A PROSPECTIVE STUDY ON THE CLINICAL PROFILE AND OUTCOME IN HAIR DYE (SUPER VASMOL) POISONING

Dissertation submitted to

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY

CHENNAI- TAMILNADU

In partial fulfillment for the Degree of

DOCTOR OF MEDICINE

BRANCH I –M.D.,(General Medicine)

APRIL-2015

DEPARTMENT OF MEDICINE

TIRUNELVELI MEDICAL COLLEGE

TIRUNELVELI- 627011

TAMILNADU

CERTIFICATE

This is to certify that the Dissertation entitled "**A PROSPECTIVE STUDY ON THE CLINICAL PROFILE AND OUTCOME IN HAIR DYE (SUPERVASMOL) POISONING**" is a bonafide original work of **Dr P.N. VIJAY AANAND SIDDHARTH,** in partial fulfillment of the requirement for M.D., BRANCH I General Medicine Examination of the The Tamilnadu Dr.M.G.R. Medical university, Chennai to be held in April 2015. The bonafide work is carried out by him under my guidance and supervision. This dissertation partially or fully has not been submitted for any other degree or diploma of this university or other.

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PROTOCOL TITLE: STUDY ON THE CLINICAL PROFILE AND OUTCOME IN SUPERVASMOL POISONING.

NAME OF PRINCIPAL INVESTIGATOR: Dr. P.N.Vijay Anand Siddarth, MBBS.,
DESIGNATION OF PRINCIPAL INVESTIGATOR: Post Graduate in MD General Medicine
DEPARTMENT & INSTITUTION: Department of General Medicine, Tirunelveli Medical College

Dear Dr. P.N.Vijay Anand Siddarth, *The Tirunelveli Medical College Institutional Ethics Committee (TIREC) reviewed and discussed your application during the IEC meeting held on 14.05.14.*

THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED

1.	TIREC Application Form	☐
2.	Study Protocol	☐
3.	Department Research Committee Approval	☐
4.	Patient Information Document and Consent Form in English and Vernacular Language	☐
5.	Investigator's Brochure	☐
6.	Proposed Methods for Patient Accrual Proposed	☐
7.	Curriculum Vitae of the Principal Investigator	☐
8.	Insurance /Compensation Policy	☐
9.	Investigator's Agreement with Sponsor	☐
10.	Investigator's Undertaking	☐
11.	DCGI/DGFT approval	☐
12.	Clinical Trial Agreement (CTA)	☐
13.	Memorandum of Understanding (MOU)/Material Transfer Agreement (MTA)	☐
14.	Clinical Trials Registry-India (CTRI) Registration	☐

THE PROTOCOL IS APPROVED IN ITS PRESENTED FORM ON THE FOLLOWING CONDITIONS

1. The approval is valid for a period of 2 year/s or duration of project whichever is later
2. The date of commencement of study should be informed
3. A written request should be submitted 3weeks before for renewal / extension of the validity
4. An annual status report should be submitted.
5. The TIREC will monitor the study
6. At the time of PI's retirement/leaving the institute, the study responsibility should be transferred to a person cleared by HOD
7. The PI should report to TIREC within 7 days of the occurrence of the SAE. If the SAE is Death, the Bioethics Cell should receive the SAE reporting form within 24 hours of the occurrence.
8. In the events of any protocol amendments, TIREC must be informed and the amendments should be highlighted in clear terms as follows:
   a. The exact alteration/amendment should be specified and indicated where the amendment occurred in the original project. (Page no. Clause no. etc.)
   b. The PI must comment how proposed amendment will affect the ongoing trial. Alteration in the budgetary status, staff requirement should be clearly indicated and the revised budget form should be submitted.
   c. If the amendments require a change in the consent form, the copy of revised Consent Form should be submitted to Ethics Committee for approval. If the amendment demands a re-look at the toxicity or side effects to patients, the same should be documented.
   d. If there are any amendments in the trial design, these must be incorporated in the protocol, and other study documents. These revised documents should be submitted for approval of the IEC, only then can they be implemented.
   e. Approval for amendment changes must be obtained prior to implementation of changes.
   f. The amendment is unlikely to be approved by the IEC unless all the above information is provided.
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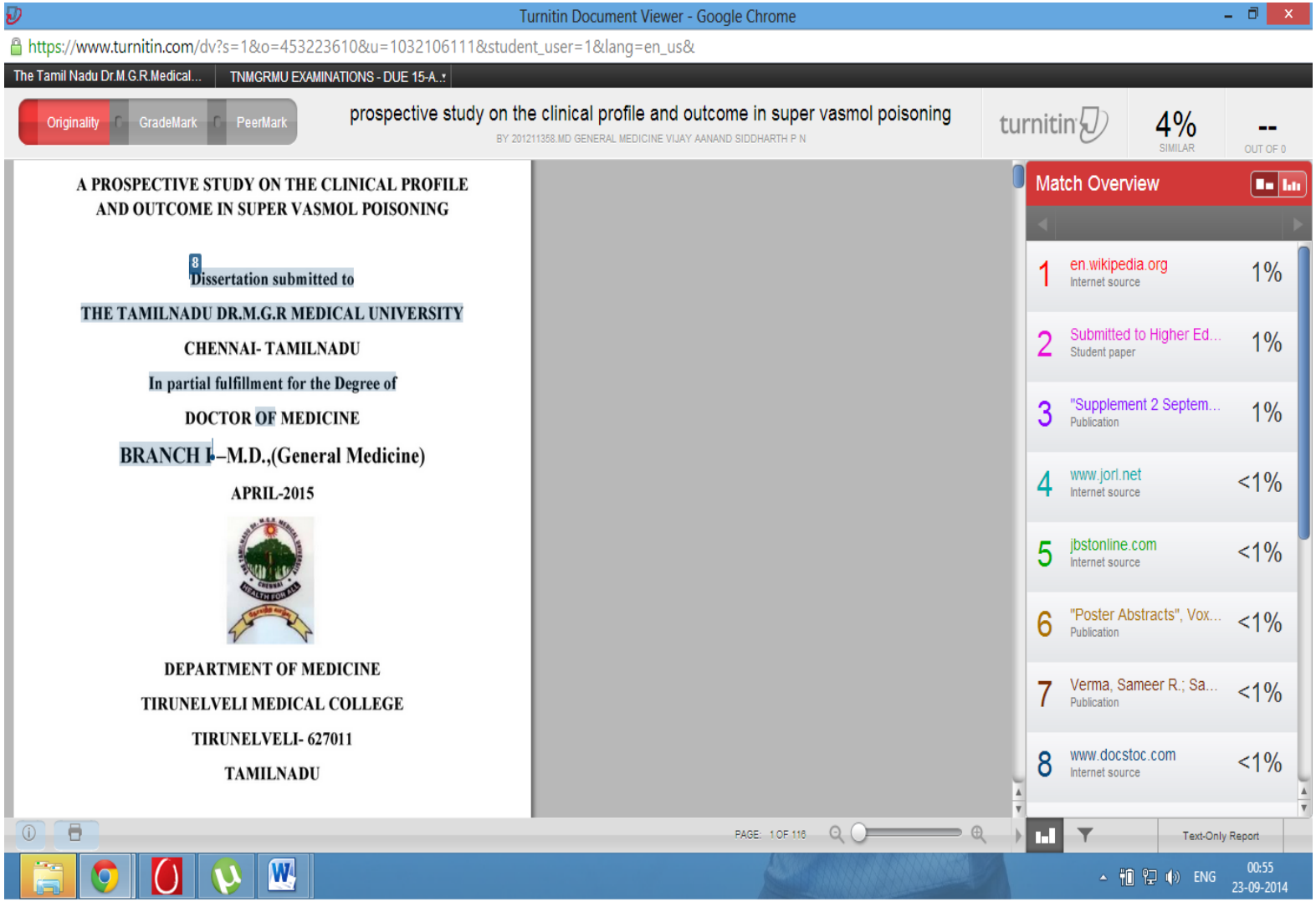
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DECLARATION

I**, Dr P.N. VIJAY AANAND SIDDHARTH**, solemnly declare that, I carried out this work on **"A PROSPECTIVE STUDY ON THE CLINICAL PROFILE AND OUTCOME IN HAIR DYE (SUPER VASMOL) POISONING"** at the department of General Medicine, Tirunelveli Medical College Hospital during the period of April 2014 to September 2014. I also declare that this bonafide work or a part of this work was not submitted by me or any others for any award, degree, diploma to any university, found either in India or abroad.

This is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfillment of the rules and regulations for the MD Degree Branch I General Medicine Examination, to be held on April 2015.

Place: Tirunelveli

DR. P.N. VIJAY AANAND SIDDHARTH

POST GRADUATE,

M.D. GENERAL MEDICINE

TIRUNELVELI MEDICAL COLLEGE

ACKNOWLEDGEMENT

THANKS TO THE IMMORTAL POWER WHO HAS BESTOWED UTMOST KINDNESS ON ME

First of all I like to express my sincere gratitude and indebtedness for our beloved **Prof**. **DR. RAJAGOPALA MARTHANDAM MD,** Chief, 6th Medical Unit, Department of Medicine, Tirunelveli Medical College, who stayed as a constant inspiration for my study and for his expert guidance and support throughout my course.

It is of immense gratitude that I like to thank our beloved **PROF.Dr.M.R.VAIRAMUTHURAJU MD**, Professor and Head, Department Of General Medicine, Tirunelveli Medical College for his kind advice and support.

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I am thankful to all my senior Assistant Professors **DR.S.MADHAVAN M.D, DR.J.BHARATH MD, DR.THOMAS EDWIN RAJ MD & DR. MONNA MOHAMMED JAFFER MD** for their valuable suggestions and help given for my study.

No words of gratitude will be enough to thank my parents for their never ending unconditional support and encouragement at each step in my way.

I sincerely thank all the patients who cooperated with me for participating in the study.

Last but not the least, on the recollection of so many and great favours and blessings, I now, with a high sense of gratitude, presume to offer up my sincere thanks to the God Almighty, the Creator and Preserver.

CONTENTS

A STUDY ON CLINICAL PROFILE AND OUTCOME IN HAIR DYE (SUPER VASMOL) POISONING

ABSTRACT:

BACKGROUND:

Hair dye poisoning with super vasmol is an emerging problem in the developing parts of the world mainly because of the easy availability of the substance. The active ingredients in the dye which has toxic effects are para-phenylene-diamine, resorcinol, propylene glycol and EDTA sodium. Para-phenylene-diamine is responsible for most of the clinical features of the patients presenting with hair dye poisoning.

AIM OF THE STUDY:

To study the clinical profile, biochemical profile, complications, management and outcome in hair dye (super vasmol) poisoning.

MATERIALS AND METHODS:

A prospective study was done on 53 patients over a period of 6 months with patients getting admitted with super vasmol poisoning. Patients with mixed poisoning and hair dye poisoning other than super vasmol poison are excluded from the study.

RESULTS:

We conducted a prospective study on 53 patients who were admitted in the intensive medical care unit of our hospital. Female predominance was seen. The commonest presentation was dyspnoea with cervicofacial edema followed by myalgia and muscle tenderness. The presence of rhabdomyolysis was evident from the muscle pain, muscle tenderness and elevated total CPK levels. 21 patients developed complications like respiratory distress and acute kidney injury. None of the individuals in the study population developed hepatitis and myocarditis. 13.2% of the patients required hemodialysis, 5.7% needed tracheostomy and 11.3% were subjected to both hemodialysis and tracheostomy. The mortality rate in the study population was 11.3%. patients were treated with supportive treatment mainly intra venous steroids, anti-histaminics and supplemental oxygen. Patients who had

evidence of rhabdomyolysis were treated with forced alkaline diuresis. Totally 6 patients expired and the cause of death being respiratory distress and acute kidney injury.

CONCLUSION:

The initial presentation, volume consumed and biochemical profile correlates directly with severity of the clinical features and the complications. The main complications were acute respiratory distress and acute kidney injury and these also being the common cause of mortality. The treatment is mainly supportive and treatment of the complications. The long term complications of these patients are not studied as the duration of the study was short and it requires further studies regarding the long term prognosis of these patients.

KEY WORDS: supervasmol, poisoning, para-phenylene-diamine, acute respiratory distress, acute kidney injury, rhabdomyolysis, forced alkaline diuresis.

INTRODUCTION:

Poison is a substance that produces toxic effects on coming into contact with the human body. It could either be oral, dermal and inhalational route. The history of poison and poisoning dates back to the early 4500 BC. Poisons have been used for many purposes mainly as weapons and anti-venoms. Initially poisons were used as a tool for hunting to ensure the death of the prey. Later poisons were used for assassination mainly in the Roman Empire. In the medievel Europe the use of poison for homicidal purposes increased and this was mainly due to the increased and easy availability of poisons.

Poisoning which was initially used as weapons later on was used for homicidal purposes and now the incidence of suicidal poisoning is increasing. According to the various studies conducted throughout India suicidal poisoning is estimated to be the commonest followed by accidental poisoning and a very low reported incidence of homicidal poisoning. The list of substances which are used as poisons is now changing with the list extending over the past few decades.

SUICIDAL POISONING:

EPIDEMIOLOGY:

INCIDENCE:

Suicidal poisoning is one of the leading causes of mortality in many parts of the world and similar trends have been observed in India as well. It has been studied that it is the fourth most common cause of mortality in rural India. Poison accounts for 2.1% of the total admissions in the Intensive care units and about 1.2% of the total number of deaths in this population. There has been a remarkable change in the incidence and type of poisoning in the past 3 to 4 decades. A variety of new compounds have started to come under the list of potentially poisonous substances. It is important to realize that poisoning and death due to the same are largely preventable and many of the cases die within 6 to 8 hours of getting exposed to the poison and a large proportion of these people die even before reaching the hospital.

Incidence of poisoning in India remains uncertain but it has been estimated that 1 to 1.5 million cases are admitted in the hospitals due to poisoning every year and out of which about 50,000 cases succumb to it. In India suicidal poisonings tops the list followed by accidental poisoning followed by homicidal poisoning which contributes much less to the problem. This trend is same as the rest of the world. But the incidence of homicidal poisoning is far more common in India than in the western world.

It has been observed that the poisoning has age, sex and socio-economic association and this varies in different parts of the world.

There has been an increasing trend in poisoning with increase in age up to the third decade after which it starts to decline, with the peak incidence being in the age group of 20 to 30 years. The incidence is least after 60 years of age Literacy has not been found to have a negative role in suicides due to poisoning.

Suicidal poisoning is has typical sex distribution where younger females tend to consume poison more than males of the same age group. But the age specific mortality due to poisoning is more in the males when compared to the females.

Socio-economic status has a great influence over the incidence of suicidal poisoning. It has been studied that incidence of suicidal poisoning is more in lower and lower middle socio-economic class people than the upper socio-economic class. The incidence in the upper middle class of the population has varied data in India.

Similar to the age, sex and socio-economic distribution, residing location of the people acts as an important determinant. Rural population has more reported poisoning due to suicidal intents when compared to the urban population. Similarly the suicidal poisoning in sub-urban areas is

comparatively less than that in rural population but more than urban incidence.

Literacy as per the previous studies conducted is not found to have a negative role in this problem worldwide and the scenario is the same in India as well.

TYPES OF POISONS:

The common substances used for suicidal poisoning in India are the following,

1. Organo phosphorus compounds
2. Organo chloride compounds
3. Rat killer poisons
4. Ant killer poisons
5. Plant poisons
6. herbicides
7. Drugs
8. Corrosives
9. Unknown
10. Miscellaneous

ORGANOPHOSPHORUS COMPOUNDS:

Out of the above list of poisons organophosphorus compounds are the most commonly used for suicidal poisoning. This is more prevalent among the

rural population than the urban sector. The subset of people who consume these compounds are housewives, farmers and laborers. The male to female ratio varies in different studies and it is more common in the younger age group less than 30 years of age and the incidence decreases with ageand is very rare beyond 60 years of age.

RAT KILLER POISONS:

These compounds which are originally used as rodenticides, is being used with the suicidal intent. The commonly used substances are bromolidene, zinc phosphide and aluminium phosphide. Bromolidene poisoning presents with bleeding manifestations while phosphides have a high mortality rate of more than 95% with death occurring due to cardiac arrhythmias, renal failure and severe dyselectrolytemias.

ANT KILLER POISONS:

Commonly used compound is dimethyl-parathion.

HERBICIDES:

The incidence poisoning due to these compounds are also increasing with the common compounds being glyphoside and paraquet.

DRUGS:

This is becoming common in patients with psychiatric disorders like depression and bipolar affective disorders who are prescribed with these drugs for therapeutic purposes. Benzodiazipenes and paracetamol poisoning

tops the list and the commonly used benzodiazipenes being alprazolam and nitrazepam. Other commonly used drugs for suicidal poisoning includes opioids, antipsychotics, antidepressants, NSAIDs.

PLANT POISONS:

Oleander seed poisoning is common in rural India followed by Oduvanthalai leaves poisoning.

MISCELLANEOUS:

The less commonly used poisoning substances include corrosives, hydrocarbon substances (petrol, diesel and brake oil), kerosene,hair dye.

HAIRDYE POISONING:

Hair dye poisoning is becoming an emerging problem in the developing part of the world mainly the Indian sub-continent (including Pakistan, Bangladesh, Sri lanka, Bhutan and Mayanmar), middle east countries and many parts of central and east Africa. The easy availability and cheaper prices have been one of the reasons for this becoming an important mode of suicidal poisoning.

There are different types of hair dyes and they can be classified as

1. Permanent or oxidative dyes
2. Semi-permanent or temporary or direct dyes
3. Metal salts
4. Natural salts

Some of the hair dyes have mainly herbal products as their active ingredients while certain other type of dyes contain predominantly synthetic chemicals mixed with herbal compounds. Most of the commercially available oxidative dyes consist of two components that are mixed prior to use and generate the dye on the hair by oxidative chemical reaction. The mixture of these chemical ingredients increases the effectiveness of the dyeing process and decreases the time needed for the same. One of the most commonly used hairdye is SUPERVASOMAL 33.

AIM OF THE STUDY

TO STUDY

1. The clinical profile
2. Biochemical profile
3. Management
4. Complications
5. Outcome

In the patients admitted with super vasmol poison in our hospital

REVIEW OF LITERATURE

SUPERVASMOL:

Supervasmol is being available in the shops easily and is cheaper, which is one of the reasons for the increasing number of poisoning admissions due to this compound in India. Now poisoning with this compound has started replacing the other types of suicidal attempts like poisoning with other compounds like herbicides, insecticides and pesticides, burns, hanging, etc.

Supervasmol is a commonly used emulsion based hair dye. The dye contains many ingredients, of which main component is para-phenylene-diamine which is responsible for life-threatening events which occur following the consumption of the dye. The other hair dyes which contain Para-phenylene-diamine come under the brand names of Godrej, Keshkala, colourmate etc.

Apart from para-phenylene-diamine there are certain other ingredients of the hair dye which includes include resorcinol, propylene glycol, EDTA sodium which also has other toxic effects on the system. .

Supervasmol is available in bottles as a solution. The net volume of the dye in the bottle is either 50ml or 100ml. 100ml of supervasmol contains 12 grams of para-phenylene-diamine. The minimal dose of para-phenylene-diamine that has to be consumed to produce systemic features of

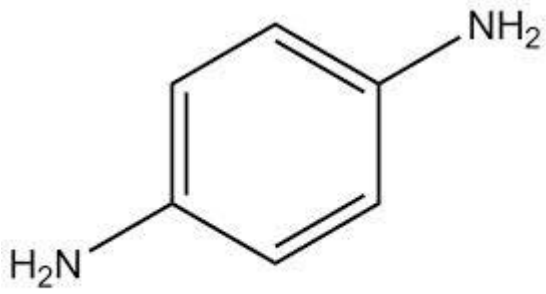
poisoning is approximately 3 grams. The dose that is required to produce fatal toxicity after oral ingestion is 7grams or more. This indicates that the consumption of more than 50ml of the hair dye is sufficient to produce significant local effects mainly edema of the face, neck, pharynx, larynx and the vocal cords thereby leading on to respiratory distress mainly due to the obstruction of the airway and systemic toxicity mainly rhabdomyolysis causing deposition and clogging of the proximal renal tubules with myoglobin. This ultimately causes acute kidney injury due acute tubular necrosis.

CONTENTS OF SUPERVASMOL HAIR DYE:

1. Para-phenylene-diamine
2. Propylene glycol
3. EDTA sodium
4. Resorcinol
5. Liquid paraffin
6. Sodium lauryl sulfate
7. Preservatives
8. Water

PARA-PHENYLENE-DIAMINE:

Para-Phenylene-diamine ($C_6H_4(NH_2)_2$) is an organic compound. The para-phenylene-diamine is not available naturally and it is synthesized from a substance called paranitroaniline which is also used as dye. This aniline derivative is a white crystalline substance. On exposure to air they get oxidized and turn brown to black colour. It is mainly used as an active ingredient in manufacturing engineering polymers and composites. It is also used as an active ingredient in hair dyes.



p-phenylenediamine

Fig.1

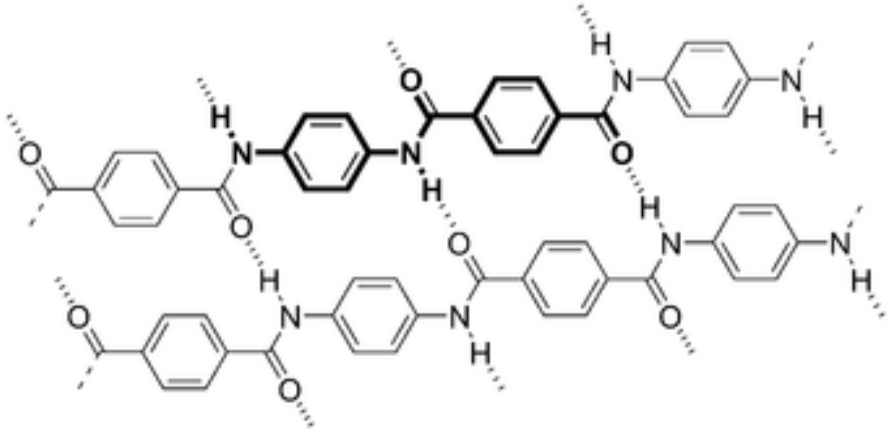


fig.2

Para-phenylene-diamine can be produced by three methods. Most common method involves the treatment of 4-nitrochlorobenzene with ammonia which yields an intermediate compound known as 4-nitroaniline. The latter compound is then subjected to hydrogenation.

$$ClC_6H_4NO_2 + 2\ NH_3 \rightarrow H_2NC_6H_4NO_2 + NH_4Cl$$

$$H_2NC_6H_4NO_2 + 3\ H_2 \rightarrow H_2NC_6H_4NH_2 + 2\ H_2$$

The other method is called as the DuPont method where aniline is converted to diphenyltriazine, which is then subjected to acid catalysis to get 4-aminoazobenzene. Hydrogenation of this compound results in the formation of para-phenylene-diamine.

USES OF PARA-PHENYLENE-DIAMINE:

Para-phenylene-diamine has its use in the following areas, namely

DYEING:

This compound is more commonly used in textile dyes. In recent times the use of this compound is starting to get replaced by its close aniline analogues and derivatives including 2,5-diamino-hydroxyethylbenzene and 2,5-diamino-toluene. Other popular derivatives include indoanilines,

indophenols and tetraaminopyrimidine. Derivatives of diaminopyrazole give red and violet colors.

HAIR DYES:

In the hair dye usually this para-phenylene-diamine is added to henna to give the black color. If henna is used alone it takes about 6 hours to a maximum of 12 hours to get d desired color of the hair. But if it is mixed with para-phenylene-diamine the time taken for this coloring is reduced to an hour or two hours. The color of the hair also depends upon the concentration of para-phenylene-diamine present in the formulation. The concentration of para-phenylene-diamine varies from a minimum of 0.20% to a maximum of 3.75% in depending upon the type of preparations of the hair dye. The concentration present is directly proportional to the shade that develops after application ranging from golden blonde to black color.

RUBBER ANTIOXIDANT:

PPD is easily oxidized, and for this reason derivatives of PPD are used as anti ozonants in production of rubber Products. The substituents, mainly naphthyl and isopropyl affect the effectiveness of their antioxidant roles as well as their properties as skin irritants.

OTHER USES:

A substituted form of PPD is also used as a developing agent photographic film development process which reacts with the silver grains in the film and creating the colored dyes that form the image.

PPD is also used for temporary tattoos as a substitute for henna . Its usage can lead to severe contact dermatitis

HENNA:

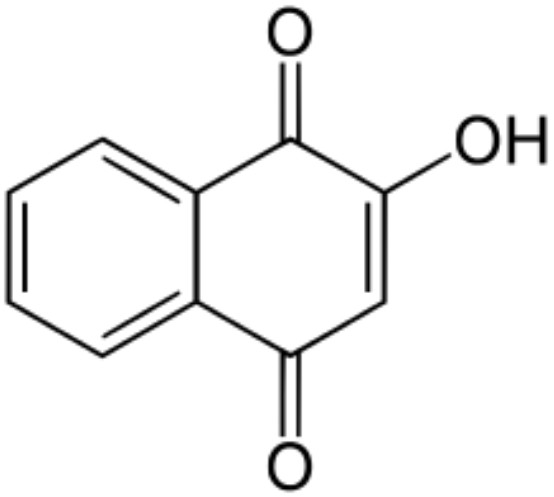


Fig.3

Henna also known as hina or the Egyptian privet called under the botanical name *Lawsonia inermis* is a flowering plant which has been used from ancient times for dyeing of hair, skin and finger nails and also for the purpose of temporary tattooing. The practice of using henna for cosmetic

purposes dates back to 15th century B.C. having its origin in ancient Egypt. The ancient Egyptians used this for the purpose of dyeing hair

. The color produced by henna on the hair depends upon the natural color of the hair. The henna leaves as such cannot produce the change in the color if they are unbroken which means it has be crushed to liberate certain active component which is found to be lawsone. The crushed leaves are then subjected to grinding and is made into a powder form. They are then applied over the skin and on doing so the active compound binds to the skin thereby giving the change in the color. The color normally produced is a reddish brown stain. And the henna has to be applied over the concerned part of the body for at least 6 hours for the stain to be evident and the intensity of stain increases as the contact time is more. As a result to reduce the time needed to stain the hair or the skin for the purpose of temporary tattooing henna in the commercial preparation are mixed with para-phenylene-diamine in varying concentrations up to 5% is being used. The para-phenylene-diamine if present in the dye reduces the time taken for staining to less than 1 hour and also even darker staining. Using henna with more than 5% para-phenylene-diamine is associated with local toxic reactions of the skin.

RESORCINAL:

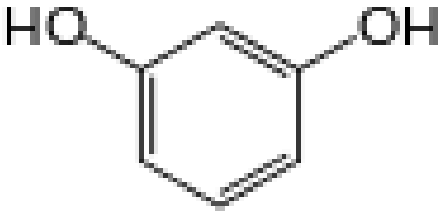


Fig.4

It is the 1,3-isomer of benzenediol and has the chemical formula $C_6H_4(OH)_2$. it is one of the commonly occurring natural phenols. It occurs in the form of colorless crystalline needle. It is easily soluble in water, alcohol and ether.

Resorcinol is a chemical which has corrosive properties and is being used in photography and cosmetic industry. In the cosmetic industry it is mainly used in as a component of hair dyes. It is also has medicinal properties and it has been used in the pharmaceutical industry for in topical preparations for certain skin diseases psoriasis, hidradenitis suppurativa, and sub-acute eczema. Resorcinol is a moderately toxic compound and causes methemoglobinuria and renal failure as a result of acute kidney injury. On chronic dermal exposure to higher concentrations of resorcinol individuals can develop allergic dermatitis.

PROPYLENE GLYCOL:

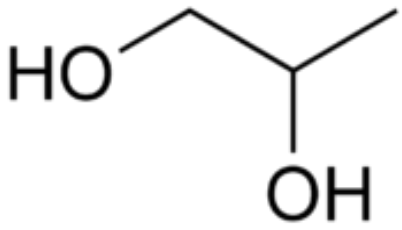


Fig.4a

This compound is used as a solvent in the hair dyes along with the above mentioned ingredients.

Of all these Para-phenylene-diamine, Propylene glycol, EDTA sodium and Resorcinol are found to have systemic toxicity of which Para-phenylene-diamine and resorcinal has the most life-threatening effects. EDTA sodium causes hypocalcemia as it chelates calcium. Resorcinol has corrosive properties and has been found to produce methhemoglobinuria subsequently renal failure due to acute kidney injury by occlusion of the renal tubules by the pigment.

Para-phenylene-diamine when taken in significant doses causes local effects as it causes severe irritation of the mucosal surfaces it comes in contact with while being ingested. On systemic absorption this Para-phenylene-diamine binds to the sarcoplasmic reticulum and causes sustained calcium release. This causes a sustained depolarization of the muscle fibers

causing continuous contraction of these fibers finally resulting in rhabdomyolysis. The myoglobin which is released from the muscle fibers are filtered in the glomeruli and cause occlusion of the renal tubules resulting in acute kidney injury.

TOXIC EFFECTS:

The toxic effects occur after oral ingestion of the dye and are due to the components present in the dye mainly para-phenylene-diamine, resorcinol, polyethylene glycol, EDTA sodium. Other components like the preservatives and emulsifiers have not found to have significant toxic profile.

PARA-PHENYLENE-DIAMNE:

Para-phenylene-diamine has as it is being used for dyeing purposes in hair dyes as well as for temporary tattooing it has toxic effects both when ingested orally as well as when applied over the skin. The hair dyes usually contain a PPD concentrations varying from 0.20% to 3.75% and at this concentrations PPD produces moderate to severe toxic effects when taken orally and it also depends on the amount of the hair dye consumed. Dermal toxicity is usually mild and occurs only when the concentration exceeds 5%.

In hair dyes PPD is usually mixed with hydrogen peroxide in the preparation of hair dyes which yields an intermediate called the Bandrowski's base. This compound is highly toxic and has allergic as well as mutagenic properties.

For skin preparations the absorption is more when it is used alone. When mixed with hydrogen peroxide in appropriate dilutions, the absorption of para-phenylene-diamine decreases to negligible levels of <0.01%. Consumption of 7 grams or more of para-phenylene-diamine is associated with very severe toxicity leading to fatalities. The effects of the dye can be classified into early manifestation and late (or delayed) manifestations and the arbitrary time interval being taken as 6 hours from the time of consumption of the dye as the time taken for systemic toxicity to occur is 6 hours.

EARLY MANIFESTATIONS:

The dye when ingested orally produces clinical features of severe allergic reaction and depends upon the quantity of hair dye consumed. This early phase occurs within 4-6 hours of ingesting the dye. The early manifestation include,

1. Angioneurotic edema
2. Fulminant myocarditis

ANGIONEUROTIC EDEMA:

This is the earliest manifestation following ingestion of the dye. It occurs as early as 1 to 2 hours from exposure. The main symptoms are dyspnoea and dysphagia. The signs include edema involving the face, tongue, throat up to the larynx. This causes severe respiratory distress. This is the most common early cause of death inupervasmol poisoning.

FULMINANT MYOCARDITIS:

This is the second most common early manifestation of para-phenylene-diamine poisoning occurring 2 to 4 hours after ingestion of the dye. Fatal arrthymias can occur including ventricular tachcardia and ventricular fibrillation resulting in sudden cardiac death.

LATE MANIFESTATIONS:

This occurs 12 hours after the ingestion of te dye and may occurs upto 1 week from the time of consumption. These includ

1. Renal toxicity
2. Hepatic toxicity
3. Cardiotoxicity
4. Skin toxicity

RENAL TOXICITY:

This is considered as the most fatal and common late manifestation of PPD poisoning. PPD causes acute tubular necrosis leading on to acute

kidney injury. The principle mechanism which PPD leads on to acute kidney injury by causing rhabdomyolysis is as follows:

Para-Phenylene-Diamine attaches itself to the sarcoplasmic reticulum of the muscle thereby causing increased calcium release from them. So the intracellular calcium levels increase which causes sustained contraction of the muscle leading on to rhabdomyolysis. Once the muscle fibres goes for lysis the myoglobin which is present inside the muscle is released in to the blood. Myoglobin is a small molecule and has a molecular weight of 17kD. This molecule then gets deposited in the renal tubules leading on to acute tubular necrosis and ultimately acute kidney injury.

The main features include presence of muscle (mainly proximal) tenderness, cola colored urine, oligura or anuria. Presence of oligura or anuria indicates the renal tubular damage thereby acute kidney injury.

This is the most common delayed cause of death in supervasmol poisoning.

HEPATOTOXICITY:

This is a rare manifestation of PPD poisoning. The main pathology is hepatic necrosis presenting as elevated bilirubin levels and serum transaminases.

CARDIOTOXICITY:

This is less recognized manifestation of PPD poisoning.The main pathology being myocarditis. The risk of myocarditis increases when the quantity of consumption of PPD is more than 10 grams. The clinical presentation being in the form of,

1. Dyspnoea
2. Chestpain
3. Palpitations
4. Presyncope
5. Syncopal attacks
6. Non-specific ST-T changes

It becomes clinically difficult to differentiate the dyspnoea of cardiac origin from respiratory and renal cause as all the complications can occur in the same patient who has consumed more than 10 grams of PPD. Patients may have varying ECG changes from sinus tachycardia, non- specific ST-T changes to any type of arrhythmias.

ECG CHANGES:

1. Sinus tachycardia
2. Premature complexes (atrial and ventricular)
3. Intra-ventricular conduction defects
4. Bundle branch blocks

5. ST segment elevation and T wave inversion

6. Tall T waves

7. Ventricular tachycardia and ventricular fibrillation

Out of these the commonest finding being sinus tachycardia and the most fatal being ventricular tachycardia and ventricular fibrillation causing sudden cardiac death.

Supportive laboratory evidence of myocardial damage include elevated cardiac troponins and creatinine phosphokinase of myocardial origin. Echocardiography shows depressed left ventricular function.

SKIN TOXICITY:

Dermal toxicity occurs in individuals who are sensitive to para-phenylene-diamine. Hairdressers and beauticians are some group of people who are at increased risk of developing skin related toxicity to hair dyes. The para-phenylene-diamine that is present in the hair dye is the compound which is responsible for these skin reactions. These adverse reactions also occur with temporary tattooing where henna is mixed with para-phenylene-diamine. para-phenylene-diamine has allergic properties and on repeated exposure has mutagenic effects as well and this the reason for the patients developing allergic contact dermatitis[28] following exposure to this compound. Typical lesion is eczematous dermatitis. The patho physiology

behind this is type 4 hypersensitivity reaction. Tattooing in early period of life can sensitize the individual to para-phenylene-diamine. para-phenylene-diamine by itself does not cause any allergic reaction because the para-phenylene-diamine that is present in the hair dyes and tattoo are in the inactive state. These when exposed to atmospheric oxygen undergoes oxidation which then produces an active component known as para-quininediamine. This compound is responsible for the allergic skin reactions that occur following tattooing.

So whenever the individual is exposed to hair dye containing para-phenylene-diamine later in his life can develop severe allergic reactions. These involve the scalp, face, eyes and neck. The scalp the main part affected and the severity varies from simple itching to sever inflammation with erythema and exudations which are prone for secondary bacterial infections. Rarely this can lead on to the development of angioneurotic edema and patient might end up in acute respiratory distress. The extension of these reactions beyond the neck is rare but can extend beyond that to reach the upper chest wall and even into the upper limbs.

CROSS REACTION TO PARA-PHENYLENE-DIAMINE:

Para-phenylene-diamine has a structure with a benzene ring and there are certain other compounds which have benzene ring and the differ in the structure that only the molecules which are attached to the para position

of the benzene ring varies. Some of the compounds are toluene-2,5-diamine, p-aminophenol, 2-nitro-PPD. These are also used as dyes and are a constituent of tattooing substances. As a result when these substances are used for tattooing and later on if Para-phenylene-diamine containing hair dyes are used for the scalp the patient can still develop allergic contact dermatitis due to cross reaction between these compounds.

PROPYLENE GLYCOL:

This compound which is used as a solvent in the hair dye preparation has also been found to have toxic effects which includes,

1. Metabolic acidosis
2. Neurotoxicity
3. Cardiac toxicity
4. Renal toxicity

METABOLIC ABNORMALITIES:

The systemic toxicity due to propylene glycol are rare following oral ingestion as the dose required to cause toxicity by this route is high but rather it is common with intravenous route. Propylene glycol induced metabolic abnormalities are rare and they remain under recognized complication in the setting of hair dye poisoning as most the clinically apparent features are mainly due to PPD toxicity. The patients tend to have

hyperosmolarity with anion gap metabolic acidosis (lactic acidosis) which has to be confirmed with an arterial blood gas analysis. Later on patients can also develop hemolysis which is related to the hyperosmolarity produced by the compound.

NEUROTOXICITY:

The neurological manifestations include alteration in the sensorium ranging from drowsiness and stupor and can even cause coma and death.

CARDIOTOXICITY:

This is also a rare entity with oral ingestion and more common with intravenous route. The common features being,

1. Hypotension
2. Pre-syncope (giddiness)
3. Syncopal attacks

The associated ECG findings being

1. Bradycardia
2. Non-specific QRS and T wave abnormalities
3. Ventricular arrhythmias

RENAL TOXICITY:

This is a very rare manifestation of propylene glycol toxicity presenting as acute kidney injury. The pathological mechanism includes

swelling and vacoulation of the epithelium in the proximal tubules later on leading to acute tubular necrosis[13].

RESORCINOL:

The fatal dose of resorcinol is estimated to be 30mg/kg or more. The common manifestations include,

1. Neurological
2. Hematological
3. endocrinological

NEUROLOGICAL:

The neurological features of resorcinol toxicity occurs usually to acute intoxication mainly by oral route. This can occur either by suicidal intent or accidental poisoning. The feature include,

1. convulsions
2. altered sensorium
3. respiratory failure

CONVULSIONS:

This occurs with acute intoxication of resorcinol and presents as generalized tonic-clonic seizures.

ALTERED SENSORIUM:

The sensorium of the patient is affected in acute toxicity . Common features include dizziness and lethargy which might be the earliest feature. Later on deterioration of the conscious level can occur starting from drowsiness, stupor and even coma and death can occur. One other rare manifestation is motor weakness.

HEMATOLOGICAL:

This itself being a complication of resorcinol poisoning mainly methemoglobinaemia, but is rarely encountered in patients with supervasmol poisoning as PPD related features occur at a smaller dose when compared to the features of resorcinol and propylene glycol which needs larger amounts of ingestion.

ENDOCRINOLOGICAL:

On chronic dermal exposure to resorcinol has been associated with thyroid abnormalities mainly hypofunctioning of the gland. Pathologically the feature is follicular epithelial cell lengthening. The free T4 and TSH levels are found to be decreased in these patients. With the withdrawal of the drug the effects usually completely reverse and the thyroid hormone levels come back to normal. The time taken for this takes a minimum of 6 weeks to a maximum of 3 months.

MANAGEMENT:

INVESTIGATIONS:

All patients who are admitted with super vasmol poisoning are to be investigated with baseline laboratory tests and they have to be monitored at regular intervals for the worsening or improvement of the condition of the patient. The following investigations are done.

1. Complete blood counts
2. Peripheral smear
3. Renal function tests
4. Serum electrolytes
5. Serum calcium levels
6. Liver function tests
7. CPK-TOTAL
8. LDH
9. Urine deposits for myoglobin
10. Urine albumin

COMPLETE BLOOD COUNTS:

This shows a increase in the total leucocyte count which is the commonest abnormality related to hemogram. The leucocytosis has predominant neutrophils[27]. Mild decrease in the hemoglobin levels were observed in some patients secondary to lysis of the red blood cells[27].

Thrombocytopenia have also been found in a study conducted previously[27].

PERIPHERAL SMEAR:

Peripheral smear study of the patients who consumed super vasmol poison might show lysed erythrocyte[27].

RENAL FUNCTION TESTS:

In the early phase or the acute phase of poisoning patients usually have a normal renal function. But the renal functions have to be monitored regularly as they are an increased risk of developing acute kidney injury secondary to myglobin related acute tubular necrosis. Progressive increase in the blood urea and serum creatinine levels will be noticed once the patients develop acute kidney injury. This is an indication for temporary renal replacement therapy. Throughout the course of stay in hospital, patient's renal functions are monitored and the decrease in the levels can be evident as the patient recovers from acute tubular necrosis and as the renal function improves.

SERUM ELECTROLYTES:

The electrolyte that is commonly deranged in patients with super vasmol poisoning is potassium levels. The abnormality is usually a hyperkalemia occurring due to lysis of the skeletal muscle cells predominantly and to some extent because of the lysis of erythrocytes,

hepatocytes and cardiac myocytes. This is usually associated with increase in the serum levels of other intracellular enzymes mainly creatine phosphokinase and lactate dehydrogenase.

SERUM CALCIUM:

The para-phenylene-diamne which is consumed by patient reaches the muscle cells. There it binds to the sarcoplasmic reticulum. This attachment causes the increase in the release of the calcium from the sarcoplasmic reticulum into the sarcoplasm. Increases intracellular concentration causes strong binding of calcium to the contractile elements of the skeletal muscle causing sustained contraction of the muscle fiber leading to lysis of the affected cells. This increased intracellular shift of calcium and its increased utilization causes a decrease in the serum calcium levels[27]. The severity of hypocalcemia increases with the volume of the poison consumed and extent of rhabdomyolysis.

LIVER FUNCTION TESTS:

One of the important late onset complication of para-phenylene-diamine poisoning is toxic hepatitis. This causes extensive hepatocyte damage leading on to increase in the serum levels of bilirubin[27] and also the increase in the serum levels of the aspartate transaminase and alanine transaminase[27]. Monitoring of the liver function tests is done regularly to

assess for the improvement and worsening of the hepatic status of the patient.

CPK-TOTAL:

Rhabdomyolysis is a late feature of super vasmol poisoning. This causes release of all the intracellular enzymes into the blood stream. Creatinr phosphokinase is an important intracellular enzyme of the muscle and hence the serum levels of these enzymes is also elevated. There are three different isomers of creatine phosphokinase and they are CPK-BB which is produced in the brain cells, CPK-MM which is present in the skeletal muscles which contains sarcomeres, CPK-MB which is present in the cardiac myocytes. Para-phenylene-diamine poisoning causes damage to the muscle cells which causes the liberation of these enzymes into the blood resulting in the increase in the serum levels of CPK. The skeletal muscles related CPK is increased predominantly when compared to others.

LACTATE DEHYDROGENASE:

This is as such is also an intracellular enzyme which causes is released in times of cellular damage. There are five isoforms of the enzyme from LDH-1 to LDH-5. Lactate dehydrogenase type 1 is present in the cardiac myocytes and in the erythrocytes. Type 2 is present in the cells of the reticulo-endothelial system. Type 3 is belong to the alveolar epithelial cells of the lungs. Type 4 is nonspecific and it is present in the placental cells,

renal epithelial cells and pancreatic cells. Type 5 is related to the hepatocytes and the skeletal myocytes. Normally the isoform of lactate dehydrogenase which is present in the peripheral blood is type 2. Usually the biochemical tests done in all routine laboratories test for this isoform. So in para-phenylene-diamine poisoning due to severe muscle damage the total LDH is elevated.

URINE MYOGLOBIN:

The ultimate effect of rhabdomyolysis is the release of the muscle pigment, myoglobin. Myoglobin when released into the blood reaches the kidney where it enters the renal tubules and are excreted into the urine which produced the dark colored or cola colored urine which occurs in super vasmol poisoning. If severe the levels of myoglobin in the serum increases which increases the renal filtration of the pigment. Once they enter into the tubules they get clogged within the tubules leading on to acute tubular necrosis which is the most dreaded complication of para-phenylene-diamine poisoning.

ALBUMINURIA:

Glomerular basement membrane damage by the myoglobin present in the blood reaching the renal glomerulus results in the entry of albumin into the tubules which is then excreted. So albuminuria is an evidence of myoglobinuria induced basement membrane damage but this

usually recovers completely without any residual permanent glomerular damage.

TREATMENT:

Universal decontamination measures are of prime importance and supportive treatment plays the major role in the treatment of poisoning with hair dyes containing PPD. Specific guidelines are still to be proposed for this type of poisoning. Forced alkaline dieresis has been tried in patients and this has shown promising results in preventing the development of acute kidney injury. The complications like acute respiratory distress is managed with emergency tracheostomy and acute kidney injury with temporary renal replacement therapy mainly hemodialysis. Metabolic acidosis if occurs is also corrected appropriately with sodium bicarbonate supplementation and the associated hyperkalemia is treated appropriately.

UNIVERSAL DECONTAMINATION:

Gastric decontamination is done as soon as the patient is admitted as this prevents the further absorption of the dye thereby preventing the systemic complications mainly acute kidney injury as it is the most common delayed cause of death which can even occur about a week after the consumption of the poison. Gastric lavage has been beneficial if done within 4 hours of consumption of the poison. But the usefulness of this has been

controversial as studies have been done and it is said that gastric lavage has to be done only after knowing the constituents of the hair dye[6].

SUPPORTIVE MEASURES:

Immediately after the patient arrives and after gastric decontamination the vitals of the patient is assessed including the pulse, blood pressure, respiratory rate and oxygen saturation. Stabilization of the patient is of prime importance in the acute setting. This is done with supplementary nasal oxygen either via mask or nasal prongs. Intravenous glucocorticoids like hydrocortisone and anti-histaminics are administered to relieve edema.

FORCED ALKALINE DIURESIS:

This involves the elimination of the acidic compounds by altering the pH of the urine i.e., by making it alkaline. This makes the acidic compounds more ionized in the renal tubular fluid. Once the fluid becomes ionized in the renal tubules, the tubular cells prevent the movement of these ions into the interstitial space. Thereby the desired compound is concentrated and excreted in the urine. This is done by initial infusion with normal saline over one hour followed by sodium bicarbonate infusion in 5% dextrose solution which is made to run over an hour and then potassium replacement is done with intravenous infusion of potassium chloride over an hour. This each 3 hour cycle is repeated till the urine color returns back to

normal. While doing this the patients are constantly monitored for urine output which must be more than 4-6ml/kg/hr. if this is not achieved patients are given intravenous loop diuretic i.e., frusemide 20mg iv bolus and then monitored for urine output. If there is no improvement of urine output then diuresis has to be stopped immediately.

The main aim of subjecting the patient to forced alkaline dieresis is to remove all the myoglobin that is being released in the circulation with enters the renal tubules because if these compounds just get accumulated in the renal tubules the progress on to pigment nephropathy, acute tubular necrosis ultimately acute kidney injury.

TREATMENT OF COMPLICATIONS:

ACUTE RESPIRATORY DISTRESS:

This is a relatively common early complication of PPD poisoning and is estimated as the commonest cause of death within 6 hours of consuming hair dye containing para-phenylene-diamine. Patients with minimal respiratory distress are treated with supplemental oxygen therapy either by face mask or nasal prongs. If patient develops severe allergic reactions with angioneurotic edema then treatment is emergency tracheostomy or cricothyroidectomy. This usually relieves the patient of respiratory distress but if acute respiratory distress syndrome develops then ventilator support becomes mandatory.

ACUTE KIDNEY INJURY:

This is the estimated to be the most fatal complication and the most common delayed cause of death in cases of poisoning with hair dye containing para-phenylene-diamine occurring secondary to rhabdomyolysis. This leads on to acute tubular necrosis. Apart from para-phenylene-diamine, resorcinol and propylene glycol also causes renal toxicity predominantly by causing damage to the proximal tubular epithelial cells. But the dosage of resorcinol and propylene glycol needed to produce these effects on the kidney is much higher than it is actually used in the hair dye preparations There is rapidly progressing renal failure and the ultimate treatment is temporary renal replacement therapy either hemodialysis or peritoneal dialysis. Certain studies have shown no difference in the outcome of patients treated with hemodialysis and peritoneal dialysis yet most prefer hemodialysis over peritoneal dialysis.

PEVIOUS STUDIES:

Study 1:

According to **A PROSPECTIVE STUDY ON PREVALENCE OF POISIONING CASES WITH FOCUS ON VASMOL POISONING BY UDAYKIRAN GELLA**[1] about 419 patients were studied who were admitted in their hospital with history of poisoning about 62% of the cases were due to poisoning with super vasmol. Their study showed a female

predominance in the incidence of super vasmol poisoning with 70% of the admitted population being females and the rest 30% being males. The age distribution showed 58% incidence in the age group of between 12-25 years both in males and females. 20% of the female population was housewives. 50 % of the male population was manual laborers belonging to different occupation like farmers. Coolie, etc. 42 patients were students. Out of the total cases admitted 38% of the cases developed edema of the face and neck. 23.3% developed stridor and 18.8% developed myalgia. 33% of the patients developed cola colored urine due to rhabdomyolysis and myoglobinuria. 17% of the cases developed respiratory distress and were treated with emergency tracheostomy. Out of the 419 patients admitted with super vasmol poisoning 19 patients expired and the leading cause of death in this study was due to cardio-respiratory failure(11 cases) followed by myocarditis(5 cases), cardiac arrest(2 cases) and acute kidney injury(1 case).

STUDY 2:

According to **a study conducted on PARAPHENYLENE DIAMINE POISONING by Bashir Ahmed Khuhro et al** [2] 16 patients were studied who were admitted with super vasmol poisoning. The sex distribution showed a female predominance with 87.5% of the admitted population being females and the rest i.e., 12.5% were males. The

distribution showed 18.8% of the patients belonged to the age group of 12-20 years, 69% belonged to the age group of 21-30 years and 12.5% belonged to the age group of 31-40 years. Majority of these patients were from the lower socio-economic status them contributing to about 94% of the admitted cases. 3/4th of the admitted cases were due to suicidal poisoning and the rest 1/4th were due to accidental consumption. According to the presentation to the hospital 100% of the admitted cases had throat pain, edema of the face and neck, dysphagia, dysphonia and difficulty in opening the mouth. 81% had myalgia, 62.5% dark urine. 19% of the patients developed acute kidney injury. Regarding the outcome 87.5% (14 cases) developed respiratory distress and were treated with emergency tracheostomy out of which 12 patients needed ventilator support. 37.5% of the cases i.e., 6 patients expired.

STUDY 3:

According to **a study on hair dye poisoning – an emerging problem in the tropics by anugrah chrispal et al** [3] which was conducted over a period of 42 months about 13 patients were admitted with super vasmol poisoning and they were studied the clinical profile and outcome. Out of the 13 patients admitted 11 patients were females and 2 were males. The age distribution showed a clustering in the age group of 23-31 years. 75% of the patients had edema of the face and neck. 38.5% of the patients

developed acute kidney injury. 84.6% of the cases developed metabolic acidosis and 61.5% of the patients had low calcium levels. 92.3% of the patients showed features of rhabdomyolysis which was evident by the elevated levels of muscle related CPK. Out of the 13 patients admitted with super vasmol poisoning 61.5% of the patients recovered completely after intensive care and supportive management. 38.5% of the patient expired and the causes of death were acute kidney injury and cardio respiratory arrest. According to their study tracheostomy done early in the course of the illness i.e., immediately when the patients developed respiratory distress due to cervicofacial edema and early institution of forced alkaline diuresis prevented the patients from developing acute kidney injury thereby decreasing the mortality rate due to super vasmol poisoning. The causes of death were studied as due to late presentation from the time of consumption of poison, poor neurological status at the time of admission and inadequate gastric lavage in the primary care set up.

STUDY 4:

According to the study on the clinical review on hair dye poisoning by Dr.D.Radhika et al [4] which was conducted over a period of 4 months included about 264 patients who were admitted in the intensive care unit with super vasmol poisoning. Out of the total cases admitted the

female admissions constituted about 65% of the total admissions. Rest of the 35% were males. Out of the total cases including both males and females 34% belonged to the age group of 15-25 years, 63% belonged to the age group of 26-35 years, 3% fell in the age group of 36-45 years and no cases of more than 45 years were admitted. Almost half of the admitted individuals the reason for consumption of the poison was family problems. Only 3 of 264 admitted cases were due to accidental consumption of the poison and the rest consumed the dye mainly with suicidal intent. The major clinical presentation was with cervicofacial edema with 74% of the patients had it when they were admitted or developed subsequently after admission. 71% had difficulty in swallowing, 60% of the cases had dark colored urine, 45% had albuminuria, 31% had muscle pain and tenderness, 21% of the patients had breathing difficulty. The main complication that these developed were acute respiratory distress and acute kidney injury. 89% of the admitted cases developed respiratory distress and emergency tracheostomy was carried out. 21% of the admitted cases developed acute kidney injury and were treated with hemodialysis. The overall mortality rate in this study was around 12% and cause of death being either respiratory failure or acute kidney injury. This was lower when compared to other studies conducted in morocco. The lower death rate was possibly due to the

early institution of gastric decontamination, early tracheostomy and temporary renal replacement therapy.

STUDY 5:

A Prospective Clinical Study of Myocarditis in Cases of Acute Ingestion of Paraphenylene Diamine (Hair dye) Poisoning was conducted by PK Jain et al [5]. This study was conducted in Uttar Pradesh. The total number of patients taken in the study was 1595. This was a prospective study and the clinical and the outcome of these patients were studied. This study made emphasis on the myocardial involvement in poisoning with hair dyes containing para-phenylene-diamine and resorcinol. 97% of the patients consumed the poison with a suicidal intent while the remaining 3% was by accidental consumption. Homicidal poisoning was also reported in 6 of these patients. 76% of the admitted cases were females and the rest 24% were males. Almost half of these patients fell in the age group of 15-25 years of age and the sex ratio was more in female preponderance. 40% of the patients were in the age group of 26-35 years of age. 11% belonged to the age group of 36-45 years of age, but in this group a male preponderance was present with number of male patients in this group were more than twice the number as that of the females in the same group. The in cadence of poison was least in age >45 years of age. The commonest presenting feature as in all other

studies was cervicofacial edema constituting for about 74% of the patients at the time of admission. This was associated with dysphagia which was the second commonly encountered symptom with 72% of the patients having it. 54% of the cases has cola colored urine and 48% has muscle pain, rigidity and tenderness. Significant percentage of patients had ECG changes with 22% having sinus tachycardia 9% developing fatal ventricular arrhythmias mainly ventricular tachycardia and ventricular fibrillation. 3% of the patients developed convulsions during the course in the hospital mainly geleralised tonic-clonic seizures. The predominant complications were respiratory distress which was present in 23% of the patient and they were taken up for emergency tracheostomy. 9% of the patients developed acute kidney injury. 15% of the patients showed evidence of cardiac involvement mainly as myocardial damage due to myocarditis. This was evident from the elevated cardiac enzymes (57.5% - 138 cases), left ventricular systolic dysfunction (54% - 130 cases) as evidenced by the echocardiography with an ejection fraction of less than or equal to 35%. Mortality due to cardiac involvement was 29% (69 cases). Sudden cardiac death was present in 9% of the cases mainly due to ventricular arrhythmias (ventricular tachycardia and ventricular fibrillation). Out of the 240 cases with myocarditis 69 patients expired which constituted 29%. Sudden cardiac death due to ventricular tachycardia and ventricular fibrillation accounted for 14 patients with

cardiac involvement which constituted 14% of the patients. Mortality benefit was found with treatment with methylprednisolone over hydrocortisone which accounted for 14% when compared to 27% for cases treated with hydrocortisone.

STUDY 6:

A study on the evaluation of treatment modalities of hair dye poisoning by akshaya srikanth et al [11]. This was a study which was conducted in south India regarding the treatment modalities that is being done for patients admitted with hair dye poisoning. Those patients with suicidal or accidental consumption of hair dyes develop clinical features due to severe allergic and irritant properties of the dye. This causes direct irritation and inflammation of the areas where it comes in contact with. This usually involves the tongue, buccal mucosa, pharynx and larynx ultimately leading to edema of the face and neck. This causes acute respiratory distress so early tracheostomy as the patient develops respiratory symptoms decreases the mortality rate. These patients are at in increased risk of developing acute hypocalcemia which is treated with intravenous 10% calcium gluconate which is given slowly over 10 minutes to avoid systolic arrest. This is repeated as and when required. Metabolic acidosis associated with hyperkalemia as treated with sodium bicarbonate infusion.

Rhabdomyolysis is a dreaded complication of hair dye poisoning as this can lead on to acute tubular necrosis due to clogging of the pigments in the tubules. This can be prevented by early institution of forced alkaline dieresis. This causes flushing of the myoglobin which is released from the lysed myocytes. Once the patient develops acute kidney injury the ultimate mode of treatment is temporary renal replacement therapy with hemodialysis or peritoneal dialysis.

STUDY 7:

According to **Ram et al** [12], a prospective study was done regarding the clinical profile of hair dye poisoning mainly those containing para-phenylene-diamine with particular emphasis for the renal toxicity and the related complications. This study was based on the admissions of patients with hair dye poisoning over a 4 year period. 10 patients were admitted.100% of the patients had features of rhabdomyolysis which was evident from elevated muscle enzymes. 7 patients (70%) developed acute kidney injury. 20% (2 patients) had severe angioedema and developed respiratory distress. They were taken up for emergency tracheostomy. 20% (2 patients) had to be ventilated. 1 patient (10%) expired to acute renal injury and other person survived. The renal biopsy done in these patients showed acute tubular necrosis.

STUDY 8:

A case report was done by **Hayman et al** [13] in which a patient with Stenotrophomonas maltophilia pneumonia was treated with high doses of trimethoprim-sulfamethoxazole intravenously and lorazepam infusion. These medications use propylene glycol as a constituent. Later on patient developed features of acute kidney injury. The drugs were replaced with drugs without propylene glycol. Renal biopsy was done. It showed involvement of the epithelial cells of the proximal part of the renal tubule. The typical finding was cellular swelling. Later stages were associated with formation of vacuoles within these cells. The clinical features include metabolic acidosis with increased anion gap. Treatment of the acute kidney injury with renal replacement therapy and metabolic acidosis was treated with intravenous sodium bicarbonate infusion. Dyselectrolytemia is corrected appropriately.

STUDY 9:

According to **a study on the Clinical manifestations of systemic paraphenylene diamine intoxication by kallel et al** [14] which included 19 patients admitted in the intensive medical units of a tertiary care centre. The study was a retrospective one and the data was collected over the previous six year admissions with para-phenylene-diamine poisoning. The

clinical profile, treatment and the outcome of the patients were studied. All the 19 patients had features of metabolic acidosis and rhabdomyolysis as evidenced by the elevated muscle enzymes. $4/5^{th}$ of the patient i.e., 79% had edema involving the face and the neck either on admission or during the stay. 74% of the patients developed cola colored or chocolate colored urine. 69% of the patients had edema involving the pharynx and larynx presenting as respiratory distress. $1/4^{th}$ of the patients had muscle pain, swelling and tenderness. Almost half (47.3%) of the patient developed renal failure in the form of acute kidney injury secondary to rhabdomyolsis and myoglobin induced acute tubular necrosis. One other significant metabolic abnormality as per this study was hyperkalemia which was present in about 26.3% of the patients. The patients were treated with gastric decontamination initially. Then supportive treatment was given with intravenous fluid correction and intravenous steroids. 26.3% of the patient who developed acute kidney injury was treated with renal replacement therapy. 84.2% of the patients required mechanical ventilation for respiratory distress. The outcome of the patients varied with six of the patient succumbed. In 5 out of the 6 the death was due to acute renal insufficiency and their histopathological examination of the renal tissue showed acute tubular necrosis with clogging of the tubules with casts. These casts are the myoglobin that has been liberated from the lysed myocytes.

STUDY 10:

Poisoning with hair-dye containing paraphenylene diamine over ten years experience was a study carried out by Suliman SM et al [15]. This was a prospective study which included ten year admissions of patients with para-phenylene-diamine poisoning. Totally 150 cases were admitted and all of them were included in the study. The study focused more towards the renal complication related to para-phenylene-diamine poisoning. 60% of the patients i.e., 90 out of the total 150 cases admitted developed myoglobinuric acute tubular necrosis presenting as acute kidney injury. All of these patients were treated with temporary renal replacement therapy ranging from 1 day to a maximum of 42 days. The average period for which these patients were on dialysis was fifteen days. 100% of the patients recovered with hemodialysis. The study also focused regarding the development of permanent damage to the glomerulus following recovery from the acute event for which renal biopsy was done and histopathological examination was carried out. The results showed a normal glomerulus in all these patients.

STUDY 11:

Bourquia A et al [16] reported 4 patients with para-phenylene-diamine poisoning from the department of nephrology. All 4 of the patients were females and they belonged to the age group of 18-35 years of age. Initial presentation in these patients was acute respiratory distress due to severe angioedema. They were stabilized in the intensive care units and 75% of them were subjected to emergency tracheostomy to relieve the distress. All of them had features of rhabdomyolysis and 50% of them developed acute renal insufficiency. They were treated with renal replacement therapy in the form of hemodialysis. After a few cycles of hemodialysis both of them improved which was attributed to the early recognition of the renal injury. All 4 patients were followed up and their long term prognosis was found to be good.

STUDY 12:

Sir Hashim M et al [17] reported a set of thirty one children admitted with history of poisoning with hair dye containing para-phenylene-diamine mixed with the traditional henna. This study focused on the type of poisoning and the complications, treatment modalities and outcome in these children. The study was reported in Sudan. 38% of the children were admitted with accidental poisoning. 32.2% children were admitted with

poisoning due to suicidal intent. 9.6% were admitted after homicidal poisoning. 19% were suspected to have experienced toxic effects due to repeated dermal exposure. 48% of the children had severe cervicofacial edema with associated edema of the pharynx and larynx on presentation and they were in acute respiratory distress and hence tracheostomy was done in all of them and assisted mechanical ventilation was given.16% of them developed acute kidney injury and were subjected to temporary renal replacement therapy in the form of peritoneal dialysis. The mortality rate was high accounting for about 41.9% (13 cases) of the total admissions and all of them occurred within the first day of presentation. 100% of them were in circulatory shock on presentation which was suspected as the cause for the high mortality in this study.

STUDY 13:

Namburi rajendra Prasad et al [22] did a retrospective study on the biochemical profile in patients who were admitted in their hospital in the intensive care and emergency care units over the previous 24 months. This included 81 patients out of which the history regarding the volume of consumption could not be obtained from 41 cases which equals to approximately 50% of the cases. In those subset where the history could be obtained, they were categorized into those consumed less than and more

than 50 ml. the biochemical profile including the renal function tests, serum transaminase levels, creatinine phosphokinase levels, lactate dehydrogenase levels and electrolyte levels mainly serum potassium were taken into consideration and were compared between the 2 groups. There was significant alterations in the levels of urea, creatinine and potassium between the 2 groups and had a p value of <0.01. this showed than the group which consumed more than 50 ml of the poison developed more severe complications when compared to the group which consumed less than 50 ml. while the other parameters did not show any significant variations between the 2 groups. 40.9% of the patients in group II required hemodialysis as they developed acute kidney injury while only 6% developed renal insufficiency and needed renal replacement therapy. 47% of cases in group I required ventilatory support while the requirement in group II was 36.6%. Eventually the duration of stay in hospital also was more in group II when compared to group I. Mortality rate was 9%(2 cases) in group II while it was 5.88% (1 patient) in group I. the long term follow up in patients who recovered were satisfactory in both the groups and they did not develop chronic kidney disease.

STUDY 14:

A retrospective study was carried out by Mary Nirmala Suganthakumar et al[23] in patients who were admitted in the medicine and oto-rhino-laryngology department of their hospital with allege history of consuming Super Vasmol hair dye. The patients included in the study were those admitted in the previous 12 month period. A total of 108 patients were studied for their clinical profile and outcome . Out of those who were admitted the preponderance was towards females who accounted for 64.8% (70 cases) of the cases. Remaining were males constituting 35.2% (38 cases). Majority of the admitted individuals were in the age group of between 21 and 25 years constituting 38.9% (42 cases 74 (68.5%) patients needed an ENT opinion. Cervico-facial oedema was the main clinical manifestation in 74 patients. 33 of them underwent tracheostomy. The mortality in 108 patients was 22.2% and after tracheostomy 21.2%.

STUDY 15:

A study was conducted in Tirupati by Suneetha et al on the clinical presentations and predictors in the outcome of super vasmol poisoning [23]. The study comprised of 234 patients who were admitted in the intensive care units and emergency care units of their hospital in the previous years. These records of these patients were taken up

retrospectively. Of the total admissions 166 (71%) were female patients and the remaining 68 patients (29%) were males. As with all other studies this study also showed a female predominance in the in super vasmol poisoning. The predominant cases were between 16 years and 34 years of age. About 161 patients (69%) had edema of the face and neck with varying levels of respiratory distress in 56% (131 cases) of them. 27% (63 patients) developed acute renal shutdown and 2.6% (5 patients developed neurological features in the form of generalized tonic-clonic seizures. Out of the 56% of the patients who developed respiratory distress some of them needed assisted mechanical ventilation and tracheostomy. This portion of the patients accounted for 37% and 22% respectively. Myocardial involvement was present in 11.2% of the patients in the form of arrhythmias probably due to myocarditis. The duration of stay of these patients in the hospital was up to 12 days with an average duration being 6 days. The average intensive care unit stay of these patients was 4 days. The mortality rate in this study was high were 53 patients expired which constituted of around 23% of the total admissions. This was attributed to the delay in arriving to the hospital and delay in the early resuscitative measures in the primary health care centers. Also the presence of poor neurological status on admission as evidenced by a Glasgow coma scale of less than 8 was also a significant predictor of the mortality rate.

STUDY 16:

Akbar et al did a study **on the clinical presentation, clinical and biochemical profile, treatment modalities and outcome in super vasmol poisoning [26].** This was an observational study which included the patients admitted in their hospital in the intensive care units. Totally five patients were studied. 100% of them were female and all of them belonge to the age group of between 20 and 25 years of age. The study emphasized on the complications due to hair dye poisoning with main concern over the renal and cardiac complications. As per this report, out of the total five patients who were studied about 40% (2 cases) of them developed renal complications in the form of acute kidney injury probably due to acute tubular necrosis secondary to rhabdomyolysis related myoglobinuria. 2 patients (40%) developed hepatitis and 1 patient developed sepsis. The cardiac involvement was not significant as per this study probably due to the relatively small study sample. The predominant ECG changes were sinus tachycardia followed by bradycardia. 1 patient had tall tented T waves. There was no death due to cardiac involvement. Out of the 5 patients one of them expired. The remaining patient who recovered from the illness had a satisfactory long term prognosis without any permanent renal damage. One of the draw-back of this study was that the study population was small.

STUDY 17:

Sushil kumar et al did a study on the biochemical profile in suicidal poisoning with para-phenylene-diamine [27]. This study was carried out in a prospective manner where twenty three patients were studied. The biochemical values are focused to study the type of organ involvement in super vasmol poisoning. Out of the 23 patients, 78% (18 cases) of the cases had leucocytosis predominantly with neutrophils. 62.5% (14 cases) had liver damage evidenced by the elevated alanine transaminase and aspartate transaminase levels and hyperbilirubinemia. 47% (11 cases) of the cases had renal involvement which was associated with an elevated serum creatinine levels. Similarly blood urea levels were elevated in 47% (11cases) of the cases. The calcium levels were significantly low in about 37.5% (9 cases) of the admitted patients. Half of the patients had albuminuria. 4 patients expired and in all of them the cause of death was acute kidney injury. The death occurred in spite of these patients being on analysis[27].

STUDY 18:

This was a case report given from a teaching institution in bengaluru where 2 patients, both of them being female were admitted in

their intensive care unit with edema of the face and neck. Both of them developed rhabdomyolysis as evidenced by the presence of myalgia and muscle tenderness. First patient developed respiratory distress which was minimal and was treated with supplemental oxygen. She recovered from respiratory distress. She was treated with supportive measures mainly fluid therapy and alkaline dieresis to prevent the development of myoglobinuric renal failure. She was treated with intravenous corticosteroids and anti-histaminics for the cervicofacial edema. She recovered with all these supportive measures and her biochemical values mainly the muscle enzymes came back to normal within a week.

The second patient developed severe respiratory distress and was taken up for emergency tracheostomy and she improved by the second day of tracheostomy. She also developed rhabdomyolysis with elevated lactate dehydrogenase and skeletal muscle related creatine phosphokinase. She was also given the routine supportive measures. This included fluid therapy, anti-edema measures which comprised of corticosteroids and anti-histaminics which were given by intravenous route. Alkaline dieresis was given to prevent the development of renal failure. This patient also recovered with progressive improvement in the enzyme levels.

Neither of the 2 patients developed acute kidney injury. One of them developed acute respiratory distress for which tracheostomy was done.

Both the patient recovered completely without any long term complications probably due to the appropriate institution of the gastric lavage and alkaline diuresis.

MATERIALS AND METHODS

MATERIALS:

All patients admitted in the IMCU and medical wards with history of supervasmol poisoning

DURATION OF STUDY: 6 months

TYPE OF STUDY: prospective study

SAMPLE SIZE: 50

INCLUSION CRITERIA:

All patients admitted in the IMCU and medical wards with history of supervasmol poisoning

EXCLUSION CRITERIA:

History of multiple poisoning

Hair dye poisoning other than supervasmol

DOES THIS STUDY REQUIRE ANY INVESTIGATIONS TO BE CONDUCTED IN PATIENTS?

COMPLETE BLOOD COUNT

DIFFERENTIAL COUNT

RFT

LFT

SERUM ELECTROLYTES

URINE DEPOSITS

CPK-TOTAL

ECG

OBSERVATIONS AND RESULTS

SEX DISTRIBUTION

Gender	Number	Percentage
Males	18	34
Females	35	66
Total	53	100

Table.1

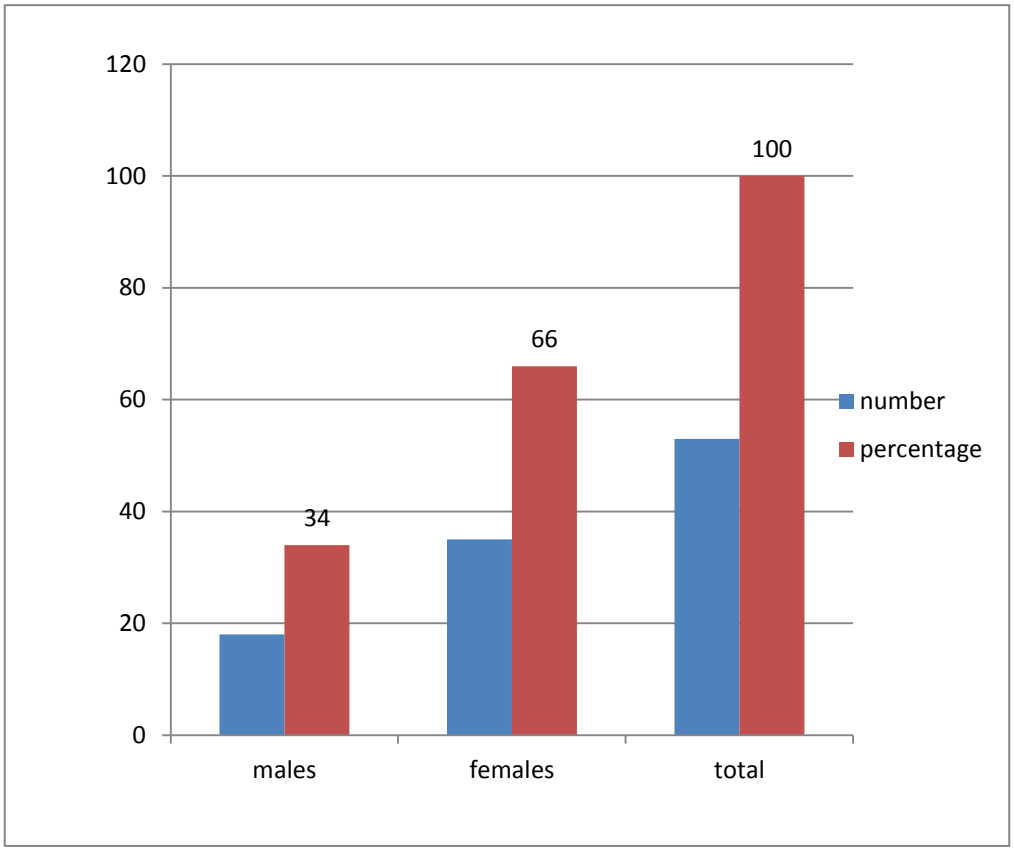


Fig.5

The graph shows the age sex distribution of the study population With females (66%) more than the males (34%).

AGE DISTRIBUITON - FREQUENCY

Age	Frequency	Percentage
<30 years	41	77.3
>31 years	12	22.7
Total	53	100

Table.2

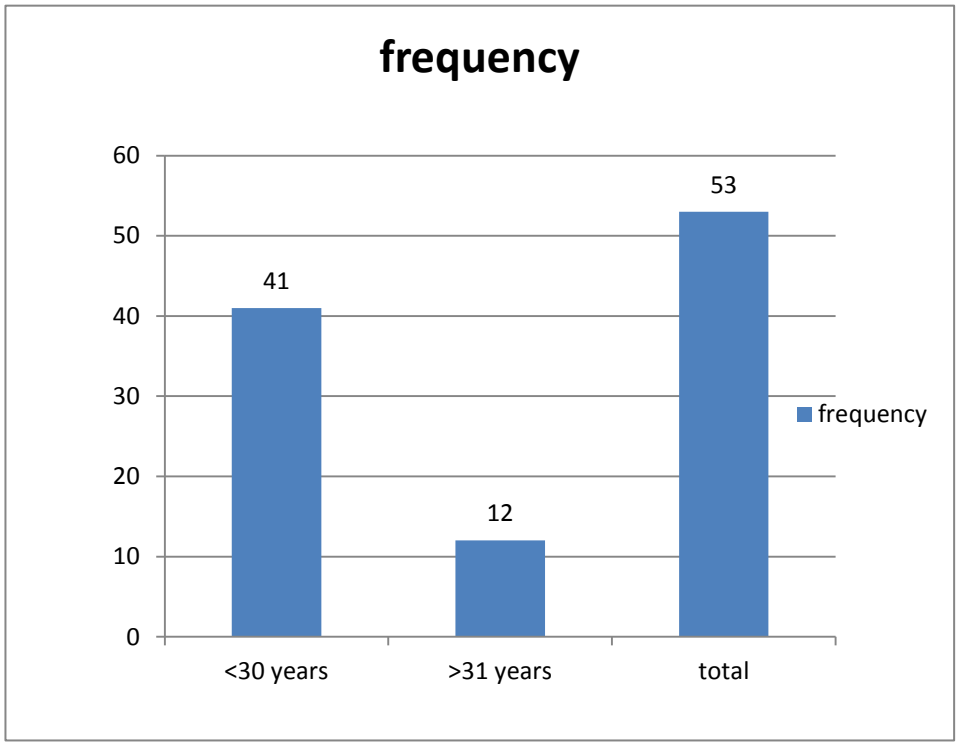


Fig.6

The graph shows the age distribution of the study population with 41 patients less than 30 years of age which constitutes for 77.3% of the study population.

SEX AND AGE RELATIONSHIP (MALES – PERCENTAGE)

	Males	
Age	Number	Percentage
<30 years	11	61.1
>31 years	7	38.9

Table.3

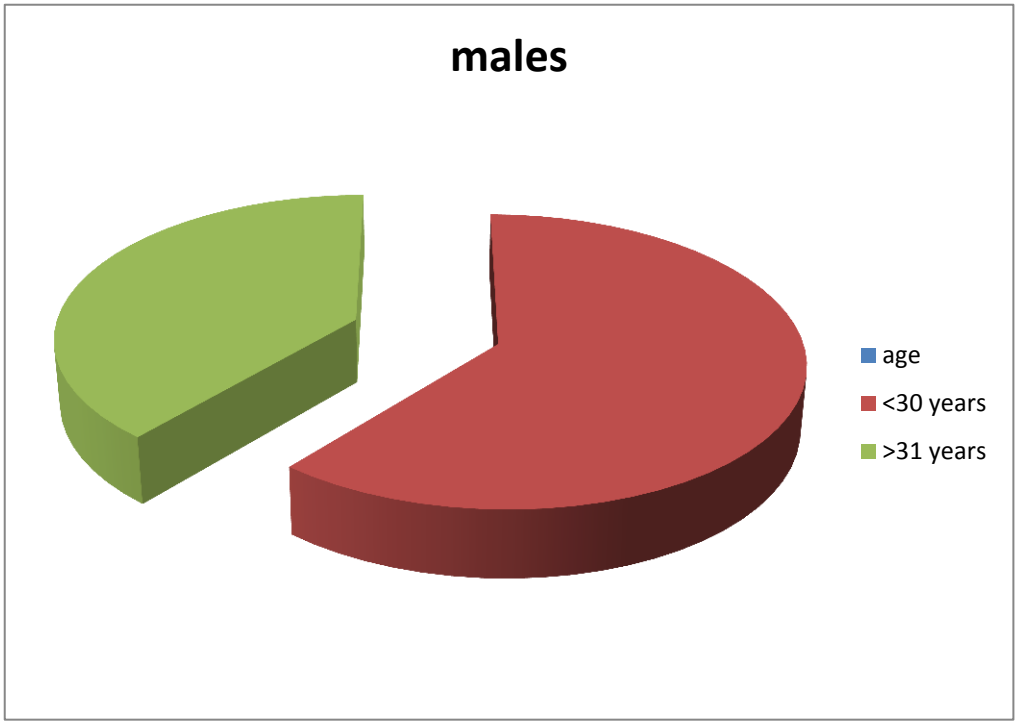


Fig.7

Among the males admitted 61.1% (11 patients) were under 30 years of age and the rest above 31 years accounting for 38.9%

SEX AND AGE RELATION (FEMALES – PERCENTAGE)

Age	Females	
	Count	Percentage
<30 years	30	85.7
>31 years	5	14.3

Table.4

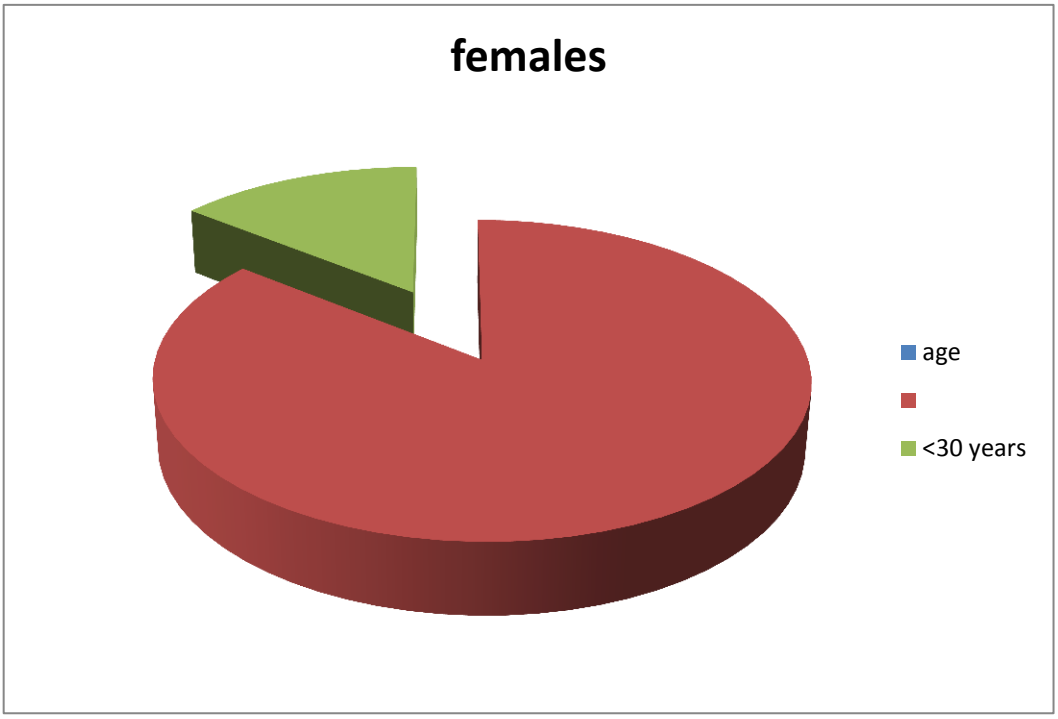


Fig.8

85.7% (30 patients) of the females were under 30 years of age and the remaining 5 patients (14.3%) were above 31 years of age.

VOLUME CONSUMED – FREQUENCY DISTRIBUTION

Volume consumed	Frequency	Percentage
<50ml	27	50.9
>50ml	26	49.1
Total	53	100

Table.5

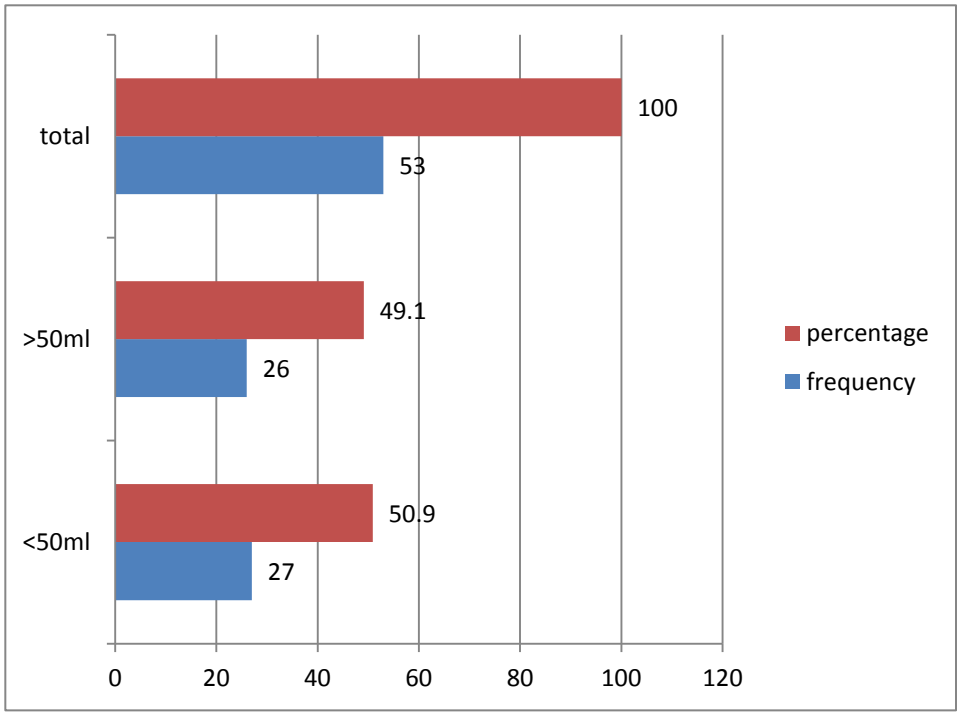


Fig.9

The bar chart shows the frequency distribution of the study population in relation to the volume consumed. Out of the 53 patients, 26 patients (49.1%) consumed more than 50 ml and 27 patients (50.9%) consumed less than 50 ml.

DYSPHAGIA – FREQUENCY DISTRIBUTION

Dysphagia	Frequency	Percentage
Present	36	67.9
Absent	17	32.1
Total	53	100

Table.6

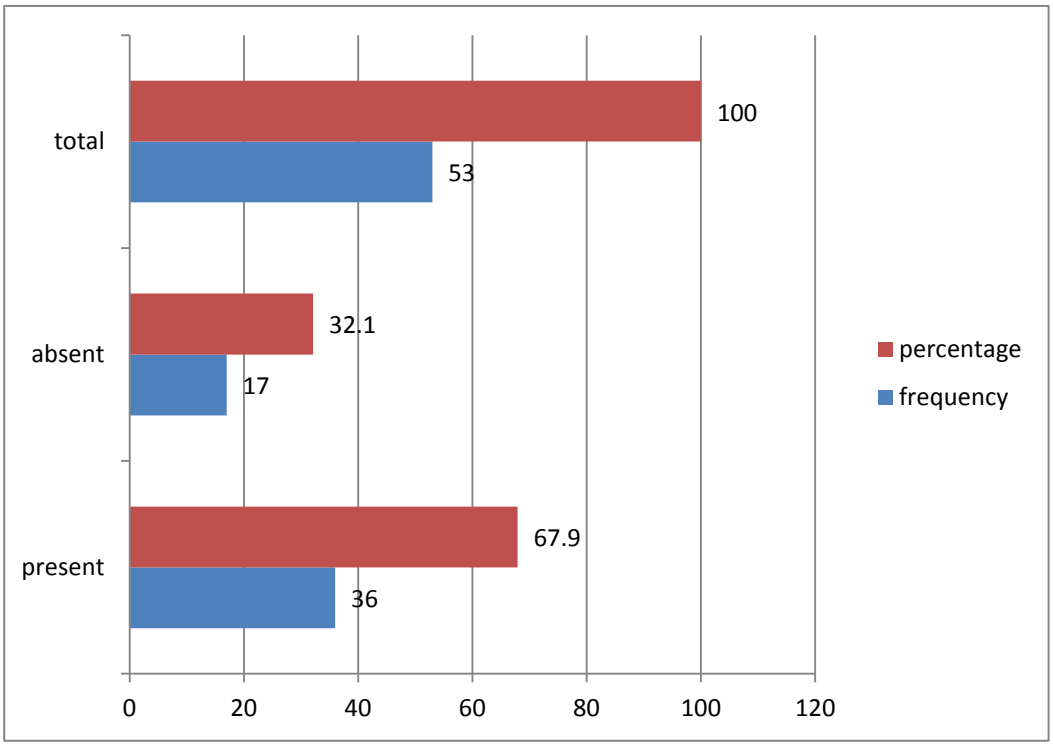


Fig.10

67.9% (36 patients) had dysphagia at the time of presentation which is due to the edema of the buccal, pharyngeal and laryngeal mucosa.

DYSPNOEA – FREQUENCY DISTRIBUTION

Dyspnoea	Frequency	Percentage
Present	14	26.4
Absent	39	73.6
Total	53	100

Table.7

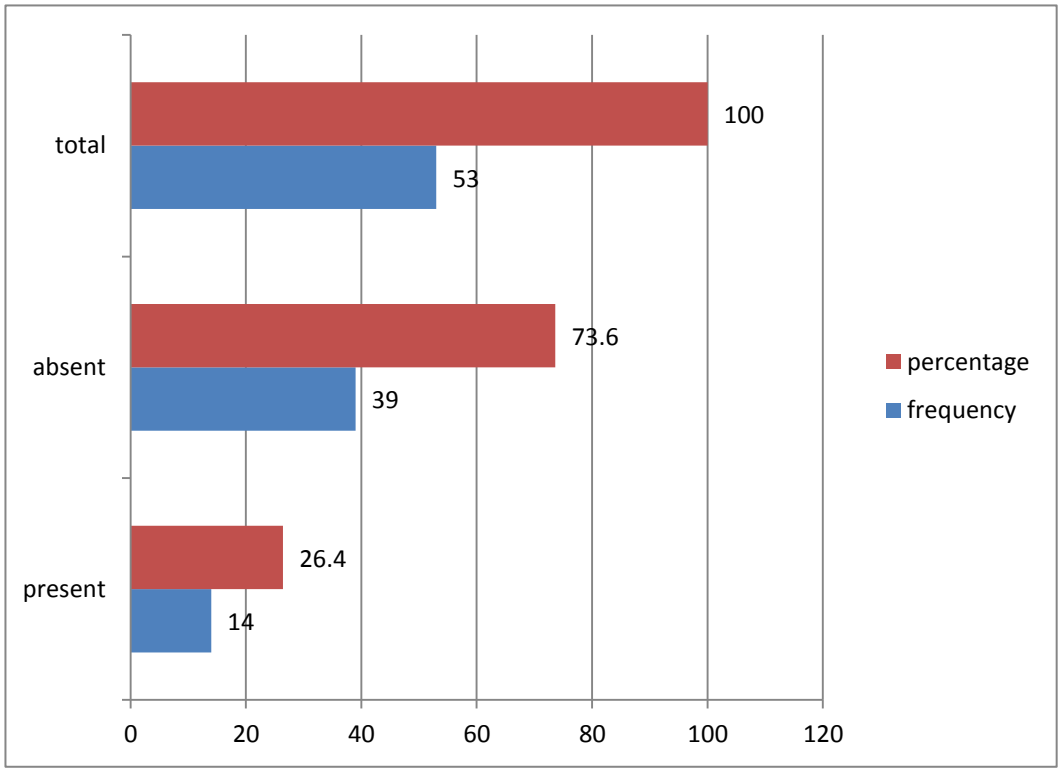


Fig.11

26.4% (14 patients) had dyspnoea at the time of presentation which is due to the edema of the respiratory tract.

MYALGIA – FREQUENCY DISTRIBUTION

Myalgia	Frequency	Percentage
Present	19	35.8
Absent	34	64.2
Total	53	100

Table.8

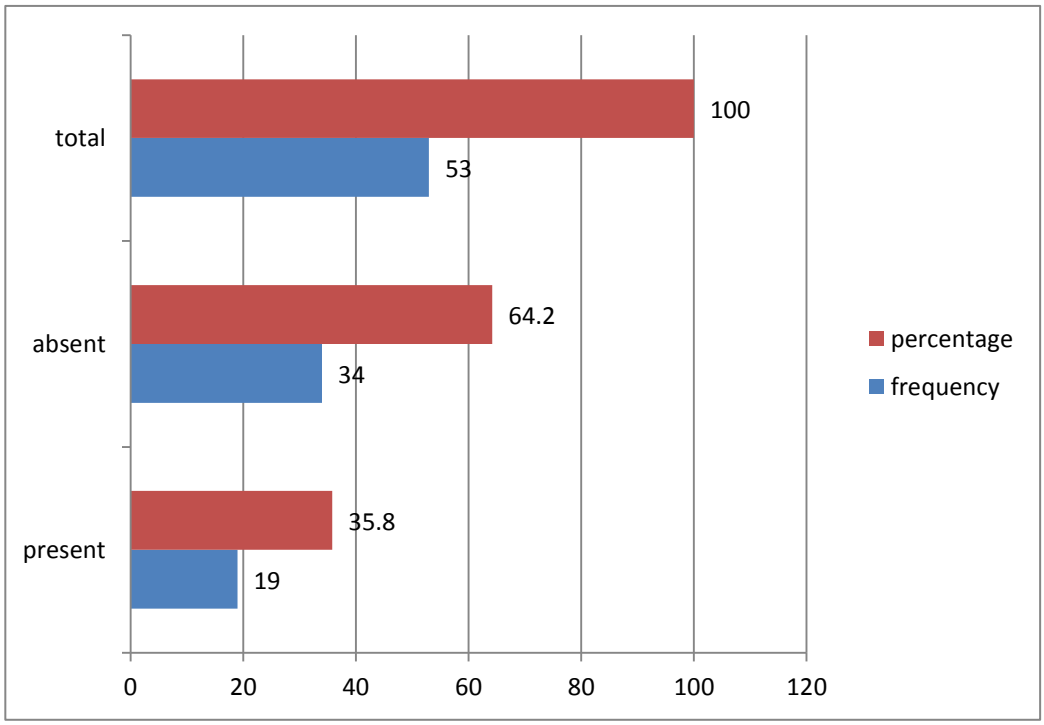


Fig.12

35.8% of the patient (19) had presented with myalgia out of the 53 patients.

URINE COLOR – FREQUENCY DISTRIBUTION

Urine color	Frequency	Percentage
Cola colored	25	47.2
Normal	28	52.8
Total	53	100

Table.9

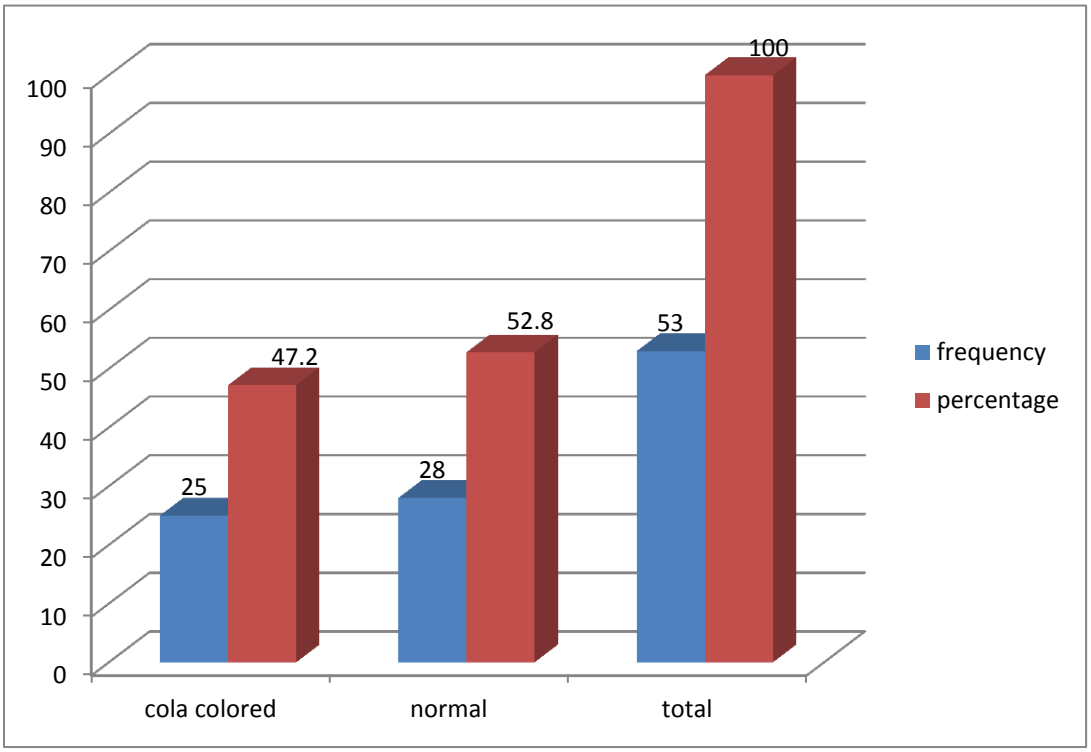


Fig.13

47.2% (25 patients) had dark colored or cola colored urine which is due to the presence of myoglobin in the urine

TACHYPNOEA – FREQUENCY DISTRIBUTION

Tachypnoea	Frequency	Percentage
Present	14	26.4
Absent	39	73.6
Total	53	100

Table.10

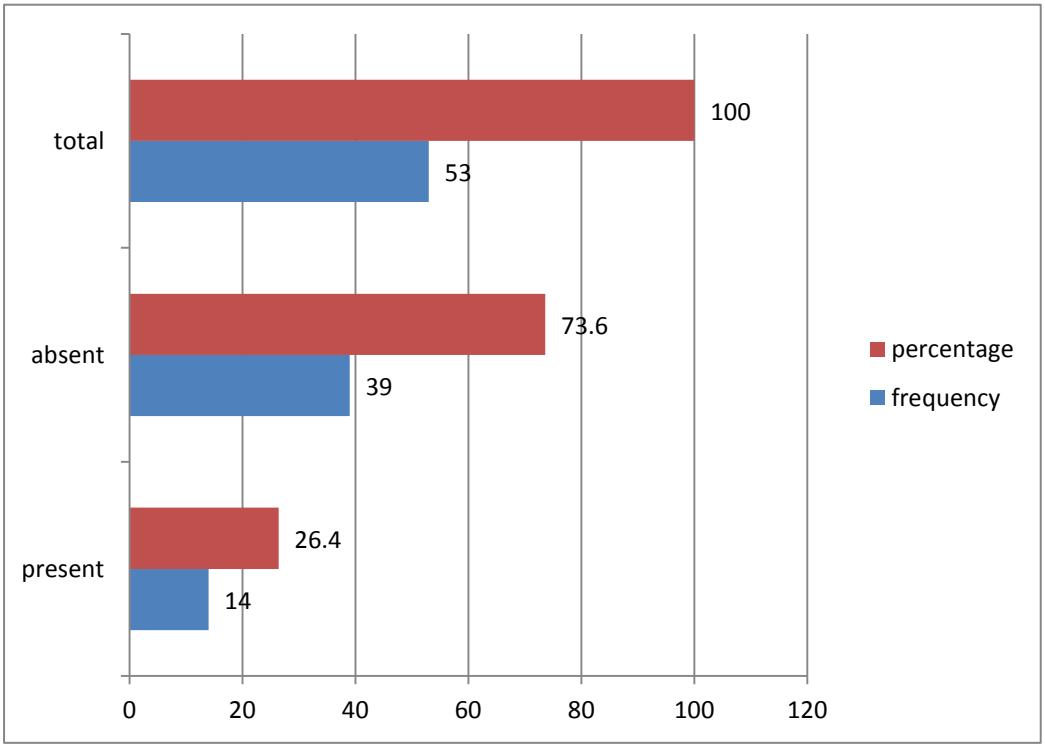


Fig.14

26.4% (14 patients) had tachypnoea and all these patients presented with dyspnoea.

CERVICOFACIAL EDEMA

Cervicofacial edema	Frequency	Percentage
Present	35	66.1
Absent	18	33.9
Total	53	100

Table.11

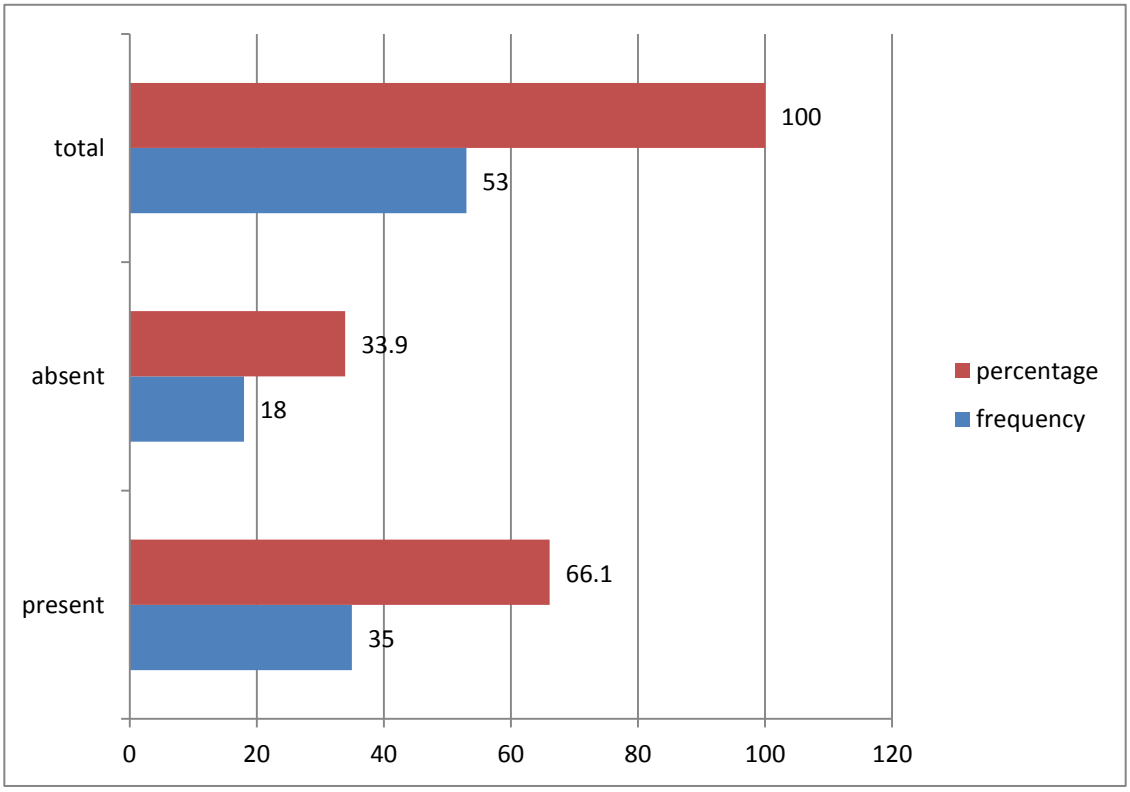


Fig.15

66.1% (35 patients) developed cervicofacial edema and this is the commonest presentation of the study population.

CERVICOFACIAL EDEMA – AGE RELATION

Age	Cervicofacial edema			
	Present		Absent	
	Count	Percentage	Count	Percentage
<30 years	21	51.4	20	48.8
>31 years	4	33.3	8	66.7

Table.12

CERVICOFACIAL EDEMA – AGE RELATIONSHIP

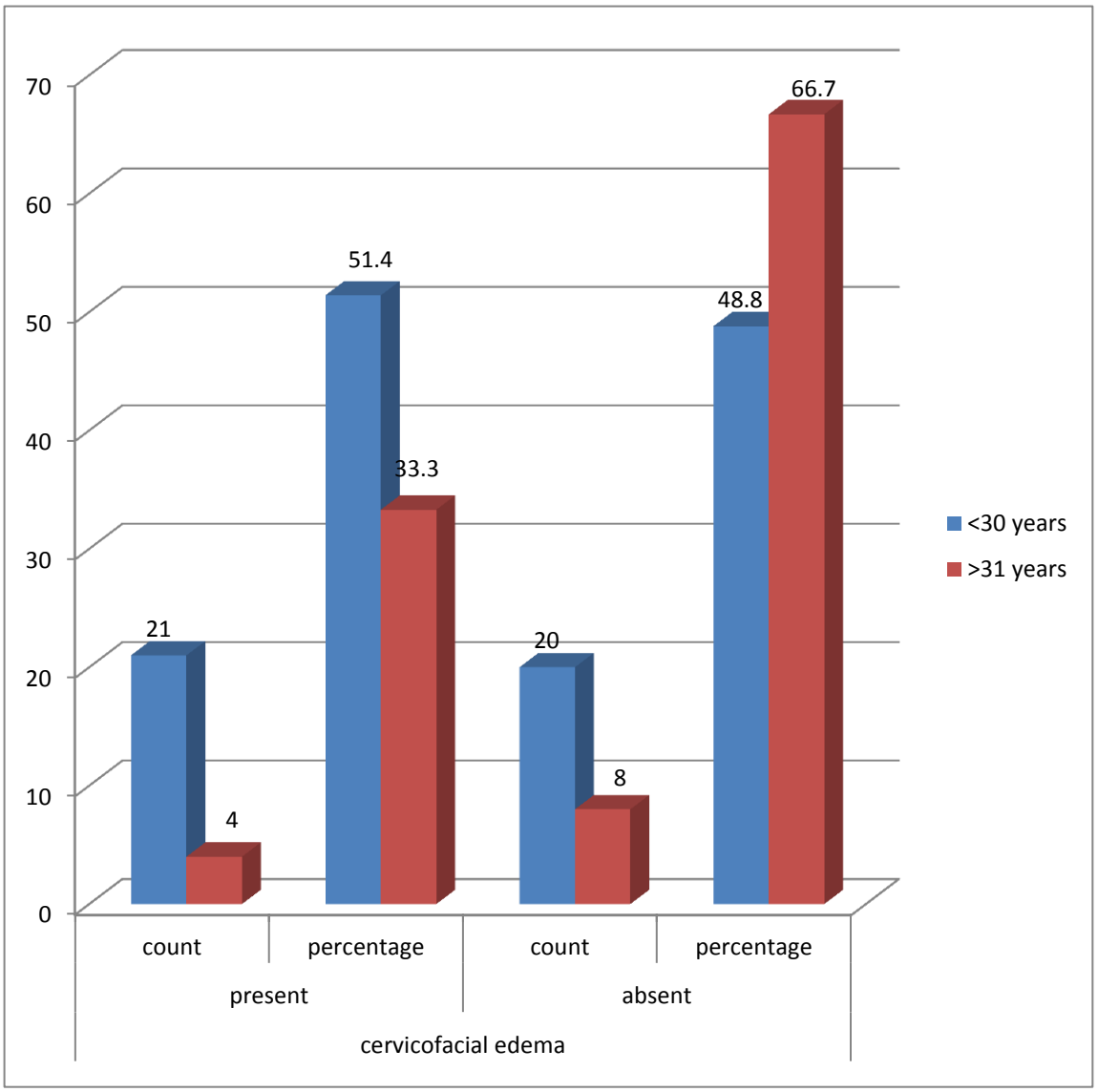


Fig.16

Out of the 25 patients who had cervicofacial edema 21 patients were under 30 years of age and 4 patients were above 30 years of age.

CERVICOFACIAL EDEMA – SEX DISTRIBUTION

Sex	Cervicofacial edema			
	Present		Absent	
	Count	Percentage	Count	Percentage
Males	4	22.2	14	77.8
Females	21	60	14	40

Table.13

CERVICOFACIAL EDEMA – SEX DISTRIBUTION

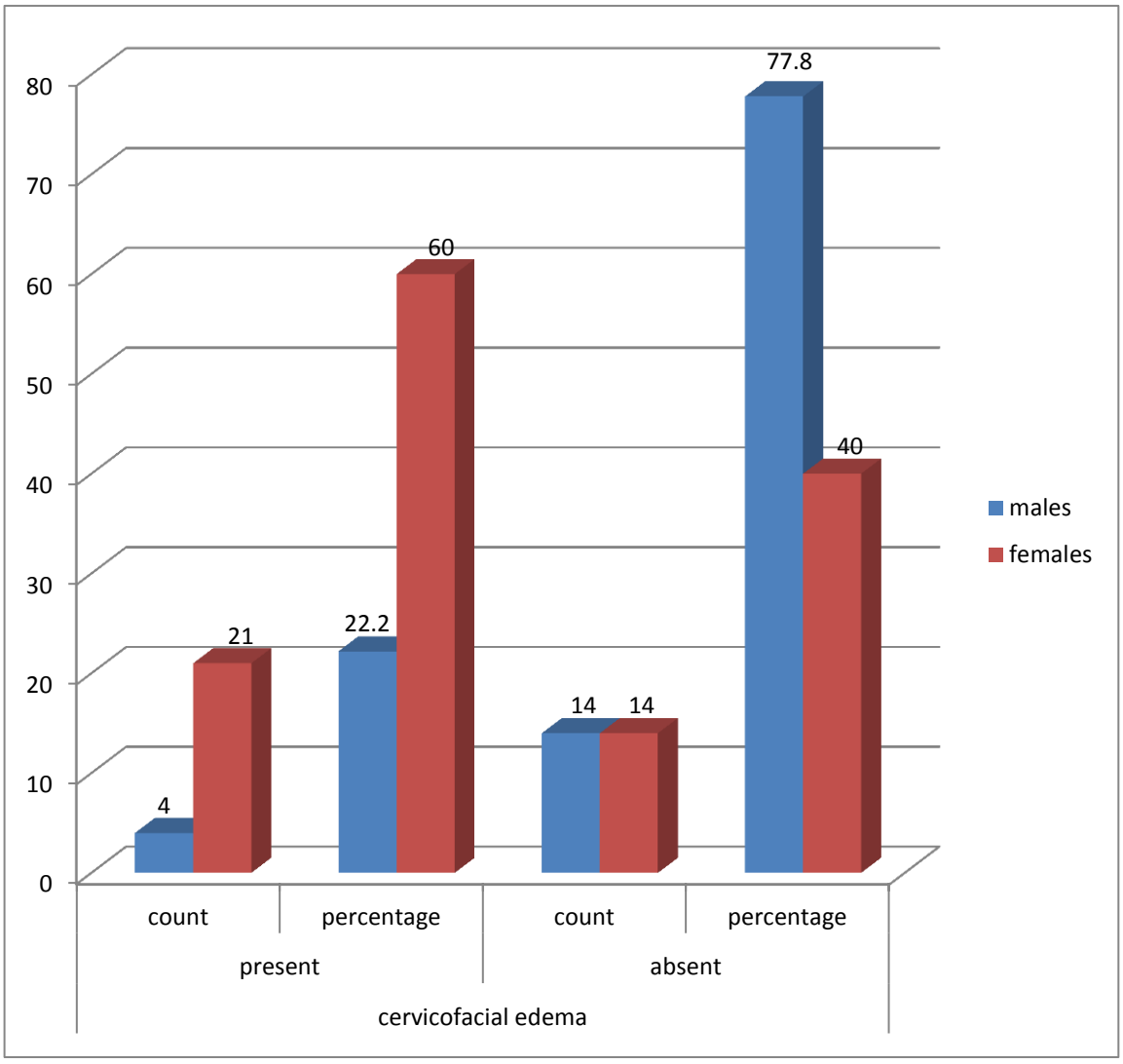


Fig.17

22.2% of the male patients had cervicofacial edema while 60% of the female patients had cervicofacial edema.

CALF TENDERNESS – FREQUENCY DISTRIBUTION

Calf tenderness	Frequency	Percentage
Present	19	35.8
Absent	34	64.2
Total	53	100

Table.14

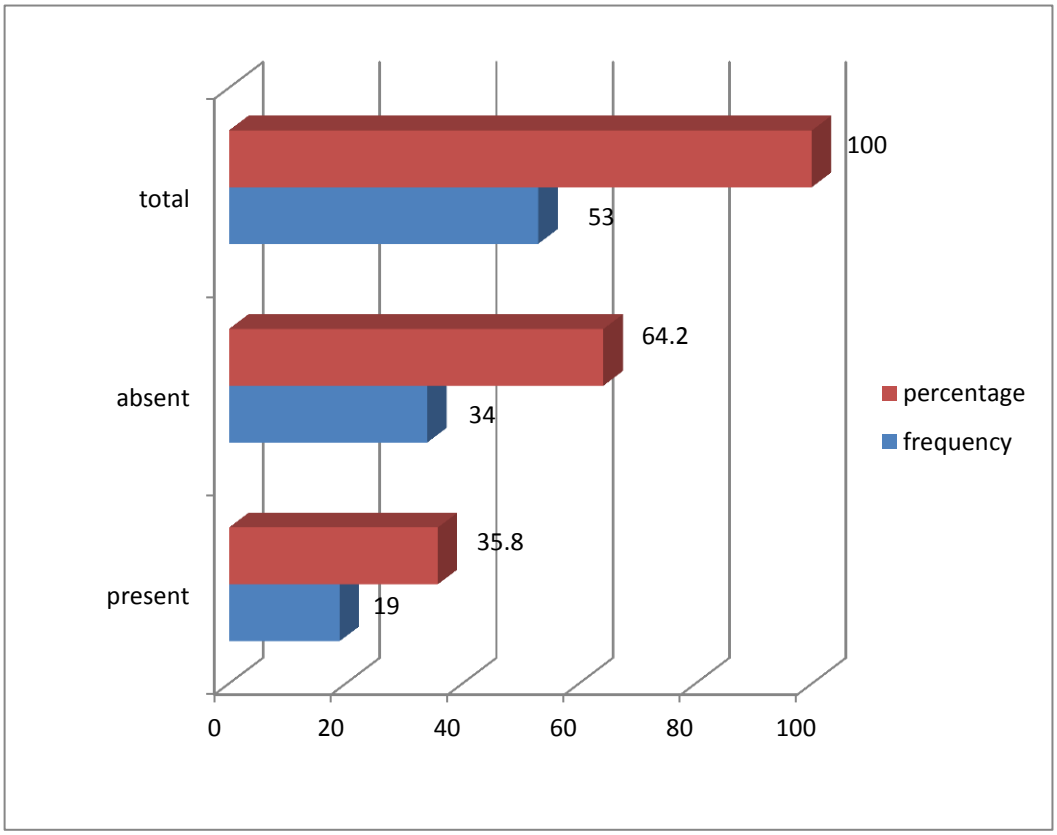


Fig.18

35.8% (19 patients) had calf tenderness on examination.

CALF TENDERNESS – AGE RELATIONSHIP

age	calf tenderness			
	present		absent	
	count	percentage	count	percentage
<30 years	14	34.1	27	65.9
>31 years	5	41.7	7	58.3

Table.15

CALF TENDERNESS – AGE DISTRIBUTION

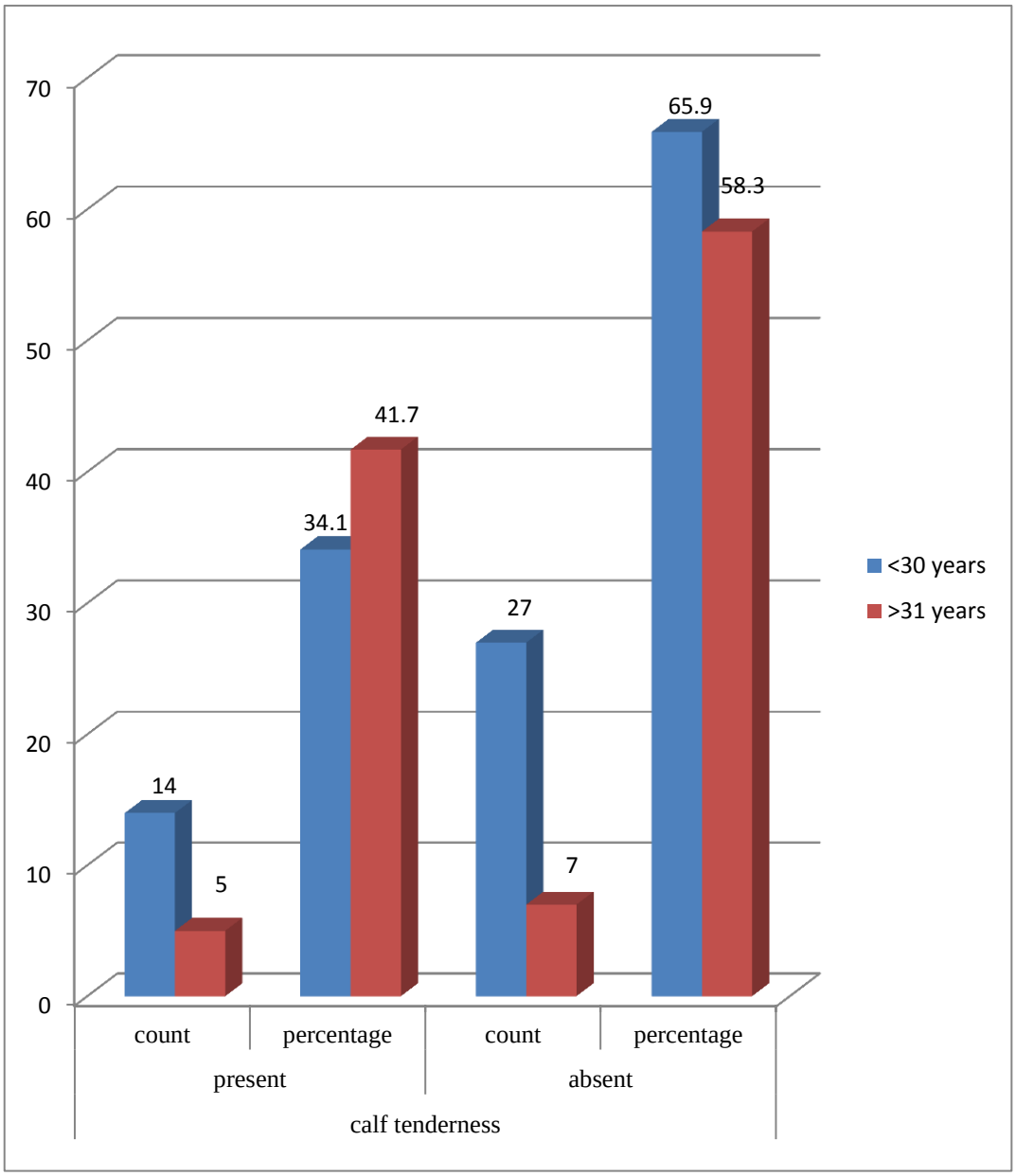


Fig.19

14 patients(34.1%) with calf tenderness were less than 30 years of age and 5 patients (41.7%) of patients above 31 years of age had calf tenderness.

CALF TENDERNESS – SEX DISTRIBUTION

Sex	Calf tenderness			
	Present		Absent	
	Count	Percentage	Count	Percentage
Male	7	38.9	11	61.6
Female	12	34.3	23	65.7

Table.16

Out of the total males admitted 38.9% (7 patients) developed calf tenderness and 34.3% (12 patients) females developed calf tenderness.

PULSE RATE – FREQUENCY DISTRIBUTION

pulse rate	frequency	percentage
tachycardia	14	26.4
normal	39	73.6
total	53	100

Table.17

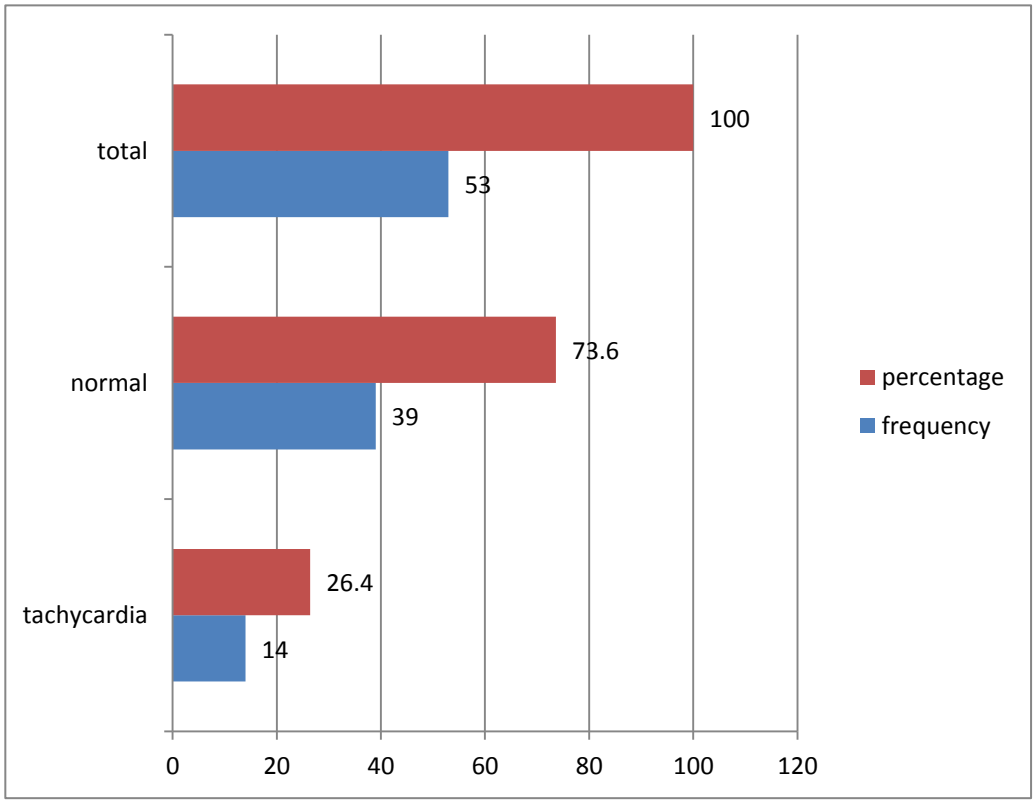


Fig.20

26.4% (14 patients) had tachycardia and the remaining 73.6% had normal heart rate.

BLOOD PRESSURE – FREQUENCY DISTRIBUTION

Blood pressure	Percentage
90/60 - 140/90	92.5
>140/90	5.5
<90/60	2

Table.18

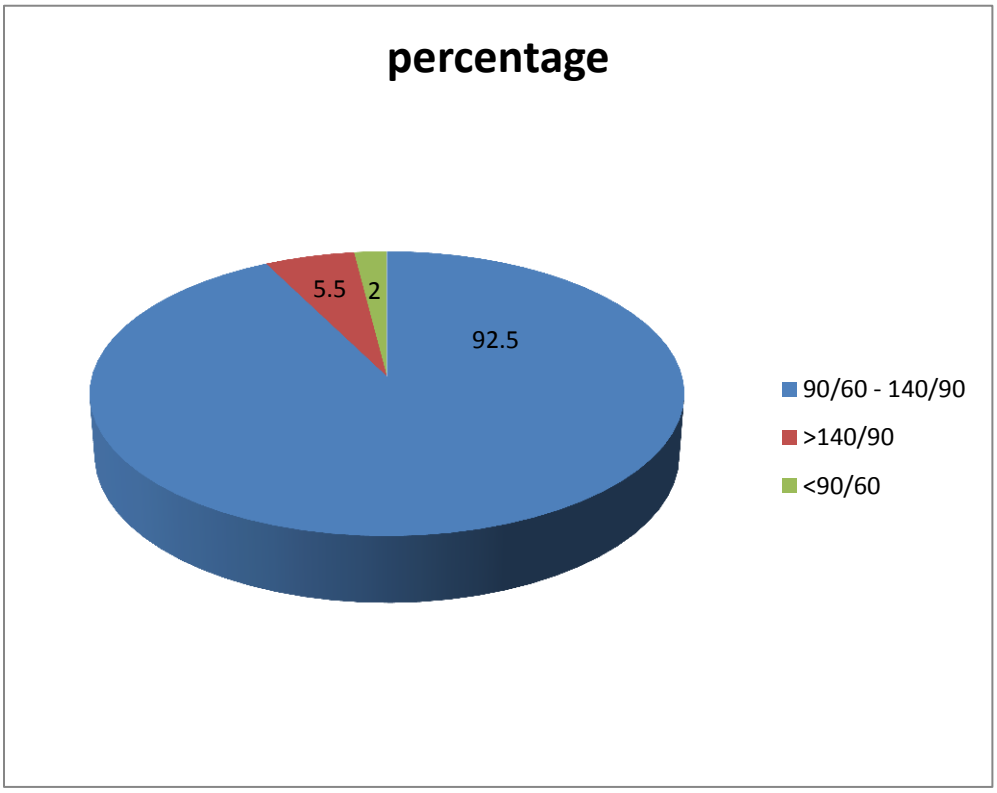


Fig.21

5.5% of the study population had BP >140/90 mm hg and 2% had a BP of < 90/60 mm hg on admission.

RESPIRATORY RATE – FREQUENCY DISTRIBUTION

Respiratory rate	Frequency	Percentage
<20/min	41	77.4
>20/min	12	22.6
Total	53	100

Table.19

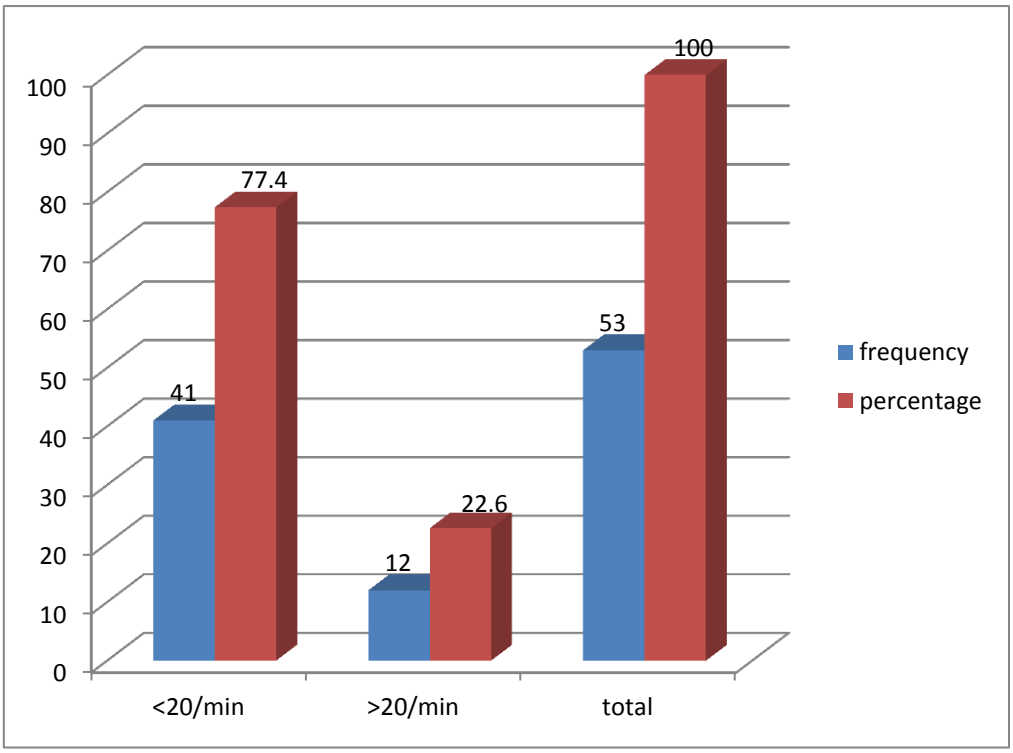


Fig.22

12 patients (22.6%) had tachypnoea at presentation.

OXYGEN SATURATION – FREQUENCY DISTRIBUTION

SpO_2	Frequency	Percenatge
> 95%	40	75.5
<95%	13	24.5
Total	53	100

Table.20

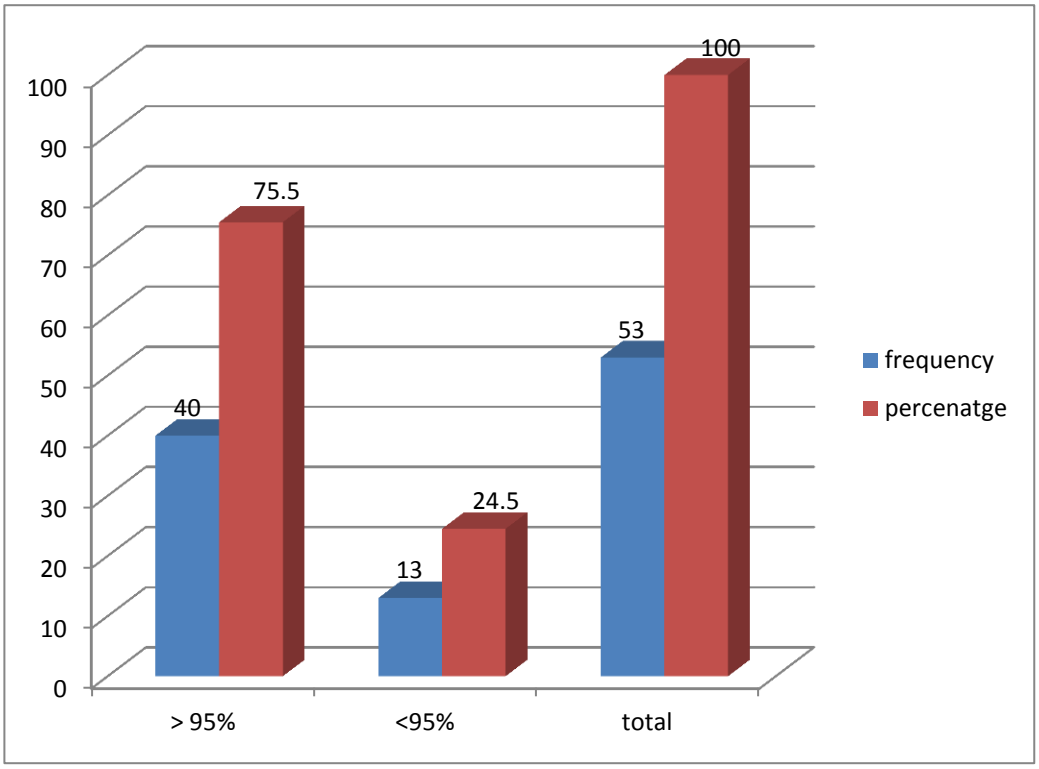


Fig.23

24.5% (13 patients) presented with desaturation which is due to the cervicofacial edema.

HEMOGLOBIN LEVELS – FREQUENCY DISTRIBUTION

Hemoglobin	Frequency	Percentage
<10 gm%	9	17
>11 gm%	44	83
Total	53	100

Table.21

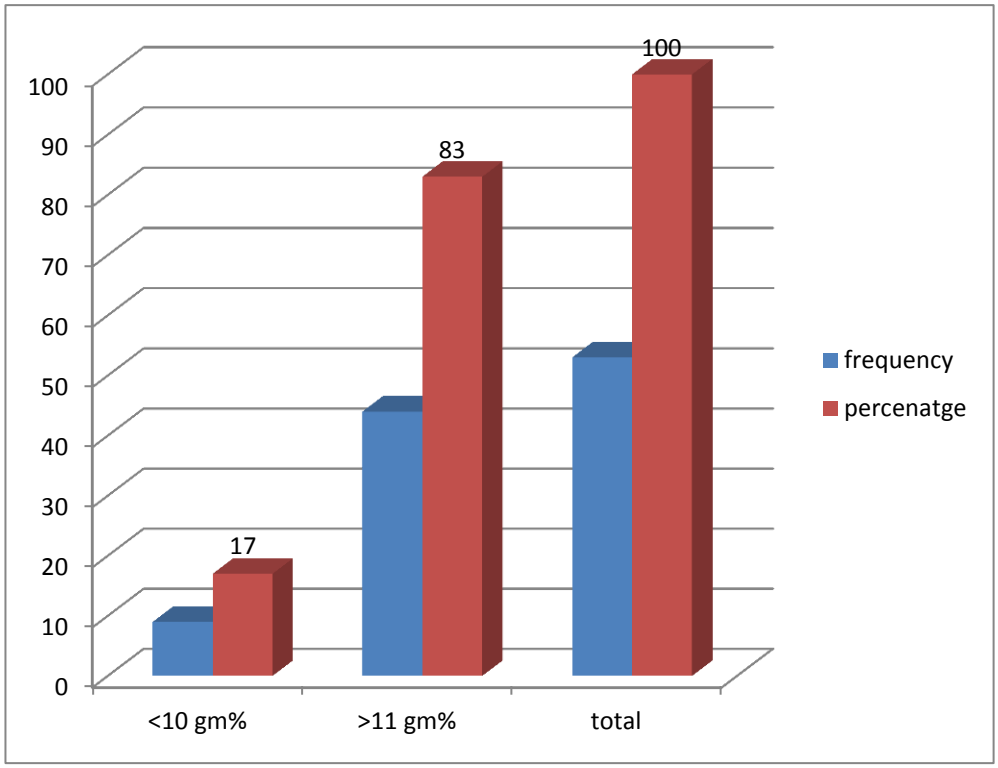


Fig.24

LEUCOCYTOSIS – FREQUENCY DISTRIBUTION

Total count	Frequency	Percentage
<11000	27	50.9
>11000	26	49.1
Total	53	100

Table.22

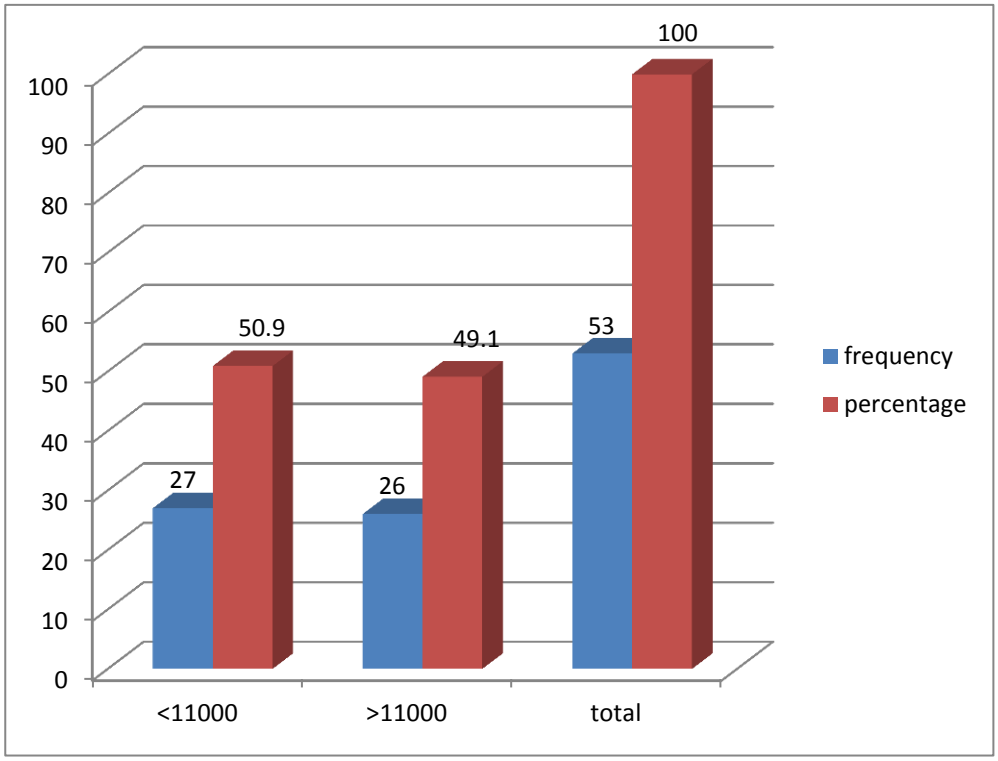


Fig.25

Leucocytosis was present in 49.1% (26 patients) of the study population.

NEUTROPHILIA – FREQUENCY DISTRIBUTION

Neutrophilia	Frequency	Percentage
Present	8	15.1
Absent	45	84.9
Total	53	100

Table.23

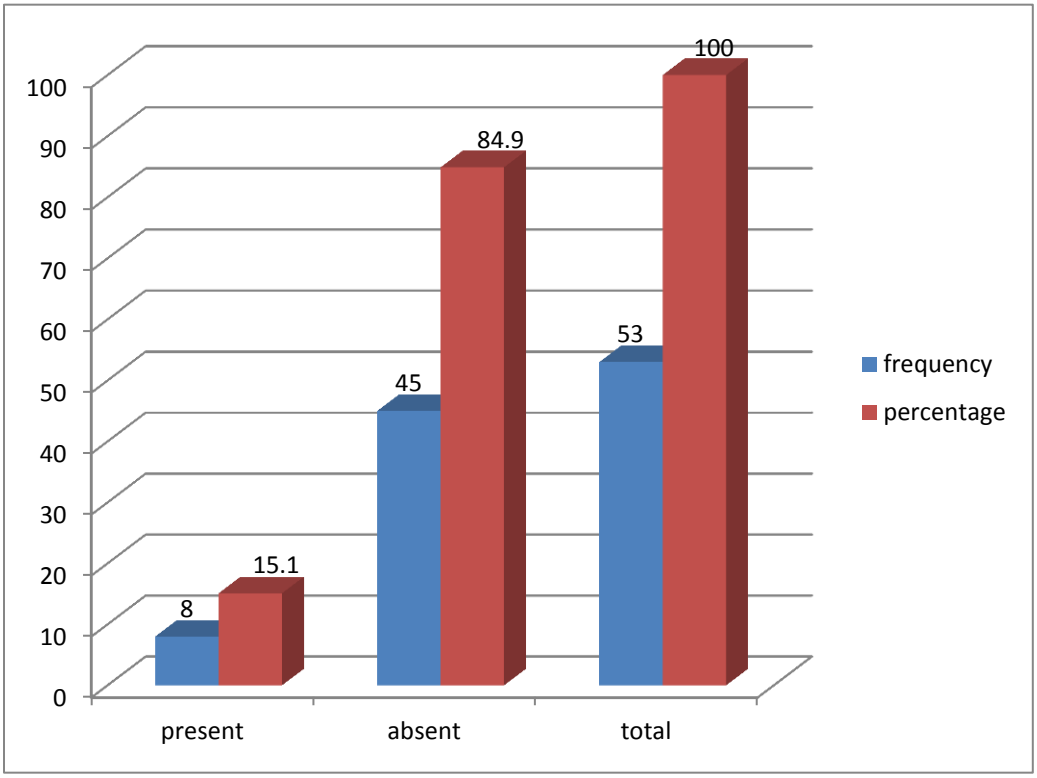


Fig.26

Neutrophilia was commonest abnormality in the differential count of the admitted cases with 15.1% (8 patients) having it.

LYMPHOCYTOSIS – FREQUENCY DISTRIBUTION

lymphocytosis	frequency	percentage
present	3	6
absent	50	94
total	53	100

Table.24

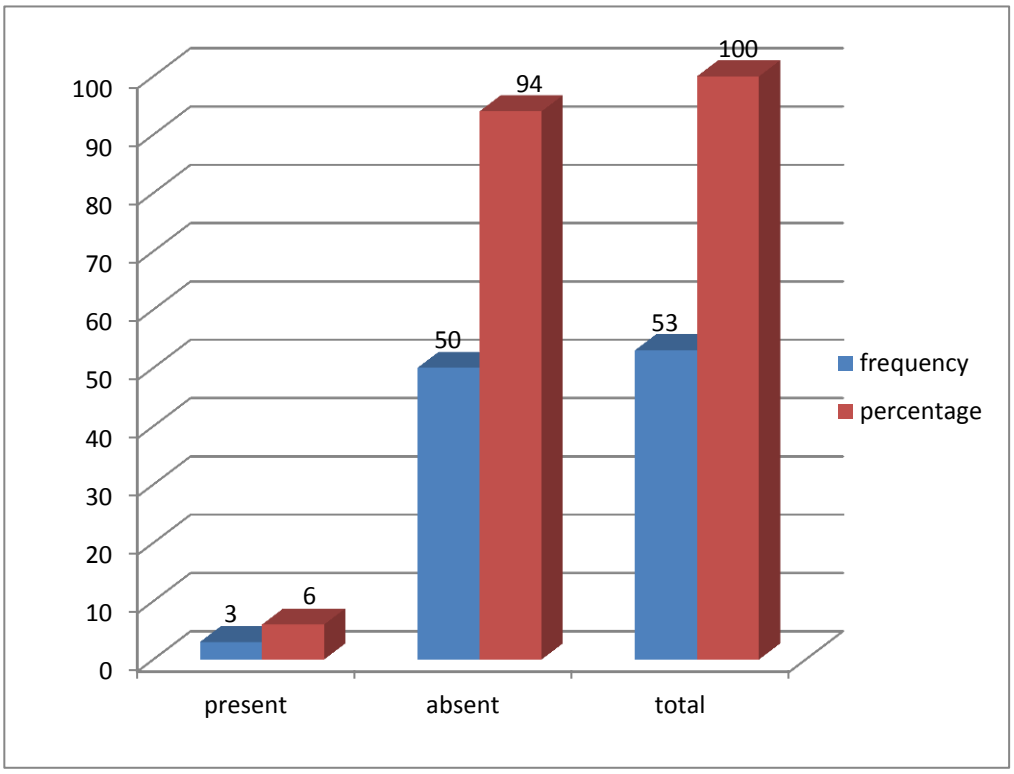


Fig.27

3 patients (6%) of the study population had lymphocytosis.

EOSINOPHILIA – FREQUENCY DISTRIBUTION

Eosinophilia	Frequency	Percentage
Present	1	1.6
Absent	52	98.4
Total	53	100

Table.25

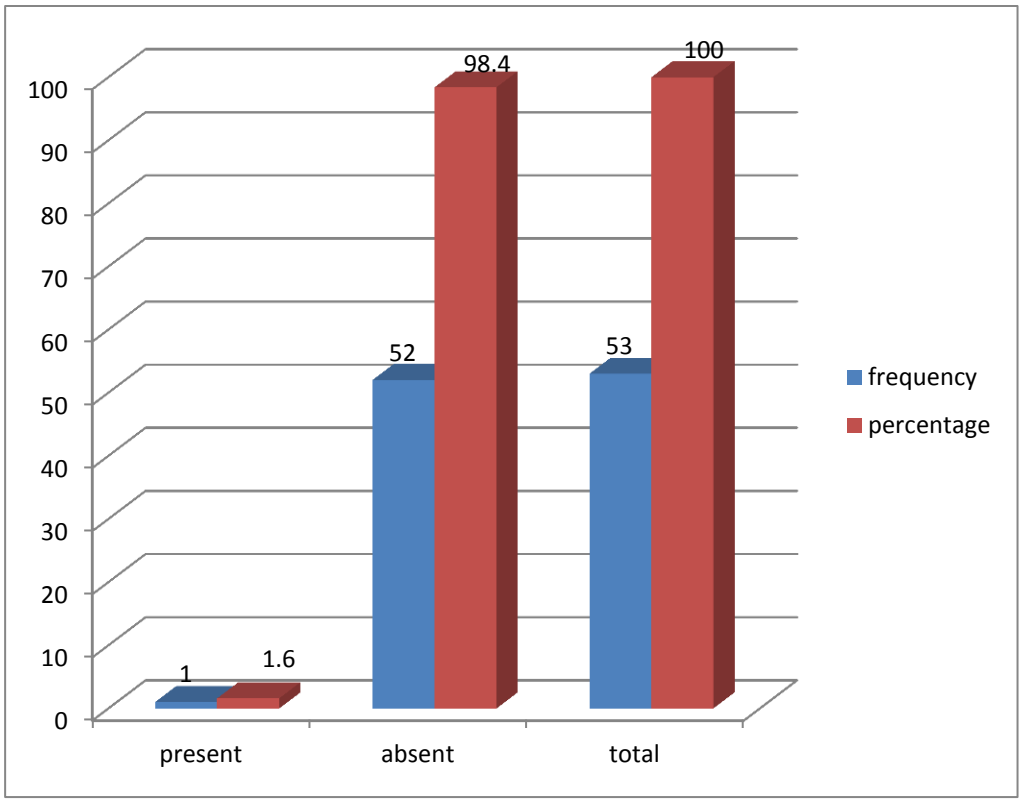


Fig.28

1.6% (1 patient) out of the total 53 patients admitted had eosiniphilia.

DIFFERENTIAL COUNT

Differential count	Percentage
Neutrophilia	15.1
Lymphocytosis	6
Eosinophilia	1.6

Table.26

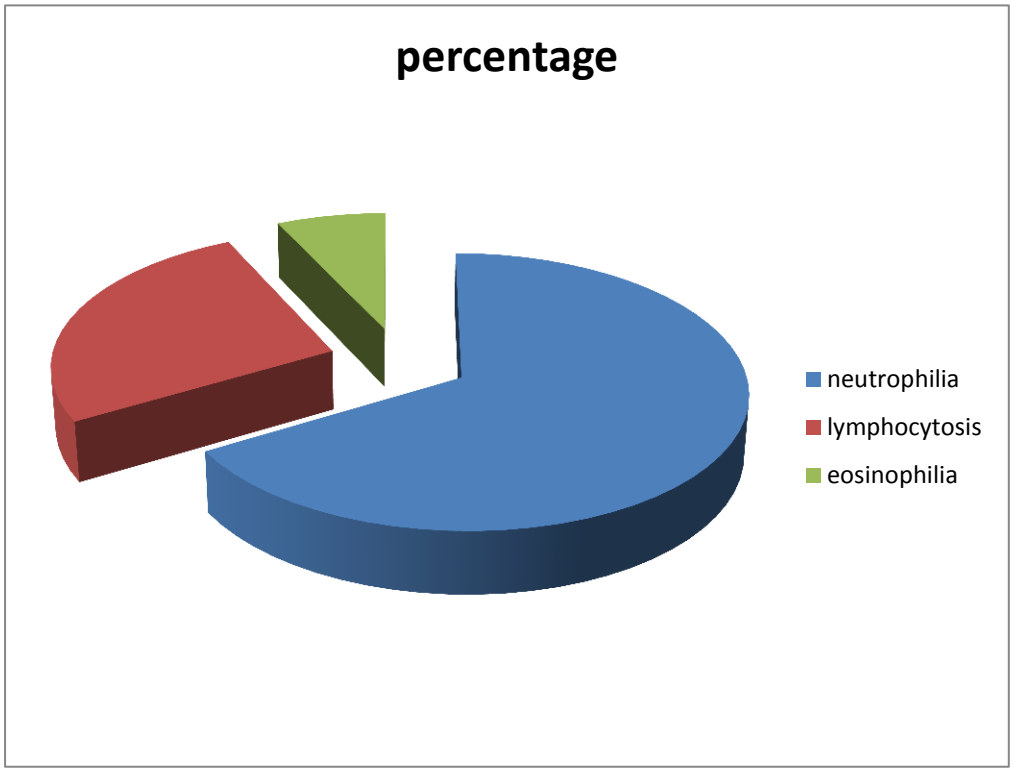


Fig.29

15.1% of the cases admitted had neutrophilia and 1.6% had eosinophilia. 6% had lymphocytosis

BLOOD UREA – FREQUENCY DISTRIBUTION

Blood urea	Frequency	Percentage
<40 mg%	39	73.6
>40 mg%	14	26.4
Total	53	100

Table.27

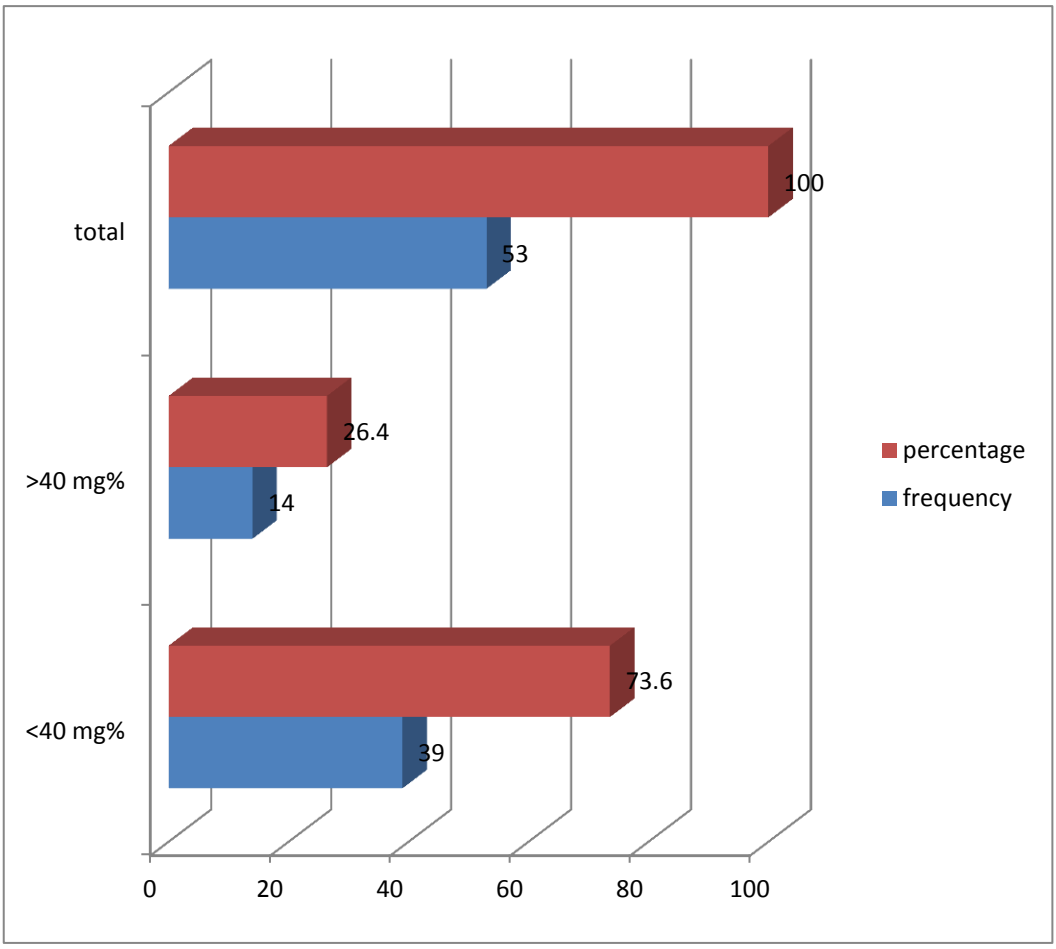


Fig.30

SERUM CREATININE – FREQUENCY DISTRIBUTION

Serum creatinine	Frequency	Percentage
<1.2	39	73.6
>1.3	14	26.4
Total	53	100

Table.28

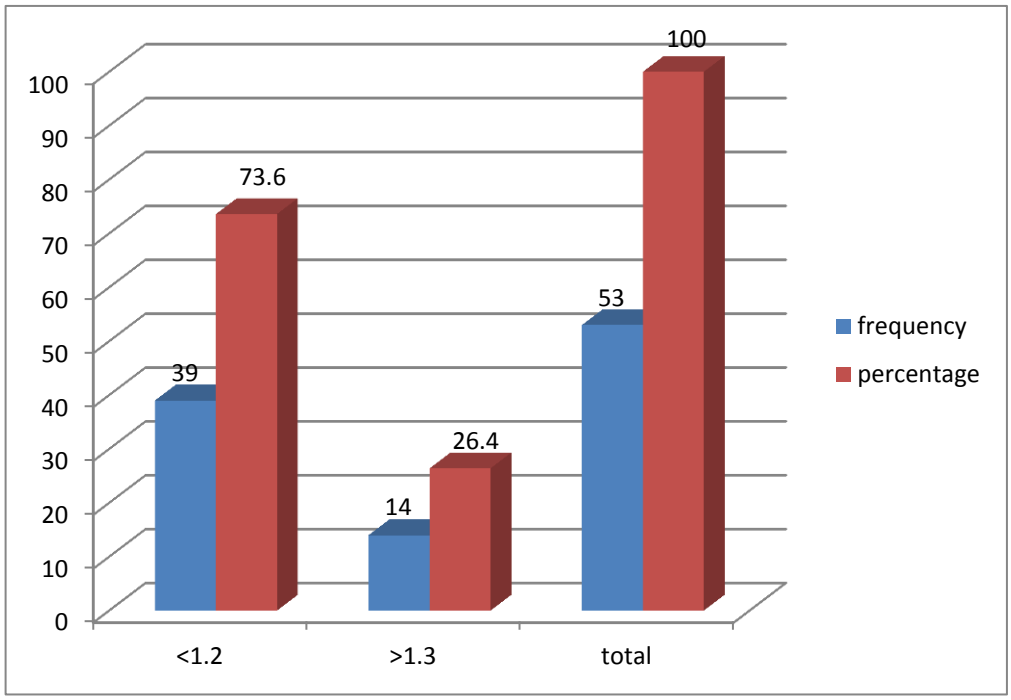


Fig.31

14 patients (26.4%) had elevated renal paramaters in the form of increased serum creatinine and blood urea.

SERUM SODIUM LEVELS – FREQUENCY DISTRIBUTION

serum sodium	frequency	percentage
normal	33	62.3
decreased	20	37.7
increased	0	0
total	53	100

Table.29

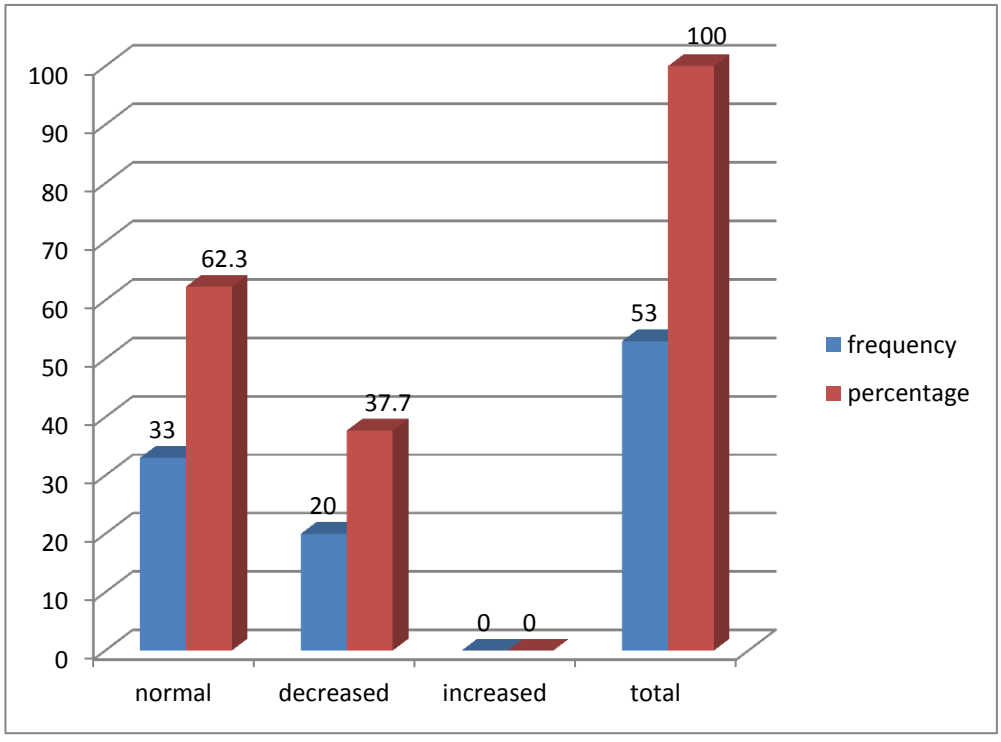


Fig.32

20 patients i.e., 37.7% of the study population had borderline hyponatremia.

SERUM POTASSIUM LEVELS – FREQUENCY DISTRIBUTION

Serum potassium	Frequency	Percentage
Normal	38	71.7
Hyperkalemia	15	28.3
Total	53	100

Table.30

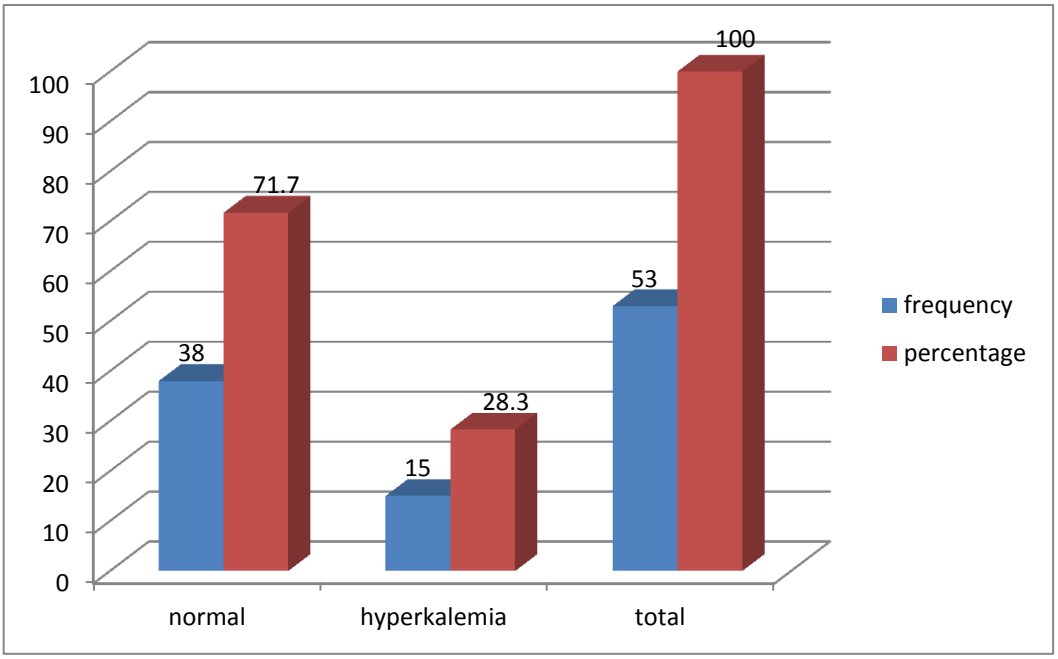


Fig.33

28.3% (15 patients) had hyperkalemia while other had serum potassium levels within normal limits.

HYPOCALCEMIA – FREQUENCY DISTRIBUITON

Hypocalcemia	Frequency	Percentage
Present	30	62.3
Absent	23	37.7
Total	53	100

Table.31

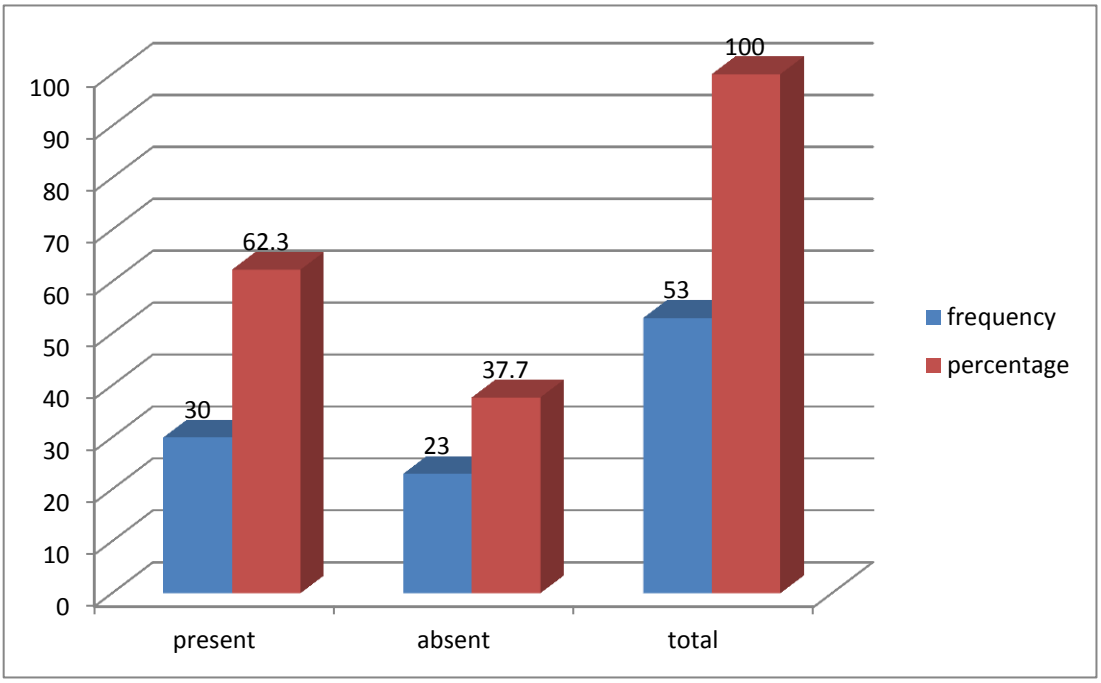


Fig.34

62.3% of the total patients admitted developed hypocalcemia which included 30 patients while the others had a normal calcium levels.

URINE DEPOSITS

Urine deposits	Frequency	Percentage
Negative	29	54.7
Positive	24	45.3

Table.32

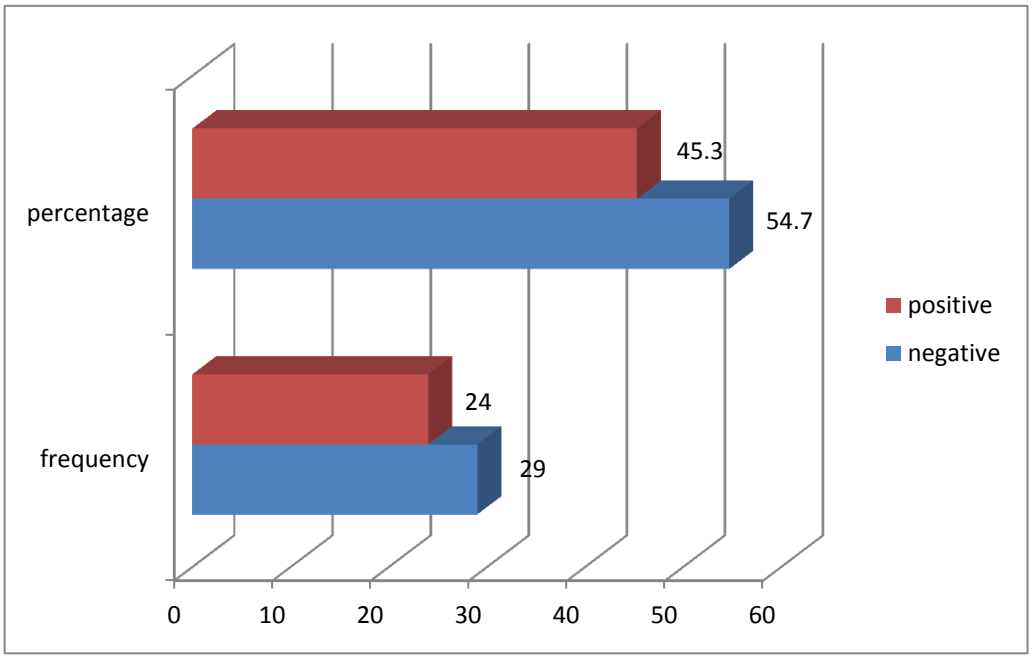


Fig.35

Urine deposits were positive in 45.3% (24 patients)of the study population.

ECG CHANGES

ECG	FREQUENCY	PERCENTAGE
Normal	36	67
Sinus tachycardia	13	24.5
Tall T V2-V3	4	7.5

Table.33

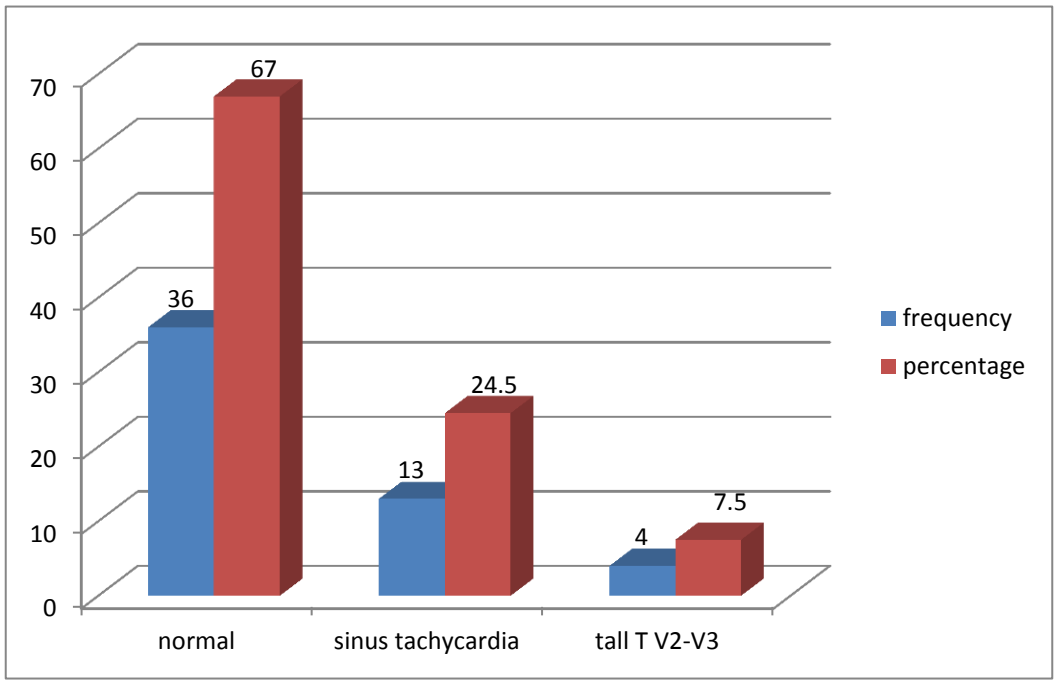


Fig.36

67% (36 patients) had a normal ECG, while the commonest ECG change was sinus tachycardia accounting 24.5% (13 patients) followed by T wave changes in 7.5% (4 patients).

TREATMENT

Treatment	Number	Percentage
Supportive	37	69.8
Tracheostomy	3	5.7
Hemodialysis	7	13.2
HD & tracheostomy	6	11.3

Table.34

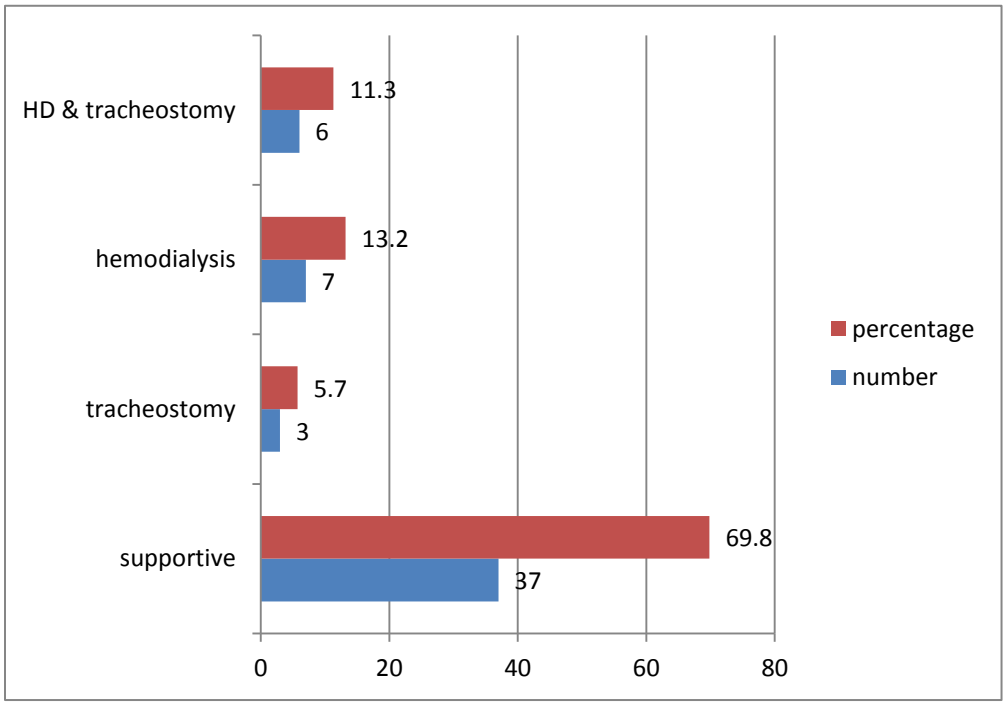


Fig.37

16 patients required renal replacement therapy and underwent emergency tracheostomy which equals to 30.2% and this includes both males and females.

TREATMENT - SEX CROSS TABULATION 1

Sex	Treatment			
	Supportive	Hd	Tracheostomy	Hd & tracheostomy
Male	16	0	0	2
Female	21	7	3	4

Table.35

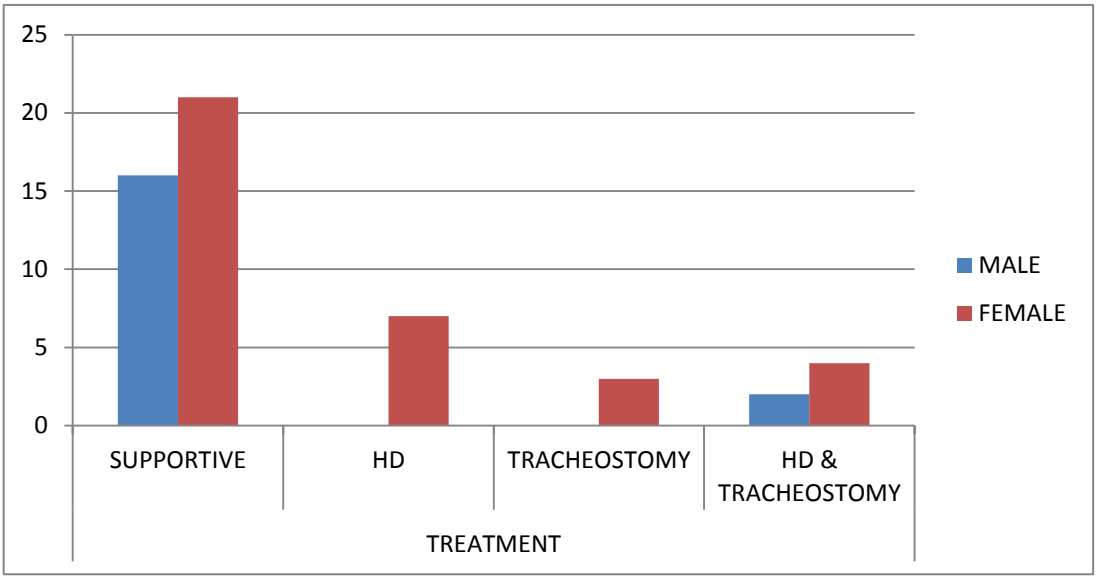


Fig.38

The requirement for HD and tracheostomy was more in case of females. 14 females developed complications while only 2 male patients developed complication.

TREATMENT – SEX CROSS TABULATION 2

SEX	TREATMENT - PERCENTAGE			
	SUPPORTIVE	HD	TRACHEOSTOMY	HD & TRACHEOSTOMY
MALE	88.9	0	0	11.1
FEMALE	60	20	8.6	11.4
TOTAL	69.8	13.2	5.7	11.3

Table.36

88.9% of the male patients required only supportive treatment and 11.1% required both hemodialysis and tracheostomy as ther developed both respiratory and renal complications.

60% of female patients required only supportive care. 20% needed hemodialysis and 8.6% needed tracheostomy. 11.4% of the females required both hemodialysis and tracheostomy.

TREATMENT – SEX CROSS TABULATION 2 CHART

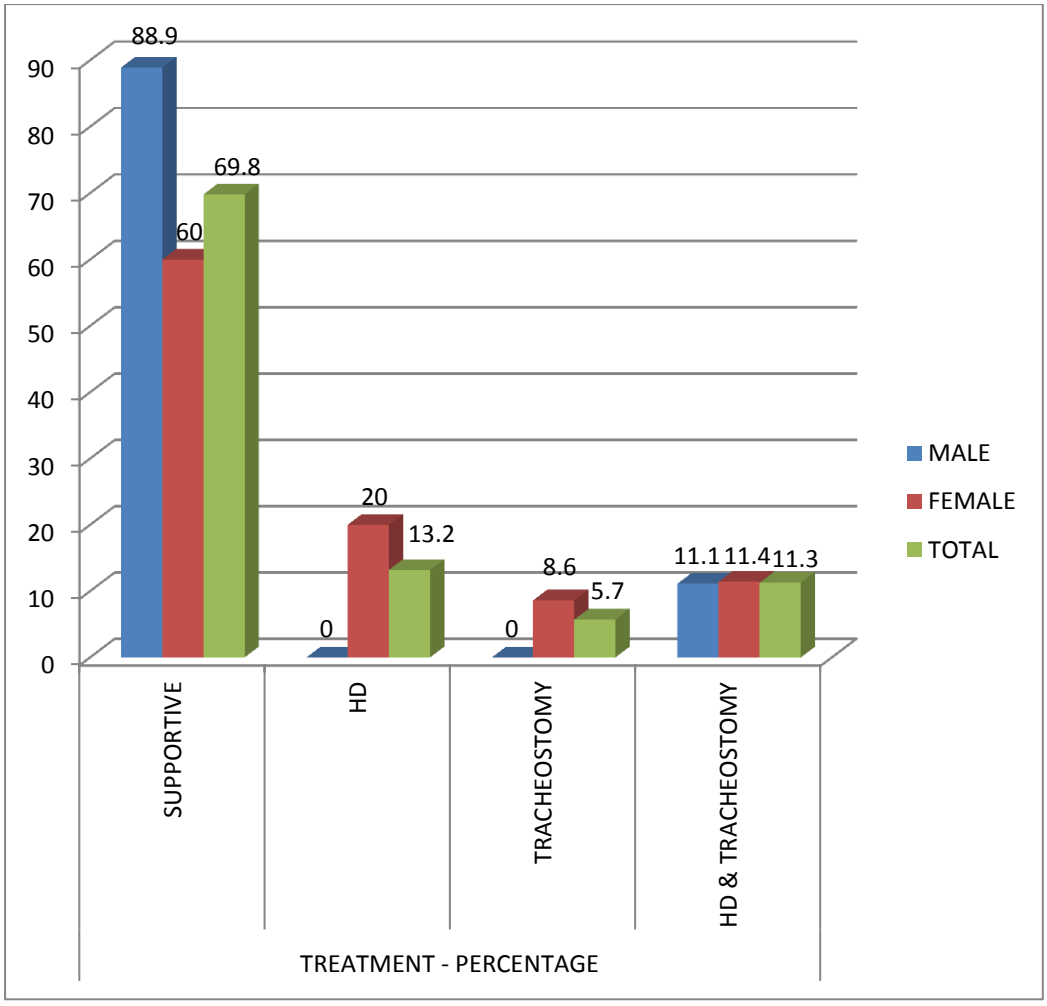


Table.39

About 69.8% of the total study population recovered with supportive care alone. 13.2% required with HD, 5.7% required tracheostomy and 11.3% needed both.

COMPLICATIONS

COMPLICATION	MALE	FEMALE	TOTAL
RESPIRATORY DISTRESS	2	7	9
AKI	2	11	13
MYOCARDITIS	0	0	0
HEPATITIS	0	0	0

Table.37

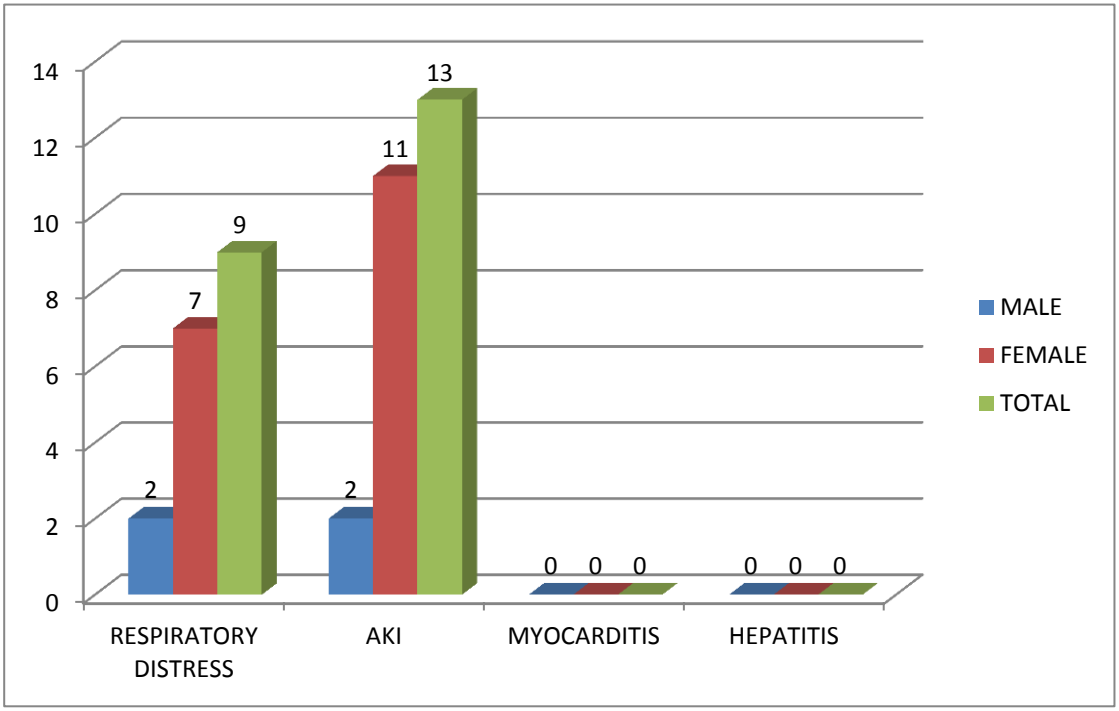


Fig.40

COMPLICATIONS – PERCENTAGE WITH SEX DISTRIBUTION

COMPLICATION	MALE	FEMALE	PERCENTAGE
RESPIRATORY DISTRESS	3.8	13.1	16.9
AKI	3.8	20.7	24.5
MYOCARDITIS	0	0	0
HEPATITIS	0	0	0

Table.38

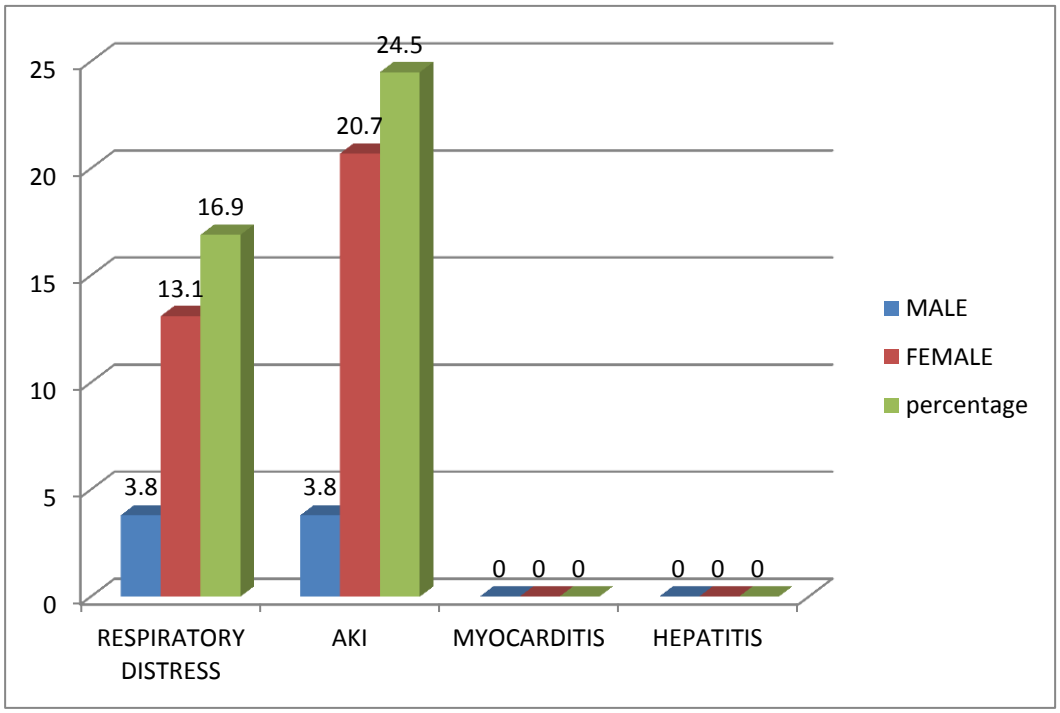


Fig.41

Out of the 53 patients admitted 9 patients (16.9%) developed respiratory distress and 13 patients (24.5%) developed acute kidney injury. 7.6% of them being males and 33.8% of them being females.

COMPLICATIONS – PERCENTAGE

COMPLICATIONS	PERCENTAGE
RESPIRATORY DISTRESS	16.9
ACUTE KIDNEY INJURY	24.5

Table.39

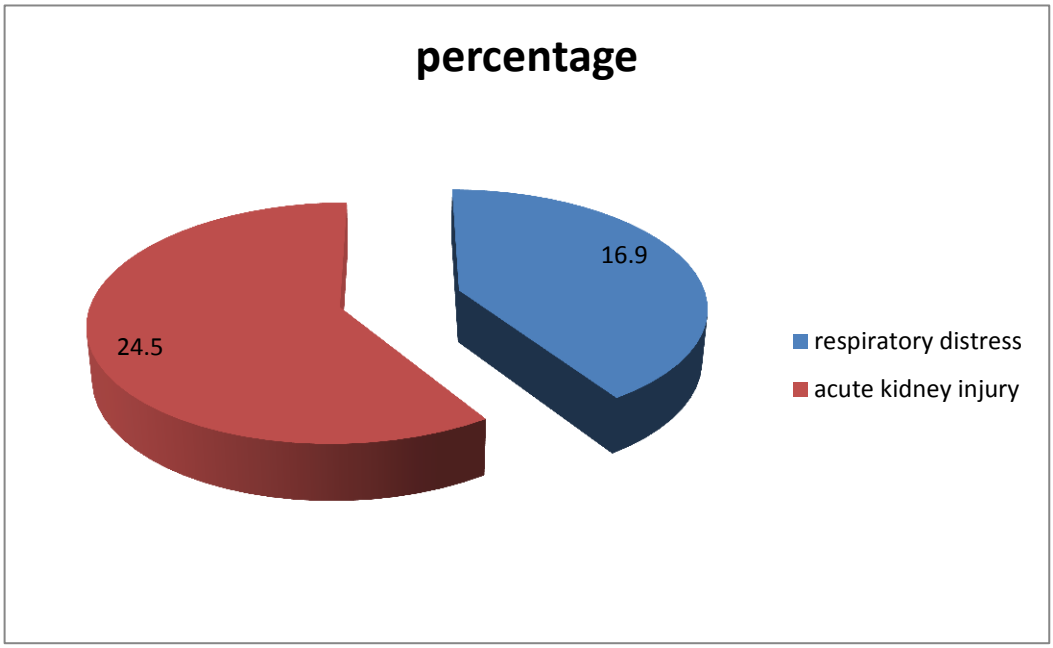


Fig.42

24.5% developed acute kidney injury and 16.9% developed respiratory distress. There was no development of complications like hepatits and myocarditis in our study population

OUTCOME

OUTCOME	NUMBER	PERCENTAGE
RECOVERED	47	88.7
EXPIRED	6	11.3

Table.40

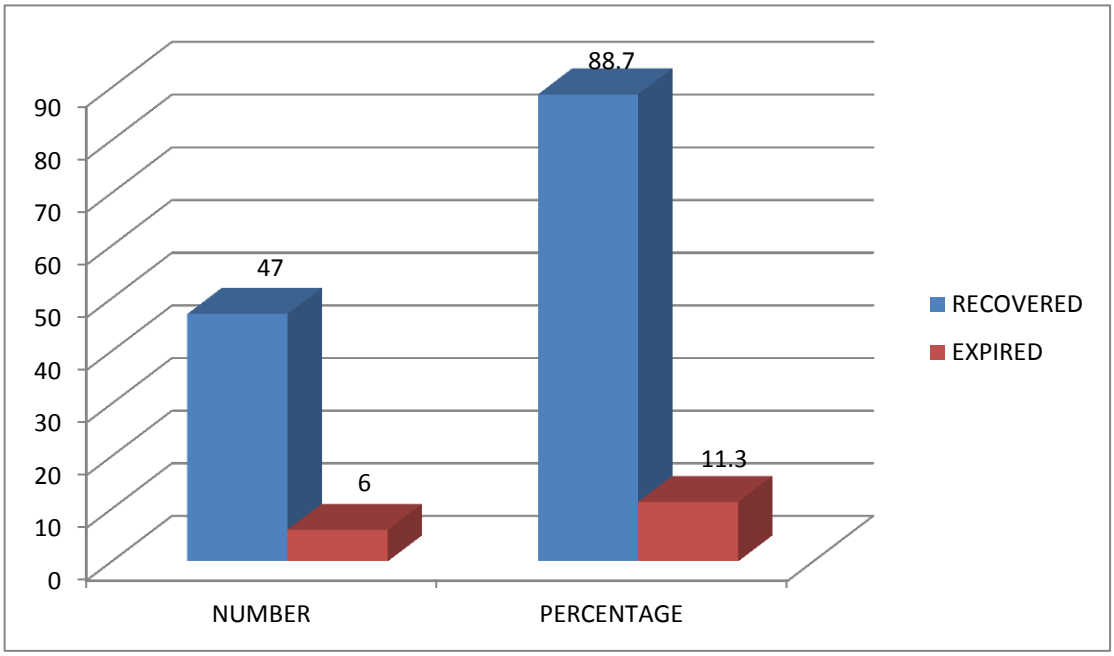


Fig.43

Out of the total 53 patients admitted 47 patients recovered completely and 6 patients expired.

OUTCOME – PERCENTAGE

OUTCOME	PERCENTAGE
RECOVERED	88.7
EXPIRED	11.3

Table.41

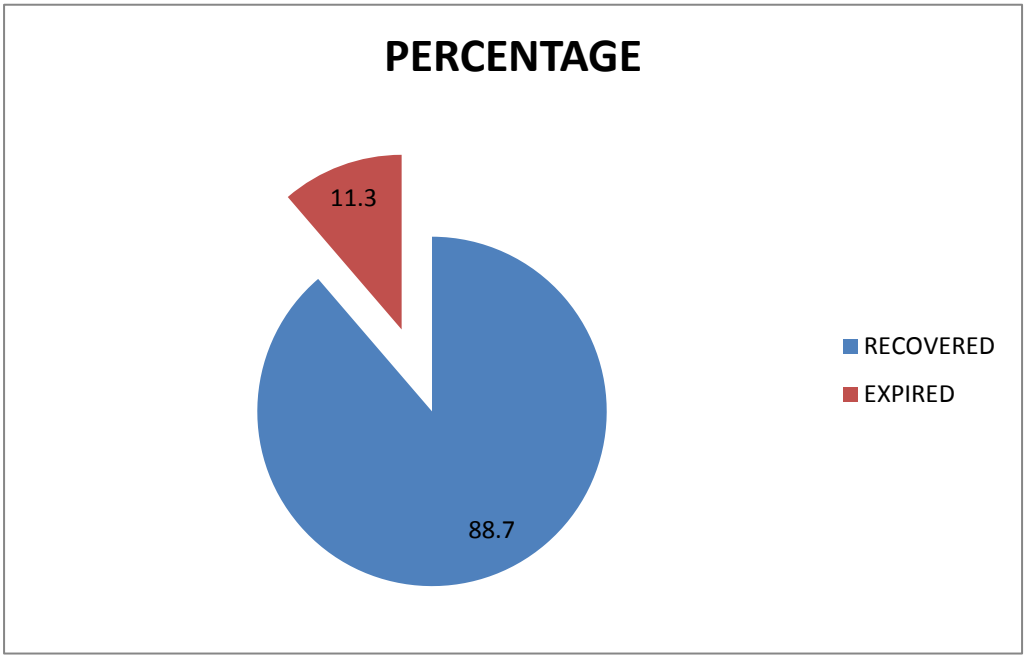


Fig.44

88.7% of the total study population recovered completely and the mortality rate was 11.3% in this study.

DISCUSSION

Suicidal poisoning in India is on the rise over the past two decades. Many commonly used substances are being added to the list of highly poisonous substances and that many of these poisons are difficult to treat. This is because no definite guidelines have been defined for the treatment of cases admitted with these poisons.

Hair dye poisoning has been increasing in incidence over the past 20 -30 years. One of the prime reasons for this is the easy availability of these hair dyes and the cheaper cost. This type of poisoning is common in the developing part of the world mainly the Indian subcontinent that includes Sri Lanka, Pakistan, Bhutan, Bangladesh and the eastern world to some extent but is rare in the western world.

The commonly used hair dye with suicidal intent is super vasmol. The active component of this being para-phenylene-dimine. The other constituents which have been studied to have toxic effects are resorcinol and propylene glycol. EDTA sodium which is also a ingredient in this hair dye causes hypocalcemia but at a higher doses. The fatal dose varies for each of the component. The most toxic compound of super vasmol is para-phenylene-diamine followed by resorcinol, propylene glycol and lastly EDTA sodium. The toxic effects of PPD is 3 grams and fatal dose of PPD is anything above 7 grams. The risk of myocarditis and sudden cardiac death is

more when the amount consumed is more than 10 grams. 50 ml of super vasmol contains 6 grams of PPD and so the mortality rate increases with consumption of more than 50 ml of the dye. The fatal dose of resorcinol is more than 30mcg/kg body weight.

Many studies and case reports have been carried out regarding the incidence, clinical presentation, complications and mortality following the consumption of super vasmol hair dye.

In this study 53 patients were studied prospectively who were admitted in the intensive medical care unit of our hospital. The study was mainly focused on the clinical profile, complications, treatment and outcome of these patients. Majority of the admitted patients were females accounting for 66% (35 patients) (table.1) with males being 34%. The age group of the study population was predominantly less than 30 years of age accounting for 77% (41 patients) (table.2) which is similar to the study done by kallel et al where the mean age was 26.2 +/- 9.1 years and a eleven year 1992 to 2002 retrospective study from Morocco described 374 cases of PPD poisoning, majority of patients (54%) were 15-24years age group and children contributed 11.5%. 50.9% of the patients consumed a poison of more than 50 ml. A 10 year prospective study including 3159 patients as done and it was reported a para-phenylene-diamine poisoning in children less than 14

years accounting to 18% of the total study population. Tthe common presenting features were dysphagia (67.9%), myalgia (35.8%) and dyspnoea (26.4%). On examination 66.1% of the patients had cervicofacial edema, 26.4% were tachypnoeic and 35.8% of the patients had calf muscle tenderness. 47.2% (25 patients) had cola colored urine. 49.1% (26 patients) had leucocytosis. The complications these patients developed are acute respiratory distress (16.9%) and acute kidney injury (24.5%). 62.3% (30 patients) developed hypocalcemia and 28.3% (15 patients) developed hyperkalemia. Most of the patients had normal ECG. 24.5% (13 patients) had sinus tachycardia followed by T wave abnormalities in 7.5%.2 males developed respiratory distress and tracheostomy was done. 2 (3.8%) other patients (3.8%) developed acute kidney injury and hemodialysis was done. In the female study population7 patients (13.1%) developed respiratory distress and tracheostomy was done and 11 (20.7%) developed acute kidney injury and hemodialysis was done. None of the patients developed hepatitis or myocarditis as they had normal liver function tests and normal ECG throughout the stay. 88.7% (47 patients) recovered completely with supportive treatment, tracheostomy and hemodialysis while 6 patients expired (11.3%). Out of this, patients 5 patients (9.4%) had both respiratory distress and acute kidney injury while the other patient (1.8%) had

respiratory distress and this patient expired on table while tracheostomy was being done.

All the patients admitted were treated with supportive measures mainly gastric lavage, supplemental oxygen, intravenous fluids, intravenous corticosteroids mainly hydrocortisone, intravenous anti-histaminics and antibiotics. 69.8% of the patients including both males and females recovered with supportive treatment alone. All the patients who developed myalgia, muscle tenderness, elevated muscle enzymes and cola colored urine were treated with forced alkaline diuresis to prevent to progression to acute tubular necrosis secondary to myoglobinuria. 13.2% of the patients required hemodialysis, 5.7% needed tracheostomy and 11.3% were subjected to both hemodialysis and tracheostomy. The mortality rate in the study population was 11.3% which was because of late presentation, large volume of ingestion and poor neurological status on presentation.

CONCLUSION

Hair dye poisoning in India is a rapidly emerging type of suicidal poisoning. The incidence has increased over the past few years. Super vasmol poisoning is a dye which is commonly used due to its easy availability. The common presenting symptoms are cervicofacial edema, and muscle tenderness. The common complications are respiratory distress, acute kidney injury, myocarditis and hepatitis. In our study we did not come across any patient with cardiac and hepatic involvement. Early treatment with activated charcoal, supportive measures and forced alkaline diuresis reduces the development of complications like acute kidney injury. So, early intervention reduced the mortality rate in patients with super vasmol poisoning.

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*Department of MEDICINE,Christian Medical College, Vellore 632004, Tamil Nadu, India

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ANNEXURE I:

PROFORMA:

NAME:

AGE / SEX: **IP.NO:**

VOLUME CONSUMED:

CLINICAL SYMPTOMS: DYSPHAGIA / DYSPNOEA / MYALGIA

GENERAL EXAMINATION:

CONSCIOUSNESS / ORIENTATION / TACHYPNOEA

CERVICOFACIAL EDEMA : YES / NO

MUSCLE TENDERNESS: YES / NO

VITALS:

PULSE RATE:

BLOOD PRESSURE:

RESPIRATORY RATE:

BLOOD OXYGEN SATURATION:

SYSTEM EXAMINATION:

CVS:

RS:

ABDOMEN:

CNS:

URINE COLOR:

INVESTIGATIONS:

TOTAL COUNT: DIFFERENTIAL COUNT:

HEMOGLOBIN:

RENAL FUNCTION TESTS:

SREUM ELECTROLYTES:

PLASMA CALCIUM LEVELS:

LIVER FUNCTION TESTS:

TOTAL CPK:

URINE DEPOSITS:

ECG:

TREATMENT GIVEN:

OUTCOME:

ANNEXURE II

MASTERCHART

S.NO	NAME	AGE/SEX	VOLUME CONSUMED (ml)	DYSPHAGIA	DYSPNOEA	MYALGIA	GENERAL EXAMINATION	PULSE RATE (BEATS/MIN)	BLOOD PRESSSURE	RESP. RATE	SpO2	CERVICOFACIAL EDEMA	CALF TENDERNESS	CVS	RS	ABD	CNS	URINE COLOUR	TOTAL COUNT (cells/cu.mm)	DIFFERENTIAL COUNT (%)	Hb(gm%)	BLOOD UREA / Sr.CREATININE	Sr.SODIUM	Sr.POTASSIUM	Sr.CALCIUM (mg%)	LIVER FUNCTION TESTS	TOTAL CPK (IU/ml)	ECG	URINE DEPOSITS	TREATMENT	OUTCOME
1	MAHESHW	24/F	<50	yes	no	no	conscious / oriente	86	120/70	20/min	97%	no	no	normal	normal	normal	normal	normal	11200	69/31	11.2	24/1.0	137	4.5	9.6	normal	normal	normal	nil	supportive	recovered
2	MADATHY	27/F	<50	yes	no	yes	conscious / oriente	90	130/80	18/min	97%	no	yes	normal	normal	normal	normal	normal	11900	71/29	12.4	31/0.9	138	4.2	9	normal	normal	normal	nil	supportive	recovered
3	MAHESH	19/M	<50	yes	no	yes	conscious / oriente	76	120/70	16/min	97%	no	yes	normal	normal	normal	normal	normal	12700	68/32	10.8	26/0.8	132	4.9	8.9	normal	normal	normal	nil	supportive	recovered
4	ANANTHI	30/F	>50	yes	yes	yes	[illegible]	110	140/80	32/min	86%	yes	yes	normal	normal	normal	normal	dark colour	15100	73/27	12.6	86/3.4	131	5.6	7.4	normal	3076	[illegible]	[illegible]	hemodialysis	recovered
5	MAHESHW	35/F	<50	no	no	no	conscious / oriente	72	110/60	18/min	97%	no	no	normal	normal	normal	normal	normal	13100	69/21/10	10.7	26/0.8	136	5.2	8.9	normal	normal	normal	nil	supportive	recovered
6	VANITHA	28/F	>50	yes	yes	yes	[illegible]	106	140/80	36/min	90%	yes	yes	normal	normal	normal	normal	dark colour	17200	62/36/2	9.8	90/4.6	141	5.9	8.4	normal	8790	[illegible]	[illegible]	hemodialysis	recovered
7	MARIAMM	37/F	>50	yes	no	yes	conscious / oriente	90	110/60	16/min	97%	yes	yes	normal	normal	normal	normal	dark colour	16900	64/33/3	11	105/4.5	140	4.9	8.2	normal	6700	normal	[illegible]	hemodialysis	recovered
8	ASHOK	32/M	<50	no	no	no	conscious / oriente	84	120/80	14/min	97%	no	no	normal	normal	normal	normal	normal	10000	61/37/2	13.4	29/0.7	136	4.9	9	normal	normal	normal	nil	supportive	recovered
9	KUMAR	30/M	<50	no	no	yes	conscious / oriente	72	110/70	18/min	97%	no	yes	normal	normal	normal	normal	normal	9500	63/37	14	24/0.9	138	4.1	8.9	normal	normal	normal	nil	supportive	recovered
10	MAHARAS	19/F	>50	yes	yes	no	[illegible]	108	150/80	32/min	85%	yes	no	normal	normal	normal	normal	dark colour	14400	78/37	10.6	74/4.5	138	5.6	7.9	normal	8755	[illegible]	[illegible]	hemodialysis	recovered
11	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	conscious / oriente	78	110/60	16/min	97%	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	19000	62/35/3	[illegible]	34/0.9	141	5.9	[illegible]	[illegible]	6250	normal	[illegible]	[illegible]	[illegible]

13	11	KAPAGAY	13/F	>50	yes	no	no	[illegible]	78	110/60	16/min	97%	yes	no	normal	normal	normal	normal	[illegible]	19000	62/35/3	11.2	31/0.9	141	5.3	8.4	normal	6750	normal	[illegible]	[illegible]	recovered
14	12	MUTHARA	44/F	<50	no	no	no	[illegible]	80	120/70	16/min	97%	no	no	normal	normal	normal	normal	normal	11500	64/32/4	11.6	26/0.8	132	4.2	9.2	normal	normal	normal	nil	[illegible]	recovered
15	13	SUDALAI	50/M	<50	no	no	no	[illegible]	90	100/70	14/min	97%	no	no	normal	normal	normal	normal	normal	8000	65/34	12.9	19/0.9	130	4.4	9.2	normal	normal	normal	nil	[illegible]	recovered
16	14	IYAPPAN	52/M	>50	yes	yes	yes	[illegible]	102	130/80	36/min	70%	yes	yes	normal	normal	normal	normal	[illegible]	17200	66/33/1	9.6	83/3.6	140	4	7.3	normal	10005	[illegible]	nil	TRACHEOS	expired
17	15	PALKANI	27/F	>50	yes	yes	yes	[illegible]	112	150/80	34/min	76%	yes	yes	normal	normal	normal	normal	[illegible]	16500	72/20	10.6	56/4.4	141	6.1	8	normal	3070	II T V2 V3	[illegible]	TRACHEOS	expired
18	16	PODLIN	17/M	<50	yes	no	no	[illegible]	76	120/80	10/min	97%	no	no	normal	normal	normal	normal	normal	9000	70/27/3	11	19/0.9	132	4.4	9.2	normal	normal	normal	nil	[illegible]	recovered
19	17	PONAMAL	22/F	>50	yes	yes	yes	[illegible]	120	140/80	30/min	72%	yes	yes	normal	normal	normal	normal	[illegible]	19000	66/34	10.6	31/0.9	140	5.0	9	normal	3070	[illegible]	[illegible]	[illegible]	recovered
20	18	JYOTHILA	16/F	<50	yes	no	no	[illegible]	78	120/70	14/min	97%	no	no	normal	normal	normal	normal	normal	8700	70/20/2	9.0	26/0.8	130	3.3	9	normal	normal	normal	nil	[illegible]	recovered
21	19	LAKSHMI	26/F	>50	yes	no	no	[illegible]	80	100/70	14/min	97%	yes	no	normal	normal	normal	normal	[illegible]	9500	67/32/1	12.9	31/0.9	140	5.0	9	normal	3640	normal	[illegible]	[illegible]	recovered
22	20	AMUDHA	28/F	>50	yes	yes	yes	[illegible]	114	140/80	32/min	86%	yes	yes	normal	normal	normal	normal	[illegible]	14200	65/33/2	11.4	30/5.3	141	6.2	9.2	normal	7060	II T V2 V3	[illegible]	[illegible]	recovered
23	21	SAKTHI	25/F	<50	yes	no	no	[illegible]	86	120/70	16/min	97%	no	no	normal	normal	normal	normal	normal	7500	71/20/1	11.7	31/0.9	140	4	8.5	normal	normal	normal	nil	[illegible]	recovered
24	22	SATHAKU	45/M	<50	yes	no	yes	[illegible]	78	110/80	10/min	97%	no	yes	normal	normal	normal	normal	normal	8900	72/26/2	12.4	26/0.8	132	4.4	9.2	normal	normal	normal	nil	[illegible]	recovered
25	23	DEVI	27/F	>50	yes	yes	no	[illegible]	122	140/80	34/min	79%	yes	no	normal	normal	normal	normal	[illegible]	15000	67/32/1	10.6	74/4.5	133	5.3	8	normal	6050	[illegible]	[illegible]	TRACHEOS	expired
26	24	SHANTHI	15/F	>50	yes	no	yes	[illegible]	92	110/60	14/min	97%	yes	yes	normal	normal	normal	normal	[illegible]	14000	66/34	11.1	79/3.7	141	5.3	8.7	normal	6530	normal	[illegible]	[illegible]	recovered
27	25	HARIMUT	27/M	<50	yes	no	no	[illegible]	76	110/70	16/min	97%	no	no	normal	normal	normal	normal	normal	9900	62/36/2	13.6	19/0.9	132	4.4	9	normal	normal	normal	nil	[illegible]	recovered
28	26	RAMESH	19/M	<50	no	no	no	[illegible]	80	100/60	10/min	97%	no	no	normal	normal	normal	normal	normal	7900	60/38/2	14.4	31/0.9	135	4.3	8.5	normal	normal	normal	nil	[illegible]	recovered
29	27	GANESHA	25/M	<50	no	no	no	[illegible]	72	120/80	16/min	97%	no	no	normal	normal	normal	normal	normal	8200	61/39	12.1	31/0.9	134	4.2	8.5	normal	normal	normal	nil	[illegible]	recovered
30	28	HEPSIBA	17/F	>50	yes	yes	yes	[illegible]	110	150/80	30/min	79%	yes	yes	normal	normal	normal	normal	[illegible]	12900	62/30	9	29/0.7	129	5.3	8.5	normal	8700	normal	[illegible]	[illegible]	recovered
31	29	RAJALING	27/M	<50	no	no	no	[illegible]	74	110/70	10/min	97%	no	no	normal	normal	normal	normal	normal	11100	65/35	13.2	26/0.8	135	4.5	9	normal	normal	[illegible]	nil	[illegible]	recovered
32	30	CHELLAM	25/F	<50	no	no	no	[illegible]	78	120/80	20/min	97%	no	no	normal	normal	normal	normal	normal	10100	71/29	12	19/0.9	132	4.2	9	normal	normal	normal	nil	[illegible]	recovered
33	31	THANGAM	20/F	>50	yes	no	no	[illegible]	86	110/60	16/min	97%	yes	no	normal	normal	normal	normal	[illegible]	8200	71/27/2	11.2	29/0.7	131	5.4	9.2	normal	6900	normal	[illegible]	[illegible]	recovered
34	32	MARY PUS	24/F	>50	yes	yes	yes	[illegible]	122	140/80	30/min	69%	yes	yes	normal	normal	normal	normal	[illegible]	10100	70/29/1	12.7	82/3.6	140	6.2	9.2	normal	10050	[illegible]	[illegible]	TRACHEOS	recovered
35	33	PALMANI	17/F	>50	yes	no	no	[illegible]	74	110/60	16/min	97%	yes	no	normal	normal	normal	normal	[illegible]	8400	66/33/1	11	31/0.9	130	5.3	9	normal	5900	normal	[illegible]	[illegible]	recovered
36	34	PETCHIAM	17/F	>50	yes	yes	no	[illegible]	116	130/80	10/min	79%	yes	no	normal	normal	normal	normal	[illegible]	16400	62/37/1	11.7	80/1.7	135	5.1	8.6	normal	4700	[illegible]	[illegible]	[illegible]	expired
37	35	MALATHI	25/F	<50	no	no	no	[illegible]	102	120/80	20/min	97%	no	no	normal	normal	normal	normal	normal	7700	66/32/2	12.2	19/0.9	136	4.2	9.2	normal	normal	[illegible]	nil	[illegible]	recovered
38	36	VINOTHA	18/F	>50	yes	yes	yes	[illegible]	116	140/80	16/min	86%	yes	yes	normal	normal	normal	normal	[illegible]	14600	69/31	12	34/4.9	137	5.7	8.3	normal	5490	[illegible]	[illegible]	[illegible]	recovered
39	37	KAVITHA	16/F	>50	yes	no	no	[illegible]	86	110/70	10/min	97%	yes	no	normal	normal	normal	normal	[illegible]	13560	67/32/1	13.6	29/0.7	131	5.3	9	normal	3460	normal	[illegible]	[illegible]	recovered
40	38	DHIVYA	17/F	<50	no	no	no	[illegible]	72	100/60	14/min	97%	no	no	normal	normal	normal	normal	normal	5200	60/32	12.0	31/0.9	130	5.0	9	normal	normal	normal	nil	[illegible]	recovered
41	39	YASHODH	55/F	<50	no	no	no	[illegible]	104	100/70	14/min	97%	no	no	normal	normal	normal	normal	normal	8400	65/34/1	11.5	26/0.8	134	4.2	9.2	normal	normal	[illegible]	nil	[illegible]	recovered
42	40	SRIJAYAK	57/M	<50	no	no	no	[illegible]	78	110/80	10/min	97%	no	no	normal	normal	normal	normal	normal	7200	60/32	13.6	19/0.9	135	4.3	8.3	normal	normal	normal	nil	[illegible]	recovered
43	41	SUNDHAR	56/M	<50	no	no	no	[illegible]	76	120/70	16/min	97%	no	no	normal	normal	normal	normal	normal	8700	67/31/2	14.2	29/0.7	136	4.2	9.2	normal	normal	normal	nil	[illegible]	recovered
44	42	SUDALAIM	20/M	<50	no	no	no	[illegible]	78	110/60	16/min	97%	no	no	normal	normal	normal	normal	normal	9000	70/30	13.6	26/0.8	135	3.3	8.3	normal	normal	normal	nil	[illegible]	recovered
45	43	AJANTHA	24/F	>50	yes	no	no	[illegible]	84	120/80	10/min	97%	yes	no	normal	normal	normal	normal	[illegible]	8600	65/34/1	8.6	31/0.9	137	4.3	9.2	normal	[illegible]	normal	[illegible]	[illegible]	recovered
46	44	ESAKKIAM	23/F	>50	yes	yes	yes	[illegible]	116	150/80	30/min	70%	yes	yes	normal	normal	normal	normal	[illegible]	15200	67/33	7.3	120/5.6	135	6.3	8.1	normal	3850	II T V2 V3	[illegible]	TRACHEOS	expired
47	45	PITCHAI	40/M	>50	yes	no	yes	[illegible]	82	100/70	14/min	97%	yes	yes	normal	normal	normal	normal	[illegible]	14600	64/36	12.0	19/0.9	130	4.3	9.7	normal	5520	normal	[illegible]	[illegible]	recovered
48	46	PRISCILLA	17/F	<50	no	no	no	[illegible]	90	110/60	14/min	97%	no	no	normal	normal	normal	normal	normal	9900	67/33	10.2	29/0.7	132	4.1	9	normal	normal	normal	nil	[illegible]	recovered
49	47	SIVARANI	40/F	<50	yes	no	no	[illegible]	72	100/70	16/min	97%	no	no	normal	normal	normal	normal	normal	10900	64/33/3	10.5	26/0.8	130	4.2	9.2	normal	normal	normal	nil	[illegible]	recovered
50	48	VADIVAIY	13/M	<50	no	no	no	[illegible]	86	120/80	10/min	97%	no	no	normal	normal	normal	normal	normal	7000	65/32/3	13.1	31/0.9	129	3.3	8.3	normal	normal	normal	nil	[illegible]	recovered
51	49	MARIYAP	33/M	>50	yes	no	yes	[illegible]	110	100/60	10/min	97%	yes	yes	normal	normal	normal	normal	[illegible]	10700	60/32	11.0	29/0.7	141	5	9.2	normal	3430	[illegible]	[illegible]	[illegible]	recovered
52	50	MANIMEG	24/F	>50	yes	no	no	[illegible]	78	100/70	20/min	97%	no	no	normal	normal	normal	normal	normal	8400	70/27/3	10.7	26/0.8	134	4.3	9	normal	normal	normal	nil	[illegible]	recovered
53	51	GANAPAT	26/F	<50	yes	no	no	[illegible]	78	100/60	16/min	97%	no	no	normal	normal	normal	normal	normal	5200	74/25	10.6	26/0.8	133	4	9.2	normal	normal	normal	nil	[illegible]	recovered
54	52	JAYAPHA	16/F	>50	yes	no	no	[illegible]	80	120/30	16/min	97%	yes	no	normal	normal	normal	normal	[illegible]	14300	66/33/1	9.7	31/0.9	132	5.2	9.1	normal	5230	normal	[illegible]	[illegible]	recovered
55	53	[illegible]	[illegible]	[illegible]	yes	yes	yes	[illegible]	110	140/80	40/min	79%	yes	yes	normal	normal	normal	normal	[illegible]	16000	60/32	[illegible]	35/4.9	134	6.4	[illegible]	[illegible]	3370	II T V2 V3	[illegible]	TRACHEOS	expired

ANNEXURE III

KEY TO MASTER CHART

NORMAL VALUES:

PULSE RATE: 60 – 100 bpm

BLOOD PRESSURE: 90/60 – 140/90 mmHg

RESPIRATORY RATE: <20/min

SpO_2: > 96%

HEMOGLOBIN: MALES – 13.3 – 16.2 g/dL FEMALES – 12-15.8 g/dL

LEUCOCYTES: 4000 – 11000 cells/cu.mm

DIFFERENTIAL COUNT:

NEUTROPHILS: 40-70%

LYMPHOCYTES: 20-50%

EOSINOPHILS: 0-6%

BLOOD UREA LEVELS: <40 mg/dL

SERUM CREATININE: 0.5 -1.2 mg/dL

SERUM SODIUM: 136 146 meq/L

SERUM POTASSIUM: 3.5 – 5 meq/L

PLASMA CALCIUM: 8.7 -10.2 mg/dL

TOTAL CPK: MALES – 51 – 294 U/L FEMALES – 39 – 238 U/L

HD – Hemodialysis

ANNEXURE IV

ABBREVIATIONS:

PPD – PARA-PHENYLENE-DIAMINE

CPK – CREATINE PHOSPHOKINASE

LDH – LACTATE DEHYDROGENASE

ECG – ELECTRO CARDIOGRAPH

ECHO - ECHOCARDIOGRAM

HD – HEMODIALYSIS