguni Parikh. Changing Trends in Poisoning.JAPI November 2009;57:739.

[8]. Namburi Rajendra Prasad, Aparna Rajeshwar Rao Bitla, Suchitra Musturu Manohar, N Harin Devi, PVLN Srinivasa Rao. A Retrospective Study on the Biochemical Profile of Self Poisoning with a Popular Indian Hair Dye. Journal of Clinical and Diagnostic Research 2011 November (Suppl-2);5 (7):1343-1346.

[9]. Krishnaswamy Sampathkumar, Yesudas Santhakumari Sooraj, Rajappannair Prabha Ajeshkumar, Amol Ramesh Mahaldar, Ramakrishnan Muthiah. Rhabdomyolysis due to hair dye poisoning. IJCCM 2007;11(4):212-14.

[10]. Anugrah Chrispal, Anisa Begum, I Ramya and Anand Zachariah. Hair dye poisoning – an emerging problem in the tropics: an experience from tertiary care hospital in South India. Trop Doct April 2010;40:2100-103.

[11]. P Bhargava, P Matthew. Hair Dye Poisoning. JAPI DECEMBER 2007;55:871-2.

[12]. Sawsan A Shalaby, Mohy K Elmasry, Amany EAbd-Elrahman, Manal A Abd-Elkarim. Clinical profile of acute paraphenylenediamine intoxication in Egypt. Toxicology and Industrial Health 2010;26(2):81-87.

[13]. S. Deepak Amalnath, Pradeep Kumar, Vikram Murmu, D. K. S. Subramanyam and T K Dutta. Hair dye poisoning in India. JIPMER (JAN –MARCH) 2007;27(1):34-5.

[14]. Ayoub Filali,Liham Semlali et al., A retrospective study of acute systemic poisoning of paraphenylene diamine in Morocco Afr. J. Trad. CAM 2006;3(1):142-149.

[15]. PK Jain, N Agarwal, P Kumar, NS Sengar, Nutan Agarwal, Asif Akhtar. Hair Dye Poisoning in Bundelkhand Region. JAPI JULY 11;59:415-18.

[16]. Chugh KS, Malik GH,Singhal PC.Acute renal failure following paraphenylene diamine(hair dye) poisoning :report of two cases.

[17]. Subramanian Senthilkumaran, J Ram, Ritesh G Menezes, Shah Sweni, Ponniah Thirumalaikolundusubramanian. Pneumothorax in hair dye poisoning: An unrecognized danger. LUNG INDIA 2011;28(4):323-24.