A STUDY OF CLINICAL, PATHOLOGICAL AND BIOCHEMICAL FINDINGS IN PATIENTS WHO ATTEMPTED SUICIDE BY HAIR DYE SUPERVASMOL 33 INGESTION

Sudheer Babu Devineni¹, Nannam Harshavardhan², Nalamala Baskara Rao³, Vuyyala Chandana⁴

HOW TO CITE THIS ARTICLE:

Sudheer Babu Devineni, Nannam Harshavardhan, Nalamala Baskara Rao, Vuyyala Chandana. "A Study of Clinical, Pathological and Biochemical Findings in Patients who Attempted Suicide by Hair Dye Supervasmol 33 Ingestion". Journal of Evolution of Medical and Dental Sciences 2015; Vol. 4, Issue 48, June 15; Page: 8361-8374, DOI: 10.14260/jemds/2015/1213

ABSTRACT: BACKGROUND: All the patient in the study consumed the hair dye available in the market with the trade name Supervasmol 33. The prevalence of Super vasmol 33 hair dye poisoning has been on a surge for the past 2-3 years as has been observed by the increase in number of cases being admitted into the hospitals. It was observed that the tendency of poisoning by Super vasmol 33 hair dye was more in females than in males and was more in the age group of 15-35 years, as with any other poisoning. METERIALS AND METHODS: Study of Clinical, pathological and biochemical findings in patients who attempted suicide by hair dye ingestion (Supervasmol 3) who were admitted in Government General Hospital/Guntur medical college, Guntur during June 2013 to March 2015 brought to emergency department and those who were admitted into the HDU, ICU and medical wards of the hospital, after the following exclusion criteria were ruled out. **RESULTS:** A significant statistical correlation was found to exist between development of AKI and the levels of CPK in blood, rhabdomyolysis and hyperkalemia. The morbidity rates were 100% for angioedema, 58% for rhabdomyolysis and 32% for acute kidney injury (AKI). Institutional mortality rate was about 8% due to refractory hemodynamic shock. **CONCLUSION:** As the burden of handling Supervasmol 33 hair dye poisoning cases has been recently increasing, primary care physicians, intensive care physicians and nephrologists need to be aware of its clinical manifestation and management

KEYWORDS: Hair dye, Supervasmol 33, Rhabdomyolysis, Hyperkalemia, AKI.

INTRODUCTION: Morbidity and mortality due to poisoning have been known to pose a significant burden on the health care institution for a long time.

Globally suicides rates have increased by 60% in the last fifty years. Suicide now ranked among the three leading causes of death in the age group between 15 and 44years.⁽¹⁾

Poisoning by hair dye ingestion has been known in African countries since its systemic toxicity was first documented by Nott H.W dating back to 1924.⁽²⁾ Many reports have followed since then from African and the Middle East countries.

Suicidal attempts by hair dye ingestion have been rare in the subcontinent of India until recently. Hence, only sporadic case reports have been published and no large studies were available until recently.

There has been surge in the number of such cases being admitted into hospitals (Due to suicidal attempts by Super vasmol 33 hair dye ingestion) in recent years in this part of state also.

Different manifestations were reported by different authors. As varied manifestations were described in different case reports and as there has been a rise in number of cases of hair dye poisoning, this study has taken up to observe the clinical and biochemical profile of Super vasmol 33 poisoning and its outcome.

AIMS AND OBJECTIVES: Study of Clinical, pathological and biochemical findings in patients who attempted suicide by hair dye ingestion.

MATERIALS & METHODOLOGY:

Study Design: Prospective observational study.
Study Population: Patients admitted in Government General Hospital/Guntur medical college Guntur with.
Study Period: From June 2013 to March 2015.

Inclusion Criteria: All patients who were brought to emergency department and those who were admitted into the HDU, ICU and medical wards of the hospital, with the alleged cause of Supervasmol 33 hair dye ingestion were included in the study after the following exclusion criteria were ruled out.

Exclusion Criteria: Patients with the following features were excluded from the study, those who consumed any other toxic substance or alcohol along with hair dye, Known diabetics, hypertensive, asthmatics and epileptics, renal, hepatic, cardiac disease, abuse like alcohol, tobacco or other drug abuse and those who were on some form of medical or radiation therapy or surgical intervention within past 3 months of admission.

METHODOLOGY: Patients of alleged Super vasmol 33 hair dye ingestion were taken up for the study after the exclusion criteria were ruled out. Informed consent was obtained from every patient or patient's relatives. Clinical history, complaints, physical examination findings, investigations, treatment modalities followed, clinical progress and outcome were all recorded on the prepared proforma.

Patients with symptoms of generalized body pains with /without generalized weakness with S.CPK levels more than 1000 IU/L (more than 5 times the upper limit of normal) were diagnosed to have rhabdomyolysis.

RIFLE criteria were followed to classify patients as to those who have developed AKI.

Statistical analysis was done with the help of SPSS 17 version software.

RESULTS: The results of this study carried out during June 2012 to June 2013 are depicted as follows:

Total no of cases of super vasmol 33 hair dye ingestion were 50.The relative frequency of suicidal poisoning by Super vasmol 33 ingestion was more in females (72%) than in males (28%). It was further observed that the tendency to commit suicide was more in the age group 21-30 years in both males and females. Nearly 42.85% of male patients & 47.22% of female patients were age group 21-30 years. Results were shown in Table 1 & 2.

All the patients (100%) had burning pain in mouth, throat and abdomen. Also, angioedema was seen in all patients (100%) though with varying severity. 70% of patients had dysphagia and /or dyspnea as a result of angioedema. Nearly 72% of patients had cola colored urine after ingestion of dye. Almost all of these patients had also developed generalized muscle pains. Generalized myalgia were complained by nearly 70% of the patients, with almost more than half of them (38% of all cases) developed self-limiting weakness (either paraparesis or quadriparesis). The next frequent symptom observed was vomiting (in 54% of cases). Nearly 32% of patients had stridor with/without wheeze.

Only wheeze without stridor was seen in 10 % of patients. 10 patients (20%) have developed pedal edema with/without Anasarca of those in whom oliguria was seen in 7 (i.e., in 70 % of those who developed pedal edema with/without anasarca). Other less frequent symptom observed is peri-oral tingling & numbness in 10% of patients. 22% of patients had developed hypotension, whereas hypertension was seen in 18% of patients. Results were shown in table 3 & 4.

Burning pain in mouth/throat/abdomen, vomiting were the two symptoms which developed within a mean duration of less than 1 hour. The mean duration of development of angioedema, dysphagia and/or dyspnea, wheeze with/without stridor, dark colored urine, seizures and peri-oral tingling was within 24 hours(less than 1 day). Muscle pains and weakness (Taken as symptom of rhabdomyolysis) had a mean duration of presentation between 24- 72 hours (1-3 days). Pedal edema with/without anasarca and hypertension appeared within a mean duration of 6-7 days. Hypotension had developed in a mean duration of 8 hours, whereas hypertension (As a feature of AKI) had developed after a mean duration of 9 days Results shown in table 5 & 6.

Burning pain in mouth/throat/abdomen, vomiting were the two symptoms which developed within a mean duration of less than 1 hour. The mean duration of development of angioedema, dysphagia and/or dyspnea, wheeze with/without stridor, dark colored urine, seizures and perioral tingling was within 24 hours (less than 1 day). Muscle pains and weakness (Taken as symptom of rhabdomyolysis) had a mean duration of presentation between 24-72 hours (1-3 days). Pedal edema with/without anasarca and hypertension appeared within a mean duration of 6-7 days. Hypotension had developed in a mean duration of 8 hours, whereas hypertension (As a feature of AKI) had developed after a mean duration of 9 days. Results were shown in table 7. Respiratory system abnormalities that included stridor/wheeze/crepitation were seen nearly 72% of patients. Central nervous system examination revealed diminished DTRs with no plantar reflex in 26% of patients. This was found in those who had rhabdomyolysis. Pupils were found to be dilated to Light in 10% of patients. Epigastric tenderness was present in 30% of patients of G.I.T examination. Results were shown in table 8.

Considerable raise in mean values were seen in levels of SGPT &S.CPK. Results were shown in table 9. When ABG analysis was done in every patient at presentation, finding suggestive of metabolic acidosis (Either compensated or uncompensated) was seen in 40% of patients. Highest level of protein in urine was seen to be of 3+ grade in 8% of patients, 2+in 20% of patients and 1+ in 28% of patients. 44% of patients had only traces of protein in the urine. On microscopic examination, pigmented cast were found in about 22% of patients. Chest X ray showed features of pulmonary edema in 28% of patient. ECG changes were found in nearly 40% which were reverted back to normal later on. ENMG done in feasible patients who complained of weakness showed features suggestive of myopathy in 10 patients (20%), were shown in table10. About 32% of patients (16 patient) required tracheostomy for a mean duration of 4 days, while nearly 38% among those who underwent tracheostomy also required mechanical ventilation (6 patients). All patients under study had developed angioedema, though of varying severity. The next most frequent complication observed was rhabdomyolysis which was seen in 58% of patients. 32% of total patients have developed AKI, of whom nearly 56% (9 patients) respond to medical therapy and remaining 44% (7 patients) required renal replacement therapy (Hemodialysis). 5 patients (10%) had developed tetanic contraction of fingers that respond to calcium administration suggesting hypocalcemia. Results were shown in table 11. About 70% of patients were discharged from hospital in good general condition after a mean duration of hospital stay of nearly 7

days (Ranging from 4-11 days.). 22% of patient left against medical advice as one or the other complication developed during hospital stay. 4 patients expired due to refractory shock within 24 hours of admission. 62% of patient with hyperkalemia developed AKI in contrast to 22% of patients without hyperkalemia. This difference in relative frequencies was statistically significant. Hence, increased levels of potassium were probably a factor that determined development of AKI. Results were shown in table 12.52% of patients with CPK levels more than 10,000 IU/L developed AKI. AKI was seen in 14% of patients with CPK levels less than 10,000 IU/L. This difference in relative frequencies was statistically significant. Results were shown in tables 13 & 14. 52% of patients with rhabdomyolysis have developed AKI whereas AKI was found in 5% of patients without rhabdomyolysis and S.CPK more than 10,000 IU/L are also probable factors determining development of AKI. AKI was seen in 38% of patients who ingested more than 50ml of dye and was seen in 19% of patients who ingested less than 50ml of dye but this difference was not statistically significant, were shown in table 15.

DISCUSSION: Poisoning by hair dye ingestion was predominant in the Africa & the Middle East countries. One study (Suliman SM et al) mentioning 150 cases in 10 years duration was reported from Khartoum, Sudan, between1983-93⁽³⁾. Another study (Ayoub Filali et al) mentioning 374 cases between 1992- 2002 was reported from morocco.⁽⁴⁾

The present study was done on 50 cases of hair dye poisoning. All the cases in the study consumed the hair dye available in the market with the trade name Supervasmol 33. Though 62 patients presented with alleged cause of Supervasmol 33 poisoning, 12 of them were excluded from the study based on the exclusion criteria for the study.

Our study has a female predominance with 72% of patient being females. The studies of Ayoub Filali et al, Suliman SM et al, and Yadavendra Reddy et al also showed a female preponderance with 77%, 80%, & 65% respectively. ⁽⁵⁾ The mean age of patients was 24.98±8.42 years in our study. Manisha sahay et al had patients with a mean age of 27±5 years in their study (26.9±4.95).⁽⁶⁾ In the study of kallel et al 27.9±16.8 years with mean (Range 18-40 years).⁽⁷⁾ The prevalence was noted to be more in the age group of 15 to 35 years in our study (72.5) with almost 81% of males (13 of 16) & 67% female patients (16 of 24) falling in age group. The study by Ayoub Filali et al also mentioned a pre ponderance in this age group accounting for 70% cases. The study of Yadavendra reddy et al also observed the same.

On ingestion of super vasmol 33, all the patient (100%) had developed burning pain in mouth, throat & abdomen with a mean duration of 23 ± 11 min (with range of 50). Vomiting occurred in 54 %patients with in a mean duration of 25 ± 28 minutes (range 120 minutes). Suliman SM et al found such G.I.T symptoms in 40 % of patients in their study. Such symptoms were also described by Sumeet singla et al & Sharma A et al in their case reports.^(8,9)

Angioedema developed in 100% of the patient with varying severity in our study within a mean duration of 3.42±1.25 hours (range 5 hours). It was described in 60 % of the patients by Ram et al.⁽¹⁰⁾ Kallel et al observed cervico facial edema in 79% of their patients. Manisha sahay et al reported that 20% of the patients had cervico facial edema. In the study of Yadavendra Reddy et al, Angioedema was found in 70% of the patients. They also described that it was earlier sign in such cases. Suliman et al described it in 100% of their patients. Almost all cases reports consistently described this feature.

As a complication of the angioedema, dysphagia observed in 70% of patient within mean duration of 3.72±3. 12hours (Range 12 hours); stridor with or without wheeze was seen in 32 % of patients with in a mean duration of 2.02±2.29 hours (Range 12 hours), only wheeze 10% of patient within mean duration of 0.48±1.54 hours (range 7). Kallel et al described the symptoms of upper airway tract edema in 68% of patients. Yadavendra Reddy et al described laryngeal edema features in 35% of their patients.

Those who presented with severe angioneurotic edema with stridor underwent emergency tracheostomy. Tracheostomy was done in 16 patients of our study (32%) and 6 of them required then mechanical ventilation for respiratory support (12% of all patients). Yadavendra reddy et al described tracheostomy done in 13% of their patients (5%). In the study of manisha et al tracheostomy done in 20% of their patients.10% of their patient's required mechanical ventilation. Kallel et al described 84% of their patient's required respiratory support with orotracheal intubation/tracheostomy with mechanical ventilation. Ram et al reported that 40% of their patients underwent tracheostomy and 20% required mechanical ventilation (6%) sachin sony et al described that 80% of their patients required emergency airway and 70% required ventilation.

Black colored urine was observed in 72% of our study population, with all of these patients developing generalized muscle pains. It was observed within mean duration of 15.46±15.60 hours (Range 52 hours). Ram et al described this features in 100% of their patients. Suliman et al also observed that the first voided urine after ingestion of dye was black in color in all (100% of their patients that later got less intensified). Kallel et al described this feature in 73.3% of their patients. High colored urine was observed in 10% of patients by Manisha sahay et al in those who presented to them at the stage of AKI.

Generalized myalgias were complained by 70% of patients who had dark urine. 38% of patients developed weakness (Almost 50% of those with myalgias). Myalgia was complained after a mean duration of 1.76 ± 1.39 days (Range 5 days). This complaint was observed in 10% of patients by Suliman et al. In the study Ram et al, muscles pain was complained by 70% and weakness by 70% and weakness by 10% of their patients. Yadavendra Reddy et al noted severe myalgia in 35% of their patients.

20% of patient had developed pedal edema with/without anasarca and 14% had oliguria in our study. It took a mean duration of 1.26±2.82 days (range 10 days) for manifestation of pedal oedma /anasarca and oliguria was observed after a mean duration of 1±2.60 days (Range 10 days). Oliguria was observed in 60 % of patients by Sachin sony et al ⁽¹²⁾. 9% of patients developed these features in the study of Yadavendra Reddy et al. Ram et al noted these features in 70 % of patients. Oiguria was seen in 37% of patients in the kallel et al study. The study by Manisha sahay et al constituted all the patients with oliguria & fluid over load.

Hypertension was noted in 18% of patients in our study after a mean duration of 150 ± 54 hours (Range 162 hours). Hypertension was observed in 33.33% of patients by Manisha sahay et al. 20% of patients presented with a shock in our study with a mean duration of 5.86 ± 2.84 hours (range 7 hours). Kallel et al reported shock in 26.3% of their patients. Sachin sony et al reported 30% frequency of shock in their study.

Other less frequent symptom observed in our study is perioral tingling &numbness (10%). Conjuctival discoloration described in all 150 of their study population (100%) by Suliman et al. Encephalopathy & seizures were noted in 30% of patients in the study Manisha sahay et al. Convulsions were also described in case reported by Ravi varma et al.⁽¹³⁾ Trismus with carpopedal spasm with

positive Chvostek's & Trosseau's signs was described in a case reported by Bhargava et al.⁽¹³⁾ Dermatitis and itching was noted in 6.6% of patients by Suliman et al. But no person in our study population had feature of dermatitis.

Clinical examination revealed respiratory abnormalities in 72% of the patients that include stridor, wheeze and or crepitations. Epigastric tenderness observed in 30 % of patients. Pupil were found to be dilated but reacting to light in 10% of patients and this features was found in the patients that presents with respiratory distress.

Muscle tenderness with flaccidity, diminished deep tendon reflex with absent plantar reflex were found in 26% of patients and these findings were all noted in patients with weakness. Sensory abnormalities were not found in an patient.

In our study, blood investigation revealed increased of SGPT in 88%, SGOT in 68%, Blood Urea in 54%, S. Creatinine in 46 %, Total WBC count in 36 % and potassium in 26% of the investigated patients, Hyponatremia noted in 36% of patients & hypocalcemia in 14 % of patients. Almost 68% of investigated patients had increased S.CPK Levels of more than 10 folds i.e more than 1000 IU/L and 18% had levels more than 30,000 IU/L. significant increase in mean values were noted in SGPT& CPK levels of the study population.

ABG analysis revealed metabolic acidosis in 40% of the investigated patients. Kallel et al noted hyperkalemia in 26% of patients, increase CPK levels in 100% patients (with a mean 77.762 IU/L). Metabolic acidosis was reported in 100% of the patients.

In the study of manisha sahey et al, rise in CPK was noted in 20% of patients and raise in SGPT was observed in10 % of patients. Of the 30 AKI patients they studied, a mean blood urea level of 166.46 ± 62 mg/dl, a mean creatinine level of 8.57 ± 1.85 mg/dl & a mean potassium levels of 5.1 ± 0.6 mEq/l was noted.

In our study proteinuria was seen in 56 % of patients. Microscopic examination of urine revealed pigmented casts in 22% of patients.

Though arrhythmias and cardiac motion abnormalities were described in a few case reports, only transient ECG changes that were suggestive of ventricular strain and ischemia were found in 40 % of patients in our study. (14,15, 16, 17,18)

Regarding complications, all 50 patients (100%) in our study has developed varying severity of angioedema. Rhabdomyolysis was noted in 58% of patients. Ram et al noted rhabdomyolysis in 100% of their patients.⁶ It was observed in 35% of patients by Yadevendra Reddy er al. Sachin sony et al observed it in 60% of the patients. Kallel et al noted this feature in 100% of patients. Manisha sahay et al reported this feature in 20% of their patients.

In our study, acute kidney injury (AKI) had developed in 32% of our patients with 18% responding to drugs and 14% of requiring dialysis. In the study by kallel et al, the frequency of AKI was 47.4% with dialysis requirement in 26.3%. Sachin sony et al reported AKI in 80% of their study group with dialysis requirement in 70%. Suliman et al reported that frequency of AKI in their study group was 80% and dialysis requirement was 60% of their patients. In the study of Yadavendra Reddy et al, AKI developed in 15% of patients and dialysis requirement was in 9% of them. Ram et al reported 70% AKI in their study group and dialysis as required by all 70%.

In our study a significant statistical correlation between rhabdomyolysis and AKI, CPK levels of more than 10,000 IU/L & AKI, hyperkalemia & AKI were found. No attempt was made to assess the

statistical correlation between the above feature & mortality, as the mortality rate in those who left the institute was unknown.

Mortality was 8% in our study population. Within the institutes, all the 4 patient who were expired, died due to refractory shock. Ram et al had 10 % mortality in their study. Mortality in the study of yadavendra reddy et al accounted for 10.5%. There was a mortality rate of 60% in the study by sachin soni et al. Mortality of 31.6% was reported by kallel et al. Manisha sahay et al reported 26.6% mortality in their study group.

CONCLUSION: Super vasmol 33 hair dye has emerged as a potential suicidal poison. The clinical profile of Super vasmol 33 hair dye poisoning is marked by respiratory, muscular, hemodynamic and renal syndromes. Severe Angioneurotic edema and acute kidney injury occurrence testifies to the severity of intoxication and predicts morbidity and mortality. There is no specific antidote for Super vasmol 33 hair-dye poisoning. The most important aspects of management is prompt supportive measures.

As the burden of handling Supervasmol 33 hair dye poisoning cases has been recently increasing, primary care physicians, intensive care physicians and nephrologists need to be aware of its clinical manifestation and management.

LIMITATIONS: The present study chiefly lags behind in assessing the efficacy of the followed treatment modalities in preventing complications, assessing the effect of dye on cardiac function, The frequency of intravascular hemolysis which was described by many authors. was not assessed in the present study, Structural defects in patients with AKI & muscle in patients of rhabdomyolysis was not assessed because of unwillingness & unaffordability of patients for kidney & muscle biopsyand also deficient in assessing the statistical correlation between those factors that were thought to influence morbidity.

REFERENCES:

- 1. Sampath Kumar K, Sooraj YS. Hair dye poisoning and the developing world. J Emerg Trauma Shock 2009; 2(2): 129:31.
- 2. Nott HW. Systemic poisoning by Hair dye. The Br Med J 1924; 1: 421-2.
- 3. 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.
- 4. Filali A, Semlali I, Ottaviano V, Soulaymani R, Furnari C, Corradini D. A retrospective study of acute systemic poisoning of paraphenylene diamine (Occidental takawt) in Merocco. Afr J Trad Cam 2006; 3: 142-9.
- 5. Yadavandra Reddy KB, Chandrababu, Venkata Subbaiah. A study of Vasmol poisoning. Abstract of free paper presentation in proceddings of annual conference of association of physicians of India from Jan 29, 2009 to Feb 1, 2009 at greater Noida, India.
- 6. Manisha Sahay, Vani R, Valli S. Hair dye ingestion–An uncommon cause of acute kidney injury. J Assoc physicians India 2009; 57: 35-8.
- 7. Kallel H, Chelly H. Dammak H, Bahloul M, Ksibi H, hamida CB. Clinical manifestation of systemic parapherylene diamine intoxication J Nephrol 2005; 18(3); 308-11.

- 8. Sumeet Singla, Sanjeev M, Lal Ak, Pulin gupta, Agarwal AK. Paraphenylene diamine (PPD) poisoning. J Ind Ac of Clin Med 2005; 6(3): 236-8.
- 9. Sharma A, Mahi S, Sharma N, Suryanarayana BS, Bhalla A, Suri et al. Intravascular hemolysis and acute renal failure following hair dye poisoning. In proceedings of the 7th international congress of Asia Pacific Association of Medical Toxicology at Chandigarh, India.
- 10. Ram R, Swarnalatha G, Prasad N, Dakshina Murthy KV. Paraphenylene diamine ingesion: An uncommon cause of acute renal failure. J post grad Med 2007; 53: 181-2.
- 11. Sachin S, Nagarik AP, Manjunath D, Gopal krishnan A, Anuradha. Systemic toxicity of paraphynylene diamine. Indian J Med Sci 2009; 63(4): 164-6.
- 12. Bargava P, Matthew P. Hair dye poisoning. J Assoc. physicians India, 2007: 55: 871-2
- 13. Ravi Verma, Nidhi tewari, Sushil Jaiswal, Virendra R, Dinesh K Singh, Arun Tewari. Fatal poisoning caused by oral ingestion of a hair dye. The internet journal of emergency and intensive care medicine 2008; 11(1)
- 14. Brahmi n, Kouraichi N, Blel Y, Mourali S, Thabet H, Mechmeche R, Amamou M. Acute myocarditis and myocardial infarction induced by paraphenylene diamine; interest of angio coronary-graphy. Int J Cardiol 2006; 11(3): E93-5.
- 15. Zeggwagh AA, Abouqcal R, Abidi K, Madani N, Zekraoui A, Karkeb O. Left ventricular thrombus and myocarditis induced by PPD poisoning. Ann fr Anesth Reanin 2003; 2: 639-41.
- 16. Lauet B, Niedraic C, Schanhwell M, Pauschinger M, Strauet BE, Schultheiss HP. Cardiac Troponin-T in patients with clinically suspected myocarditis. J Am Cardiol 1997; 30: 1354-9.
- 17. Abidi K, Himdi B, Lamalmi N, Alhamanyz, Abougal R. Myocardial Lysis in a fetus induced by maternal PPD poisoning following an intentional ingestion to induce abortion. Hum Exp Toxivol 2008; 27(5): 435-8.
- 18. Neelima Singh, Jatar OP, Gupta RK, Tailor MR. Myocardial damage in hair dye poisoning An uncommon presentation. J Assoc Physicians India 2008; 56: 463-4.

Age group(years)	No. of patients	Percentage	
<=20	19	38%	
21-30	23	46%	
31& above	8	16%	
Total	50	100%	
Table 1: Age distribution of patients			

Sex	No. of patients	Percentage		
Female	36	72%		
Male	14	28%		
Total	50	100%		
Table 2: Sex distribution of patients				

Sl. No.	Symptoms	No. of patients	Percentage
1	Burning pain	50	100%
2	Vomiting	27	54%
3	Angioedema	50	100%
4	Dysphagia	35	70%
Table 3: Signs & symptoms of presentation among patients			

Symptoms	No. of patients	Percentage	
Dark urine	36	72%	
Muscle pain	35	70%	
Weakness	19	38%	
Stridor	16	32%	
Hypotension	11	22%	
Pedal edema/Anasarca	10	20%	
Hypertension	9	18%	
Oliguria	7	14%	
Wheeze	5	10%	
Perioral tingling &numbness	5	10%	
Table 4: Other symptoms and signs in the patients			

Symptoms	Time of development		
Symptoms	Mean SD		Range
Dark urine(hours)	22.19	13.95	48
Stridor(hours)	6.31	2.52	10
Hypotension(hours)	7.81	3.81	14
Wheeze (hours)	5.20	1.48	4
Perioral tingling &numbness	18.00	7.36	16
Pedal edema/Anasarca(days)	6.90	1.79	6.0
Oliguria(days)	7.14	1.95	6.0
Muscle pain(days)	2.57	0.81	4
Weakness(days)	3.36	0.89	3
Hypertension(days)	8.22	1.78	5
Table 5: Average time of development of various symptoms and signs			

Symptoms	Time of development in hours				
Symptoms	Mean	SD	Range		
Burning pain(min)	23.10	10.97	50		
Vomiting(min)	46.29	22.08	110		
Angioedema	3.42	1.25	5		
Dysphagia	5.31	2.31	11		
Table (. Average time of development of other comptone					

Table 6: Average time of development of other symptoms

Symptoms	Male (%) N = 14	Females (%)=36	Statistical	
Symptoms	Male (70) N = 14	remates (70)=50	significant	
Dark urine	12(85.71%)	24(66.66%)	0.29 NS	
Muscle pain	11(78.50%)	24(66.66%)	0.50 NS	
Weakness	4(28.57%)	15(41.66%)	1.00 NS	
Stridor	6(42.90%)	15(4.70%)	1.00 NS	
Hypotension	2(14.28%)	9(25.00%)	0.70 NS	
Pedal oedema /			0.42 NC	
Anasrca	4(28.50%)	6(16.66%)	0.43 NS	
Hypertension	4(28.57%)	5(13.88%)	0.24 NS	
Oliguria	4(28.57%)	3(8.3%)	0.08 NS	
Wheeze	2(14.28%)	3(8.3%)	0.61 NS	
Burning pain	14(100%)	36(100%)	1.00 NS	
Vomitings	8(57.14%)	19(52.77%)	1.00 NS	
Angioedema	14(100%)	36(100%)	1.00 NS	
Dysphagia	8(57.14%)	27(75%)	0.30 NS	
Perioral tingling &	1(7.14%)	4(11.11%)	1.00 NS	
numbness	1(/.17/0)	τ(11.1170)	1.00 105	
Table 7: Symptoms & Signs in the patients				

Sl. No.	System &findings		No. of subjects	Percentage
1.		Respiratory system		
	a)	Normal	14	28%
	b)	Stridor	16	32%
	(c)	Wheeze	5	10%
	(d)	Crepitation	11	22%
	(e)	Wheeze &crepitation	4	8%
2.	Cardiovascular system			
	a)	Normal	48	96%

	1-)	CDD	1	20/
	b)	CPR	1	2%
	c)	ESM	1	2%
3	Ga	astrointestinal system		
	a)	Normal	35	70%
	b)	Epigastric tenderness	15	30%
4	Central nervous system			
	a)	Normal	32	64%
		Diminished deep		
	b)	tendon reflex with no	13	26%
		plantar reflex		
	c)	Pupil dilation	5	10%
	Table 8: Findings in important body systems			

Sl. No.	Investigations	Mean	SD	Range	
1	Blood urea	58.28	31.49	156	
2	Serum creatinine	1.91	1.38	5.2	
3	Serum bilirubin	1.03	0.50	2.6	
4	SGOT	91.32	73.21	292	
5	SGPT	98.24	88.63	439	
6	СРК	12047.40	17165.87	72023.93	
7	Sodium	136.4	5.23	23	
8	Potassium	4.65	0.69	3	
9	Calcium	8.75	0.65	3	
	Table 9: Other Investigative findings				

Sl. No.	System & findings		No. of subjects	Percentage	
1.		Chest X ray			
	(a)	Normal	36	72%	
	(b)	Bilateral	14	28%	
	(0)	pulmonary edema	17	2070	
2.	ECG				
	(a)	Normal	30	60%	
	(b)	Pathological	20	40%	
	(D)	changes	20	40 %	
3.		ENMG			
	(a)	Normal	40	80%	
	(b)	Myopathy	10	20%	
	Table 10: Investigative findings in patients				

Sl. No.	Complication	No. of patients	Percentage
1.	Angioedema	50	100%
2.	Rhabdomyolysis	29	58%
3.	AKI responding to medication	9	18%
4.	AKI failure to medication	7	14%
5.	Hypocalcemia	5	10%
Table 11: Complication among patients			

Hyperkalemia	I	Total		
пурегкатенна	Yes	No	Total	
Yes	8(61.5%)	5(38.46%)	13(100.0)	
No	8(21.6%)	29(78.37%)	37(100.0)	
Total	16(32%)	34(68%)	50(100.0)	
Table 12: Hyperkalemia by AKI				

Raised CPK (>10,000)	A	Total		
Raiseu CFR (>10,000)	Yes	No	IUtai	
Yes	12(52.17%)	11(47.8%)	23	
No	4(14.18%)	23(85.18%)	27	
Total	16(32%)	34(65%)	50	
Table 13: Raised CPK (>10,000) by AKI				

 χ^2 =7.97; p=0.005; Significant

Rhabdomyolysis	A	Total			
Kilabuoliiyoiysis	Yes	No	Total		
Yes	15(51.72%)	14(48.2%)	29(100%)		
No	1(4.76%)	20(95.23%)	21(100%)		
Total	16(32%)	34(68%)	50(100%)		
Table 14: Rhabdomyolysis by AKI					

 χ^2 =12.34; P =0.0004; Very High Significant

Amount of dye (>50 ml)	A	Total		
Amount of uye (>30 m)	Yes	No	TULAI	
Yes	13(38.23)	21(61.76)	34	
No	3(18.75)	13(81.25)	16	
Total	16	34	50	
Table 15: Amount of dye (>50ml) by AKI				

 χ^2 = 1.90; P value =0.168; Not Significant.

Sl. No.		Kallel et al study	Suliman et al study	Manisha et al study (ARF group)	Yadavendra Reddy et al study	Ram et al study	Our Study
1	Study Population	19	150	30	225	10	50
2	Age (Mean ± S.D) (in years)	27.9±16.8	40.0±6.89	26.9±4.95	26.8±7.34	23.2±7.6	24.98±8.42
3	Female: Male	11:8	4:1	1:4	65:35	4:1	2.5:1
Sy	mptoms & Signs						
4	G.I.T Symptoms	60%	40%	-	-	-	100%
5	Cervicofacial edema	79%	100%	20%	70%	60%	100%
6	Stridor ± Wheeze	68%	-	-	35%	-	42%
7	Dark urine	73.3%	100%	10%	80%	100%	72%
8	Myalgian± weakness	60%	10%	-	35%	70%	70%
9	Oliguria	37%	60%	100%	9%	70%	14%
10	Haemodynamic shock	26.3%	30%	-	-	-	22%
	Investigation						
11	Hyperkalemia	26%	-	-	-	-	26%
12	High CPK levels	100%	-	20%	-	-	46%
13	High SGPT levels	-	-	10%	-	-	88%
14	Metabolic Acidosis	100%	-	80%	-	-	40%
(Complications						
15	Angioedema	79%	100%	20%	70%	60%	100%
16	Rhadomyolysis	100%	68%	20%	35%	100%	58%
17	AKI	47.4%	80%	100%	15%	70%	32%
	Outcome						
18	Outcome Mortality	31.6%	-	26.6%	10.5%	10%	8%
		Table 16:	Comparisio	n with other	Studies		

AUTHORS:

- 1. Sudheer Babu Devineni
- 2. Nannam Harshavardhan
- 3. Nalamala Baskara Rao
- 4. Vuyyala Chandana

PARTICULARS OF CONTRIBUTORS:

- 1. Associate Professor, I/C Professor, (ID Ward), Department of General Medicine, Government Fever Hospital, Guntur Medical College, Guntur.
- 2. Assistant Professor (ID Ward), Department of General Medicine, Government Fever Hospital, Guntur Medical College, Guntur.

FINANCIAL OR OTHER COMPETING INTERESTS: None

- 3. Assistant Professor, Department of Pulmonary Medicine, GMC, Guntur, Andhra Pradesh.
- 4. Senior Resident, Department of General Medicine, GMC, Guntur, Andhra Pradesh.

NAME ADDRESS EMAIL ID OF THE CORRESPONDING AUTHOR:

Dr. Sudheer Babu Devineni, Associate Professor I/C Professor of ID Ward, Department of General Medicine, Guntur Medical College/Government General Hospital, Guntur. E-mail: sudheerbabudevineni@gmail.com

> Date of Submission: 25/05/2015. Date of Peer Review: 26/05/2015. Date of Acceptance: 06/06/2015. Date of Publishing: 13/06/2015.