“PREDICTION OF ILLNESS SEVERITY AND OUTCOME OF CHILDREN WITH KEROSENE INGESTION ADMITTED IN TIRUNELVELI MEDICAL COLLEGE USING THE SCORING SYSTEM FOR HYDROCARBON POISONING”

Dissertation submitted in partial fulfilment of the requirement for the award of the Degree of

M.D. DEGREE – BRANCH VII

PAEDIATRICS

APRIL 2016

TIRUNELVELI MEDICAL COLLEGE HOSPITAL

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI,

TAMILNADU

CERTIFICATE

This is to certify that Dissertation entitled **"PREDICTION OF ILLNESS SEVERITY AND OUTCOME OF CHILDREN WITH KEROSENE INGESTION ADMITTED IN TIRUNELVELI MEDICAL COLLEGE USING THE SCORING SYSTEM FOR HYDROCARBON POISONING"** submitted by Dr. Senthil. P, M.B.B.S. to The Tamilnadu Dr. M.G.R. Medical University, Chennai, in partial fulfilment for the award of M.D. Degree (Paediatrics) is a bonafide work carried out by him under my guidance and supervision during the academic year 2013 – 2016. This dissertation partially or fully has not been submitted for any other degree or diploma of this university or other.

Prof.Dr.M.MATHIVANAN, MD,
GUIDE, Associate Professor,
Unit Chief, UNIT II,
Department of Paediatrics
Tirunelveli Medical College
Tirunelveli – 627011.

Prof. Dr.C.KRISHNAMURTHY, MD,
Professor and HOD,
Department of Paediatrics
Tirunelveli Medical College
Tirunelveli – 627011
.

The DEAN,
Tirunelveli Medical College,
Tirunelveli

DECLARATION

I, Dr. Senthil. P, M.B.B.S. solemnly declare that the Dissertation titled **"PREDICTION OF ILLNESS SEVERITY AND OUTCOME OF CHILDREN WITH KEROSENE INGESTION ADMITTED IN TIRUNELVELI MEDICAL COLLEGE USING THE SCORING SYSTEM FOR HYDROCARBON POISONING"** has been prepared by me under the expert guidance and supervision of **Prof. Dr. M. MATHIVANAN, MD., DCH.,** Associate professor, Department of Paediatrics, Tirunelveli Medical College Hospital, Tirunelveli.

This dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University, Chennai, in partial fulfilment of the regulations for the award of MD Degree BRANCH VII (PAEDIATRICS)

It was not submitted to the award of any degree/diploma to any University either in part or in full previously.

Place: Tirunelveli
Date:

Dr. Senthil. P, MBBS
Post graduate
M.D. (Paediatrics),
Tirunelveli Medical College,
Tirunelveli

ACKNOWLEGEMENT

I am very much thankful to the Dean **Dr. K. Sithy Athiya Munarvah**, Tirunelveli Medical College Hospital, Tirunelveli, who had granted permission to do this study in this institution,

I take this opportunity to express my deepest sense of gratitude to professor **Prof. Dr. C. Krishnamurthy, MD(Paediatrics).,** Head of the Department of Paediatrics, Tirunelveli Medical College Hospital, Tirunelveli for encouraging me and rendering timely suggestions and guiding me throughout the course of this study. I will be forever indebted to him for his constant support.

I am extremely thankful to my guide **Prof. Dr. M. MATHIVANAN, MD., DCH.,** Associate professor for guiding me throughout the study.

I sincerely thank my professor **Dr. T.R.R. Ananthy Shri, M.D(Paediatrics)., Dr. A.S. Babu Kandakumar, MD., DCH., DNB.,** for their support and guidance.

I am thankful to my assistant professors **Dr. Baskar.C, Dr. Senthil kumaran, Dr. Suresh** for their guidance and support throughout my study period in this institution.

I am also grateful to my statistician Mr. Selva Prakash, statistician for their useful inputs.

I also thank the Department of Radiology for their support and co-operation to this study

I wish to express my gratitude to my parents Mrs. Inthirani. P, my brother Mr. Ravikumar. P, my wife Dr. Anbarasi. C.P., and my children Harini and Dhanvine for their love and support in doing my dissertation.

I also like to express my gratitude to my friends and colleagues who have always been a source of love, support and encouragement.

TIRUNELVELI MEDICAL COLLEGE

INSTITUTIONAL RESEARCH ETHICS COMMITTEE
TIRUNELVELI, STATE OF TAMILNADU, SOUTH INDIA PIN 627011
91-462-2572733-EXT; 91-462-2572944; 91-462-2579785; 91-462-2572611-16
online@tvmc.ac.in, tirec@tvmc.ac.in; www.tvmc.ac.in

CERTIFICATE OF REGISTRATION & APPROVAL OF THE TIREC REF NO: 533/PAED/2014/40

PROTOCOL TITLE: PREDICTION OF ILLNESS SEVERITY AND OUTCOME OF CHILDREN WITH KEROSENE INGESTION ADMITTED IN TIRUNELVELI MEDICAL COLLEGE USING THE SCORING SYSTEM FOR HYDROCARBON POISONING.

NAME OF PRINCIPAL INVESTIGATOR: Dr. Senthil .P, MBBS.,
DESIGNATION OF PRINCIPAL INVESTIGATOR: Post Graduate in MD Paediatrics
DEPARTMENT & INSTITUTION: Department of Paediatrics, Tirunelveli Medical College

Dear Dr. Senthil .P, *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 ☐

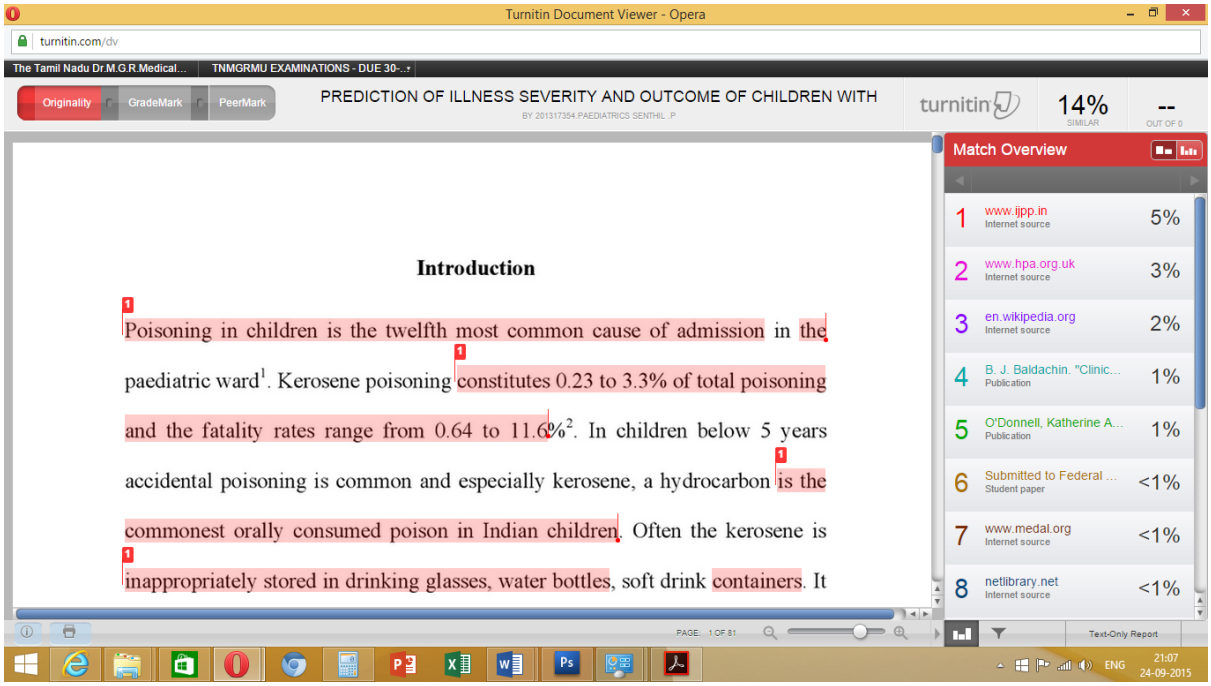
THE PROTOCOL IS APPROVED IN ITS PRESENTED FORM ON THE FOLLOWING CONDITIONS

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.
    g. Any deviation/violation/waiver in the protocol must be informed

STANDS APPROVED UNDER SEAL

Dr.K.Shantaramma MD
Registrar, TIREC
Tirunelveli Medical College. Tirunelveli – 627011

Dr.V.Ramasubramanian MD DM
Member Secretary, TIREC
Tirunelveli Medical College. Tirunelveli – 627011


Turnitin Document Viewer - Opera
turnitin.com/dv
The Tamil Nadu Dr.M.G.R.Medical...
TNMGRMU EXAMINATIONS - DUE 30-...
Originality
GradeMark
PeerMark
PREDICTION OF ILLNESS SEVERITY AND OUTCOME OF CHILDREN WITH
BY 201317354.PAEDIATRICS SENTHIL .P
turnitin
14%
SIMILAR
--
OUT OF 0
Introduction
Poisoning in children is the twelfth most common cause of admission in the paediatric ward[1]. Kerosene poisoning constitutes 0.23 to 3.3% of total poisoning and the fatality rates range from 0.64 to 11.6%[2]. In children below 5 years accidental poisoning is common and especially kerosene, a hydrocarbon is the commonest orally consumed poison in Indian children. Often the kerosene is inappropriately stored in drinking glasses, water bottles, soft drink containers. It
PAGE: 1 OF 81
Match Overview
1 www.ijpp.in Internet source 5%
2 www.hpa.org.uk Internet source 3%
3 en.wikipedia.org Internet source 2%
4 B. J. Baldachin. "Clinic... Publication 1%
5 O'Donnell, Katherine A... Publication 1%
6 Submitted to Federal ... Student paper <1%
7 www.medal.org Internet source <1%
8 netlibrary.net Internet source <1%
Text-Only Report
ENG
21:07
24-09-2015

CONTENTS

ABSTRACT

Introduction

Kerosene poisoning is common in children in developing countries like India. Kerosene is used for cooking and lighting purpose in India especially in rural areas. Kerosene is commonly stored in water bottles or in soft drink bottles and the colour of kerosene makes them attractive to children. Kerosene because of its low surface tension, gets aspirated while ingestion and commonly following vomiting and causes chemical pneumonitis.

Aim of the study:

To predict the illness severity and outcome of children less than or equal to 12 years with kerosene ingestion admitted at Tirunelveli Medical College on admission using the Scoring System for Hydrocarbon poisoning.

Method of study:

It is a hospital based observational study. Children with ingestion of kerosene poisoning are evaluated with the scoring system of hydrocarbon poisoning. The score attained is used to assess the illness severity in terms of duration of hospitalization, complications like chemical pneumonitis, ICU treatment, ventilator support and death.

The scoring system consists of 6 parameters. They are cyanosis, SpO2 level, CNS manifestations, Pulmonary involvement, Respiratory distress and history of vomiting. The maximum score is 15.

Observations and results:

40 children with kerosene ingestion were included in the study. 80% of children were less than 3 years of age, 72% of children were boys. Majority of cases occurred during summer. 82% of children with poisoning were from rural areas. 75% of children had history of vomiting, of which 67% were induced. Chest complications were developed in 60% of children who had vomiting and no complications in children without vomiting. The mean duration of hospitalization after kerosene ingestion was two and half hours

45% of children had complications. Among those who developed complications 11% of children were with score 5 to 8, 83% of children were with score 9 to 11 and there was one death (6%) with the score 12 to 15. The correlation coefficient of the score and outcome was 0.849 which is a very high correlation.

Conclusion:

The Scoring system for hydrocarbon poisoning is very useful in assessing the clinical severity and outcome of children with kerosene poisoning. Kerosene poisoning is common among boys, less than 3 years of age from rural population.

Poisoning occurs commonly during summer. Vomiting is an important risk factor for developing chemical pneumonitis

Key words:

Kerosene poisoning, chemical pneumonitis, aspiration and Scoring system for Hydrocarbon poisoning.

Introduction

Poisoning in children is the twelfth most common cause of admission in the paediatric ward[1]. Kerosene poisoning constitutes 0.23 to 3.3% of total poisoning and the fatality rates range from 0.64 to 11.6%[2]. In children below 5 years accidental poisoning is common and especially kerosene, a hydrocarbon is the commonest orally consumed poison in Indian children. Often the kerosene is inappropriately stored in drinking glasses, water bottles, soft drink containers. It may be attractive to children

Hydrocarbons:

Kerosene is a hydrocarbon. Hydrocarbons represent a diverse group of substances. Occasionally hydrocarbon and petroleum distillate are used interchangeably.

Classification of hydrocarbons[3]:

I Based on their chemical and clinical properties:

1. Aliphatic Hydrocarbons:

   Easily aspirated following ingestion and poorly absorbed from GI tract. They have minimal systemic effects.

   Examples: Kerosene, mineral spirits, gasoline, lubricating oil, naphtha and mineral oils

2. Halogenated hydrocarbons:

   Minimal aspiration following ingestion, readily absorbed from GI tract. They produces systemic toxicity.

   Examples: Trichloro ethane, methylene bromide, lindane, chlordane.

3. Aromatic hydrocarbons:

   They are commonly used for inhalation.

   Examples: toluene, benzene, xylene

II Based on viscosity:

1. Very low:

   Mineral seal oil (furniture polish)

2. Low:

   Benzene

   Aniline

   Pine oil

   Toluene

   Nitrobenzene

   Chlorinated hydrocarbons

   Camphor

Pesticides with hydrocarbon

3. Middle:

    Kerosene

    Gasoline

    Lighter oil

4. High:

    Lubricating greases and oils

    Petroleum jelly

    Motor oil

    Paraffin wax

Halogenated hydrocarbons such as methylene chloride and solvent Trichloro ethane can produce liver and renal toxicity following chronic exposure, and with acute exposure produce central nervous system effects. The solvents xylene and toluene are commonly abused by inhalation through ‘huffing’ or ‘bagging’ for the euphoric effects. Cardiac arrhythmias may occur due to sensitization of the heart to catecholamines. Chronic exposure can cause electrolyte abnormalities, peripheral neuropathies and renal toxicity.

Chronic exposure to benzene is implicated in the development of leukaemia and aplastic anaemia. Hydrocarbon also used as solvents for highly toxic ingredients such as camphor, organophosphate insecticides and heavy metals.

Kerosene, also known as lamp oil, is a liquid mixture of chemicals produced from the distillation of crude oil. The word kerosene comes from the Greek word ‘keros’, meaning ‘wax’. Kerosene is also called Paraffin in the UK, Southeast Asia, East Africa and South Africa.

Kerosene:

Kerosene is a hydrocarbon produced on an industrial scale by fractional distillation of crude oil between 150 °C and 275 °C in a process similar to that used to produce diesel or petrol. It results in a mixture with density of 0.78 – 0.81 g/cm^3. It contains carbon chains between 6 and 16 carbon atoms per molecule. It is immiscible in water, but miscible in petroleum.

Branched and straight chain alkanes and naphthenes form major components of kerosene which normally account for at least 70% of the volume. The flash point of kerosene is between 100 F and 150 F (37^{o}C and 65^{o}C). Its auto ignition temperature is 428 F (220^{o}C).

History:

Kerosene initially called as “coal oil” in 1700s. It was produced as a by-product of making coal tar and coal gas. In 1846, Abraham Gesner, a Canadian geologist had coined the name ‘Kerosene’. The cost of extracting kerosene from coal was high.

Samuel Martin Kier in 1851 distilled kerosene from crude oil by a new process of his own invention. He called it as ‘Carbon Oil’. Following petroleum discovery in Canada in 1858 and in Pennsylvania in 1859, the increased supply of petroleum allowed kerosene to be produced in large scale from oil refiners. Thereby kerosene production entirely stepped from coal to petroleum in the 1860s. In the UK although the kerosene was increasingly produced from petroleum, manufacturing from coal continued into the early 20^{th} century.

Uses:

Kerosene is a major component (> 60%) of aviation (jet) fuels, is used for “oil” central heating systems and can be used as a cleaning agent or solvent. Kerosene is able to remove other petroleum products such as chain grease. It also used as a lubricant, with less risk of combustion compared to using other petroleum products. It can also be sued as a cooling agent in metal production and treatment.

Kerosene is an effective pesticide, working against all large number of insects, especially against bed bugs and head lice. It can also be used to kill mosquito larvae by applying to standing pools of water. Kerosene covers the insect's trachea with a thin film of paraffin, smothering the insects by preventing the exchange of oxygen. Approximately 7½ million tons of kerosene was used in the UK in 2005. The use of paraffin heaters in developed countries has substantially decreased since the Second World War due to improved electrical and gas supplies.

However, kerosene is still extensively used for cooking, heating and lighting in the developing world and so cases of accidental poisoning by children are still relatively common in developing countries like India, other South – East Asian countries, African countries[4].

In such countries kerosene is the main fuel used for cooking, especially by the poor. Kerosene stoves have replaced traditional wood based cooking. Kerosene is used as a fuel in portable stoves. It also used in outdoor activities and mountaineering. The Indian government keep the price of kerosene low by subsidizing the fuel. As such, increase in the price of kerosene by reducing the subsidy can have a major political and environmental consequence. In Nigeria an attempt to remove kerosene subsidy by the government met with strong opposition. Total Kerosene

consumption for all purposes is equivalent to about 1.2 million barrels per day.

Kerosene Poisoning:

Young children are at high risk for accidental ingestion because of their immature metabolic and respiratory system. They are more likely to ingest because of their undeveloped sense of smell and taste. They mistake kerosene for water or soft drinks believing it as a pleasant drink with which they are familiar[5,6].

Accidental ingestion of hydrocarbons in middle and low income countries are 50 times more common than high income countries because in low and middle income regions hydrocarbons are the extensively used for cooking, lighting and heating[7].

In study conducted in a tertiary care centre in New Delhi over a period of 10 months reported 52 cases of accidental poisoning of kerosene ingestion which formed 1% of all paediatric admissions and overall mortality was 7.7% [8]

Ingestion

Signs of oral kerosene poisoning include diarrhoea, nausea and vomiting. Approximately 30 – 50% of children presenting with suspected kerosene ingestion are asymptomatic[9].

Children have survived ingestion of up to 1.7 g kg-1 and recorded instances of fatal poisoning have been associated with doses ranging from ~ 2 to 17 g kg-1[10,11]. However, death following oral exposure is normally associated with aspiration of vomit rather than systemic toxicity per se. Vomiting occurs in approximately one third to one half of patients.

Kerosene easily aspirated during ingestion and mainly vomiting because of its low surface tension and rapidly spread over the surface of contact. This is particularly dangerous in young children, who form the bulk of patients, because of the relatively smaller surface area of the tracheo-bronchial tree.

REVIEW OF LITERATURE

Usage of kerosene:

In India Kerosene is widely used for cooking, lighting and heating purposes.

In a study conducted at Egypt found that the hydrocarbon was used in 60% for cleaning purposes, for lighting and cooking 30% and as a machine fuel in 10% of poisoning cases.

In 86% of cases the hydrocarbon was kept in discarded water bottles and discarded soft drink bottles. It was kept within the reach of the children in 90% of poisoning cases like kitchen, bathroom and below staircases.

Age and Sex distribution:

In a study on 48 children with hydrocarbon poisoning admitted to PICU in PGI, Chandigarh[12] found that mean age of poisoning is 2 years and boys are more commonly involved with the Male:female ratio of 3.4:1

In a study of clinical profile of children with kerosene aspiration by Venkatesh conducted at JIPMER, Pondicherry, India, reported that Boys aged less than three years formed a significant portion of study population[13].

In a 5 year retrospective study conducted in Israel on 274 children, 61% of them boys and 39% were girls. The ages ranged from 6 months to 18 year with a median of one and half year [14].

In study conducted in a tertiary care centre in New Delhi over a period of 10 months reported 52 cases of accidental poisoning of kerosene ingestion 80% of the children were between 1 to 5 years with male predominance[8].

In a 10 year prospective study conducted in Sri Lanka on 526 children with Kerosene poisoning reported that 62% of children were boys and 38% were girls. 83% of children were in the age group 1 to 3 year[15].

In a study conducted in Chennai by Santhanakrishnan et al., reported that 83% of children were in age group one to two year and 58% of children were boys[11].

In a study conducted at Egypt 70% of cases were between 1 and 3 years old. They also found that 62% are male children and 38% are female[16].

In another study of 200 cases over a period of 5 year it was found that average age was 19 months with majority are in the range of 5 months to 5 years[17].

In a study conducted on 122 cases of kerosene poisoning for six years by Shotar et. al., in Jordan found that 61% of children were boys and majority (80%) of children were below two year[18].

Distribution of poisoning in relation to season:

In a study conducted in a series of 200 cases of kerosene poisoning showed a definite seasonal variation. The number were high uniformly during the summer and a sharp decline during the winter months[17].

Another study conducted at Israel on 274 children found that majority group of children admitted during the summer months[4].

In a study conducted in Jordan, the largest group of children with kerosene poisoning was admitted in summer months.

Quantity of ingestion:

The quantity of ingestion is divided into below or above 30ml in studies. One study found that 74% of cases had ingested less than 30ml and the remaining 26% ingested more than 30ml.

Pathophysiology of chemical pneumonitis[3]:

The most important manifestation of kerosene poisoning is chemical pneumonitis due to aspiration. Aspiration occurs usually during coughing and gagging at the time of ingestion or vomiting. The physical properties of the hydrocarbons contribute to pulmonary manifestation. The risk of aspiration is inversely proportional to viscosity. Viscosity is measured in Saybolt Seconds Universal (SSU). It is the time required for a liquid to flow through a calibrated orifice. Products with low viscosity (less than 60 SSU)

like Kerosene, gasoline, naphtha are associated with a high aspiration potential.

The physical properties of high volatility and low surface tension of kerosene contribute to respiratory injury. High volatility displaces alveolar gas. Low surface tension promotes spreading of the kerosene on lung tissue, thereby interferes with ventilation when aspiration occurs. Only small quantities (<1 ml) of kerosene need be aspirated to produce significant injury. Aspiration of mineral seal oil which has a very low viscosity of 47 SSU can produce severe pulmonary complications.

Hydrophobic nature of kerosene allows it to penetrate deep into the tracheo-bronchial tree, producing inflammation. Bronchiolar exudates containing primarily polymorphonuclear leukocytes found within hours of aspiration. This may clinically manifest as cough, rales, bronchospasm and radiographic changes. Another postulated mechanism of pulmonary damage is from inactivation of the type II pneumocytes and results in secondary surfactant deficiency. The volatile chemical displace alveolar oxygen, leading to hypoxia. Direct contact of kerosene with alveolar membranes may lead to haemorrhage, hyperaemia, oedema, vascular thrombosis and leukocyte infiltration. The result is pneumonitis, poor oxygen exchange and atelectasis.

Pneumatoceles following kerosene ingestion generally occurs. In the radiograph it is seen as dense infiltrates. There are two postulated mechanism for pneumatoceles formation. One is necrosis of pulmonary tissue. Second is the local obstruction leading to over distension and rupture of alveoli.

Clinical manifestations usually begin in the few hours after ingestion and usually resolve in 2-8 days.

Complications include hypoxia, mechanical ventilation induced barotrauma and Acute Respiratory Distress Syndrome (ARDS). Prolonged hypoxia may result in seizures, encephalopathy and death.

Pathological features[19]:

Acute Lung Injury (ALI) and Acute Respiratory Distress Syndrome (ARDS) are the clinical syndromes associated with chemical pneumonitis and the corresponding pathology is Diffuse Alveolar Damage (DAD).

Distinction between ALI and ARDS is not possible on pathological grounds. But there may be some features at the ultrastructural level. On occasion lung biopsies may be performed on patients with the clinical features of ALI or ARDS to confirm the nature of the process.

Pathologically DAD can be divided into different phases. These represent a dynamic, continuous process rather than discrete, pathological

steps. The early events are described as the “exudative” phase, followed by the “proliferative” phase. The proliferative phase may be followed by organization with the development of established fibrosis. Alternatively resolution of the proliferative phase may occur with a return to normal or near normal lung architecture. These two possibilities are, however, not mutually exclusive and may overlap histologically in different areas of the lung.

Exudative phase:

In the very earliest stages of this phase the lungs may appear macroscopically normal. As the process develops the lungs show increasing degrees of oedema. At autopsy they are heavy, often weighing in excess of 1 kilogram each and the cut surface is red and beefy in appearance with the lung slices being rather firm (Figure 1).

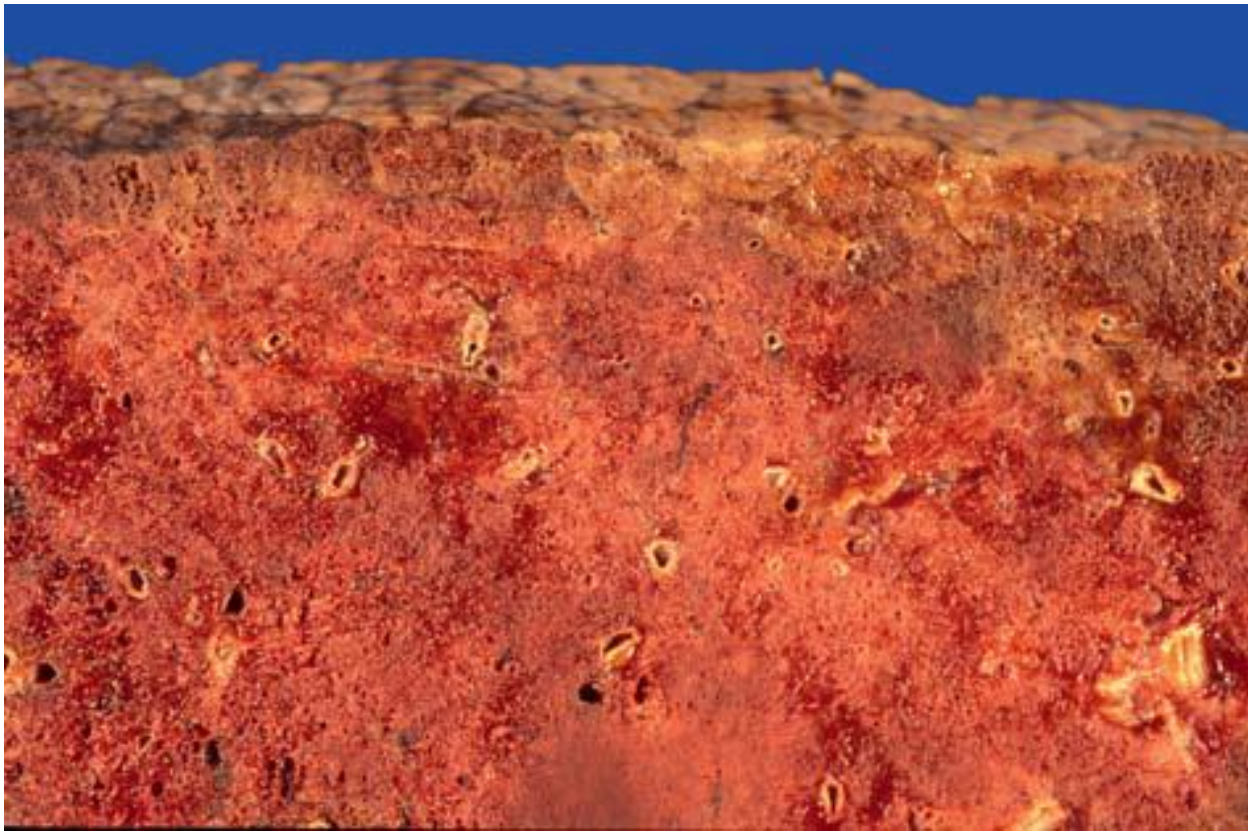


Figure 1. Showing macroscopic appearance. The cut surface of the lung is red and beefy. The lungs are heavy and firm in consistency.

The earliest changes identifiable in the lung are only detectable by electron microscopy. At the ultrastructural level there is evidence of injury and necrosis of types I and II pneumocytes, leaving a denuded and in some cases damaged basement membrane (Figures 2 and 3).

The alveolar capillaries are typically described as showing increased numbers of marginated neutrophils, although this is not a universal finding, and there is interstitial oedema. Small fibrin thrombi may be identified. The endothelial cells may also show injury but this is variable, and there may also be some evidence of capillary proliferation.

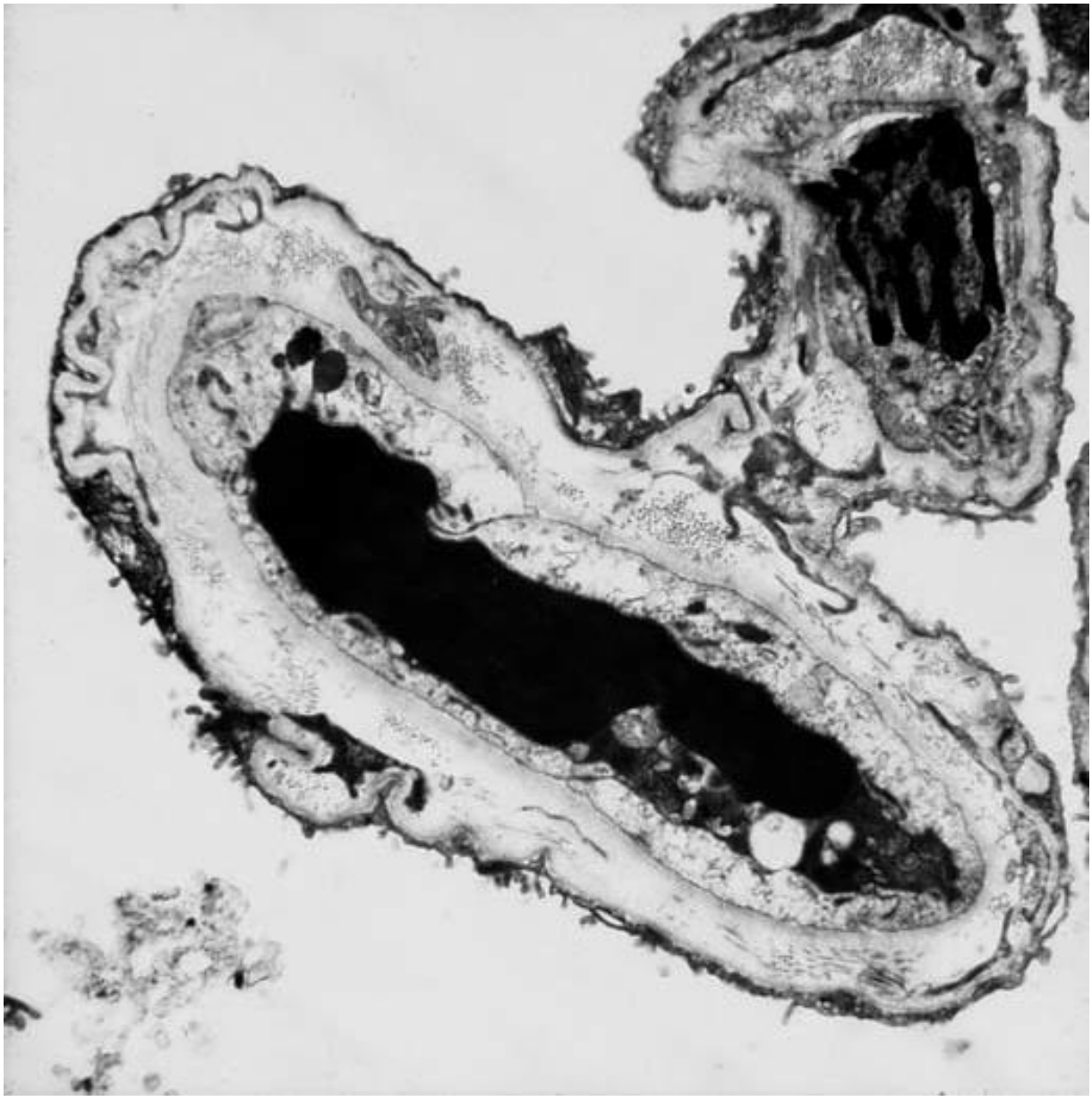


Fig 2. Electron micrograph demonstrating evidence of papillary processes on the surface of type I epithelial cells, oedema of the basement membrane and electron lucency of the endothelial cells consistent with early alveolar injury.

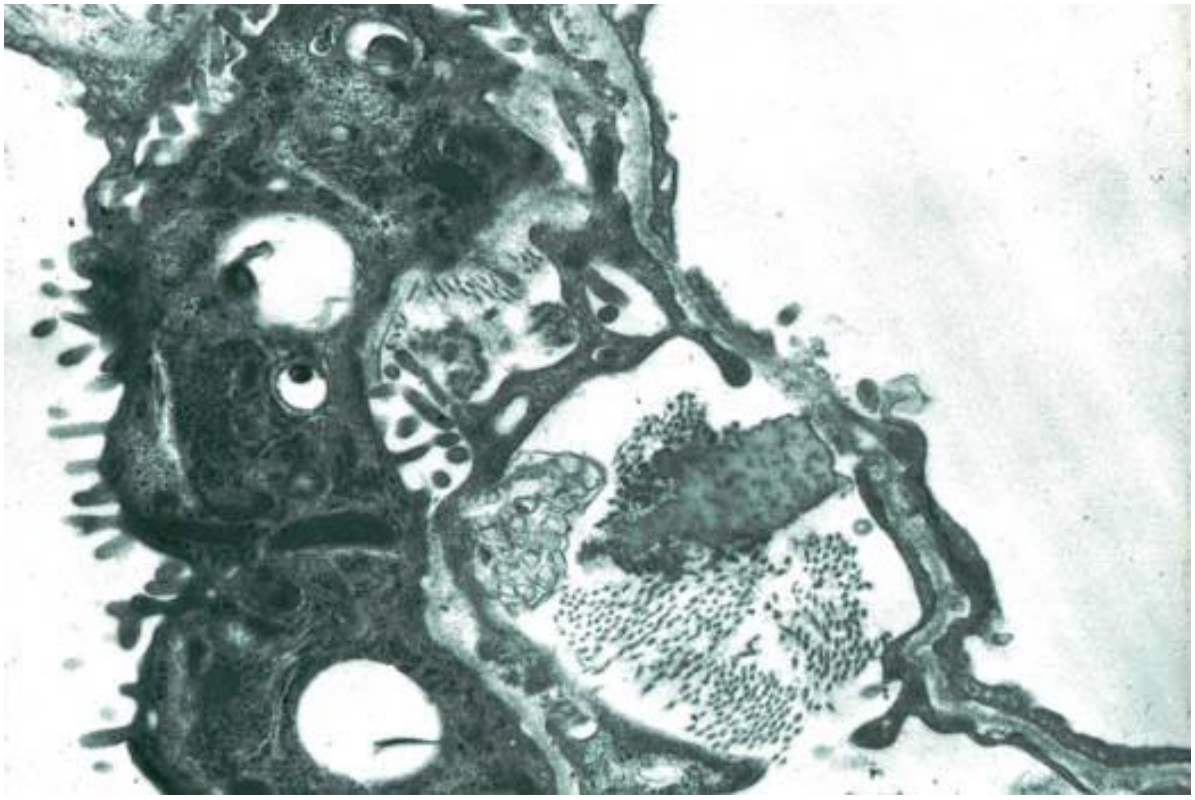


Fig 3. Electron micrograph showing disruption and a break in the basement membrane between the capillary space and the adjacent alveolar space.

Two to three days following injury intra-alveolar oedema will be apparent at the light microscopic level (Figure 4). Sometimes haemorrhagic oedema fluid is an exudate; rich in fibrin due to the “leaky” alveolar walls. This exudate, mixed with necrotic cellular debris, condenses to form hyaline membranes. These membranes are often regarded has the characteristic histological feature of the exudative phase (Figure 5).

In the early stages hyaline membranes may be relatively focal but as the injury develops they may be more widespread. These are characterized

on H&E staining as intensely eosinophilic relatively dense bands of proteinaceous material lining alveolar airspaces and ducts.

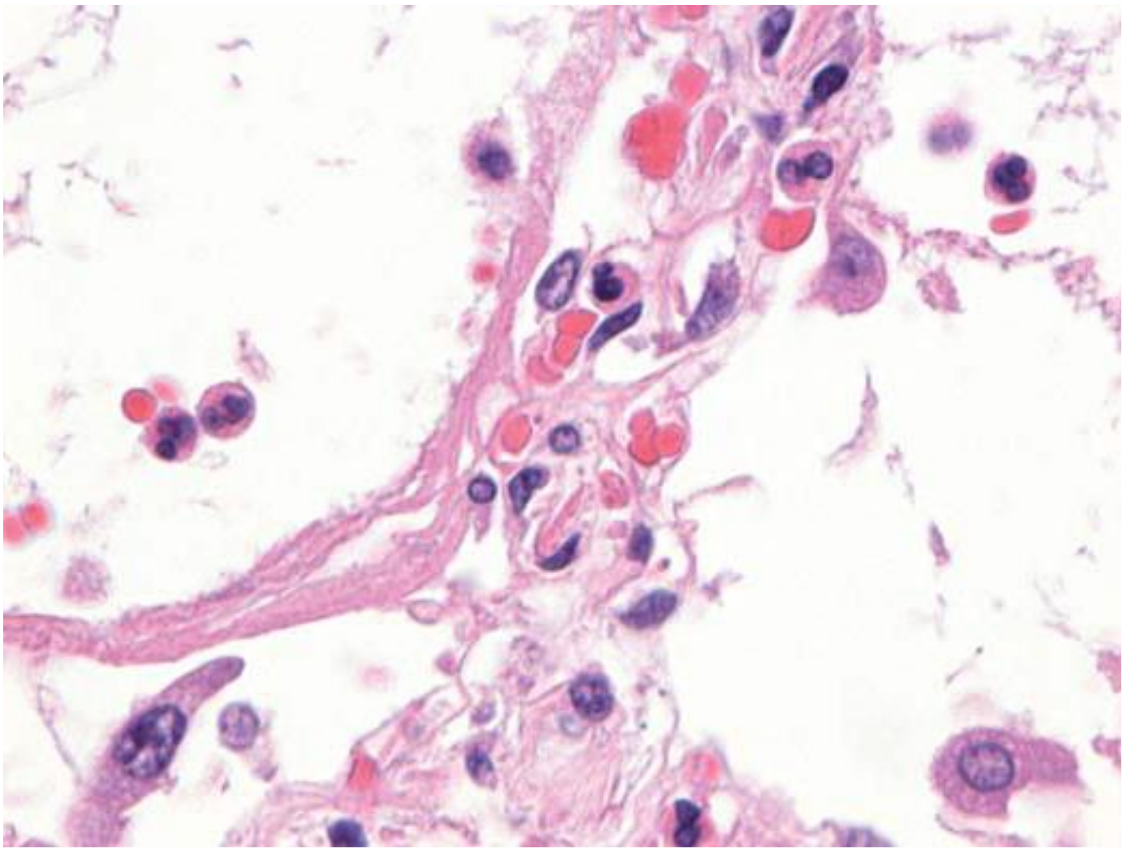


Figure 4. The alveolar capillaries are congested and oedematous.

Neutrophils can be prominent

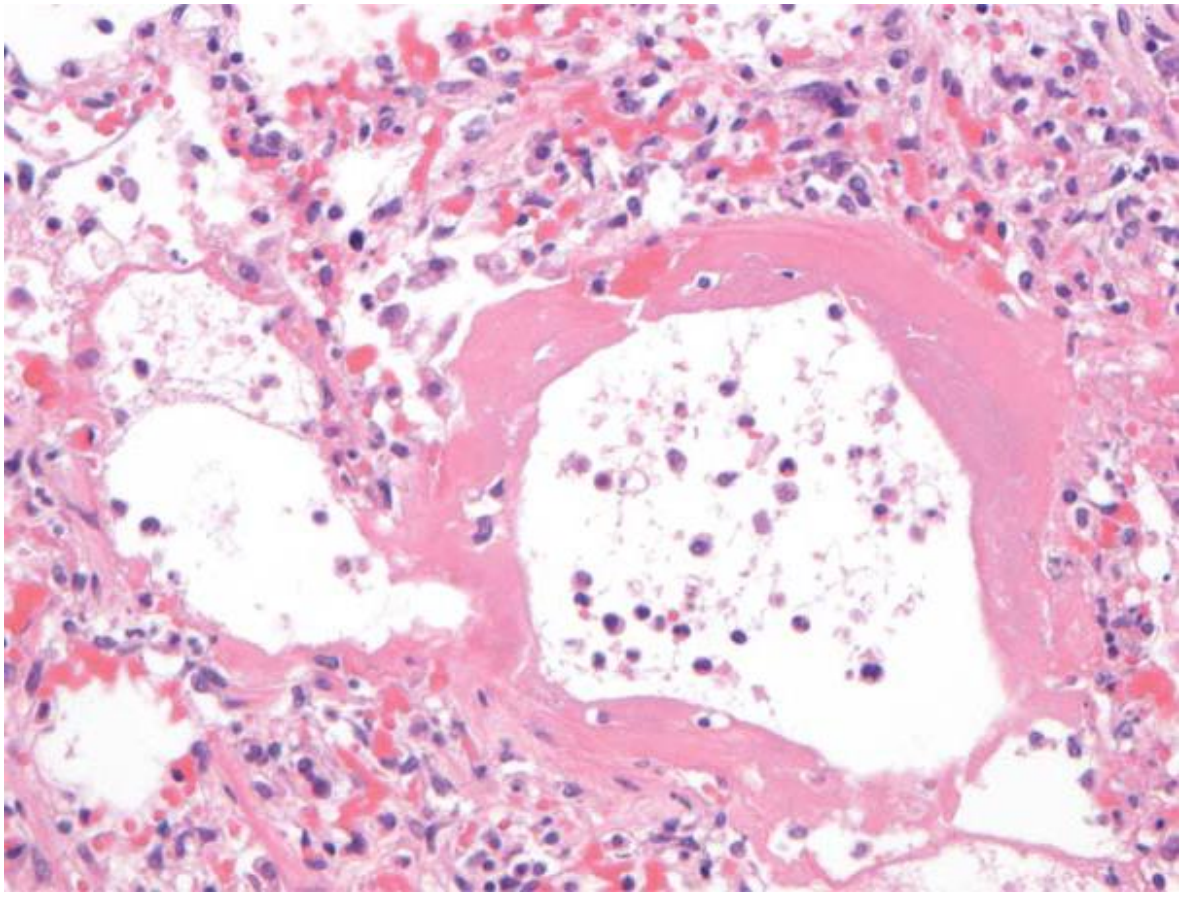


Figure 5. Showing hyaline membranes

Hyaline membranes are associated with a rather variable and often patchy increase in interstitial chronic inflammatory cells. Small fibrin thrombi may also be identified (Figure 6).

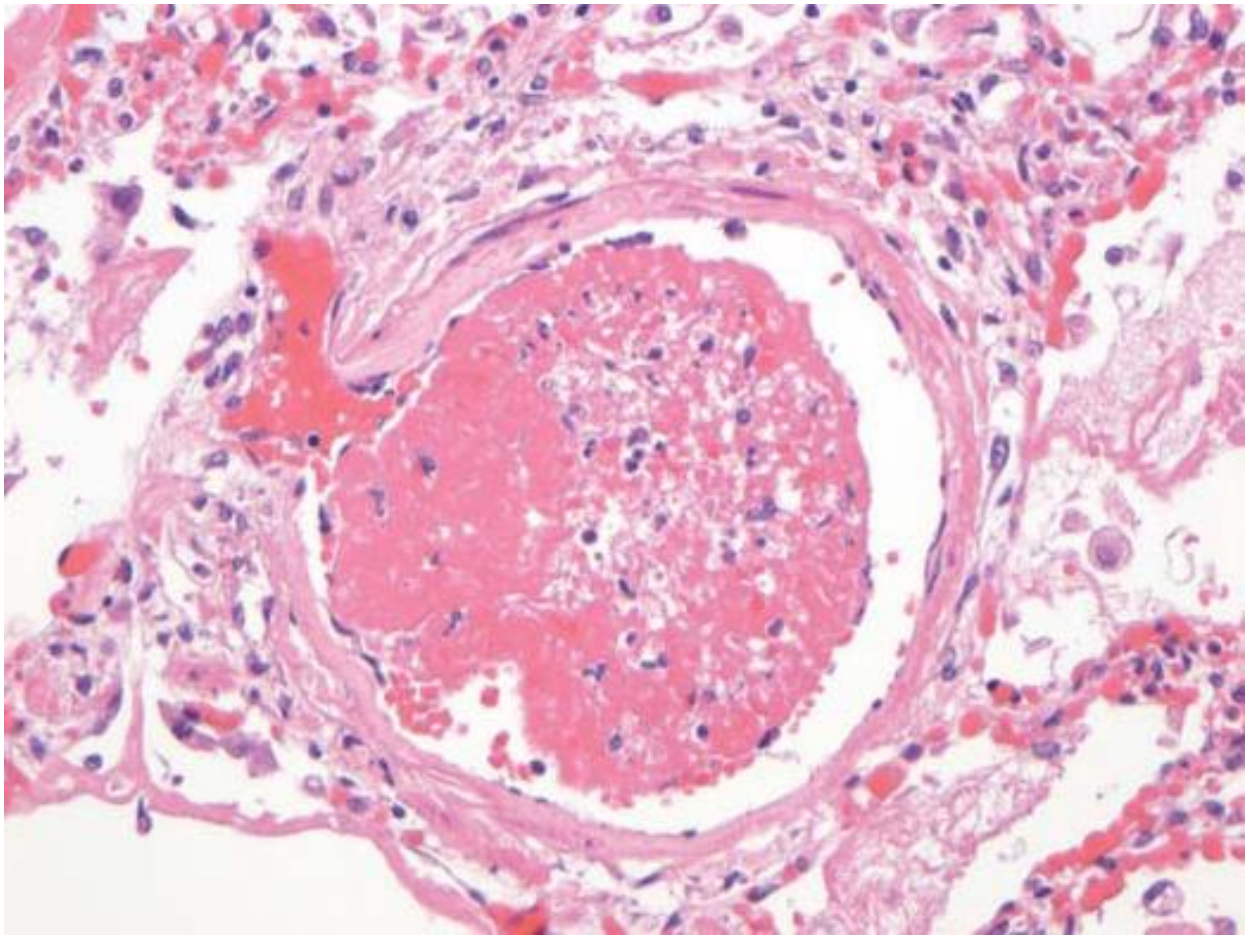


Figure 6. Diffuse Alveolar Damage with thrombus.

Although neutrophils are believed to be important in the pathogenesis of ALI and are detectable in Broncho Alveolar lavage samples early in the disease process, they are often inconspicuous on light microscopy in the airspaces. In cases where significant numbers of neutrophils are evident associated with fibrinous exudates, the alternative diagnosis of pneumonia needs to be considered.

Proliferative phase:

The proliferative phase is usually evident by 5–7 days from the onset of the process. Macroscopically the lungs remain heavy and firm but by this stage begin to develop a greyer consolidated appearance (Figure 7).

Figure 7. Macroscopic view. The cut surface of the lung features geographic grey consolidation. This appearance represents the proliferative phase.

Histologically this phase is characterized by organization of the exudative fluid with the appearance of myofibroblasts and the development of granulation tissue polyps within alveolar spaces and ducts (Figure 8).

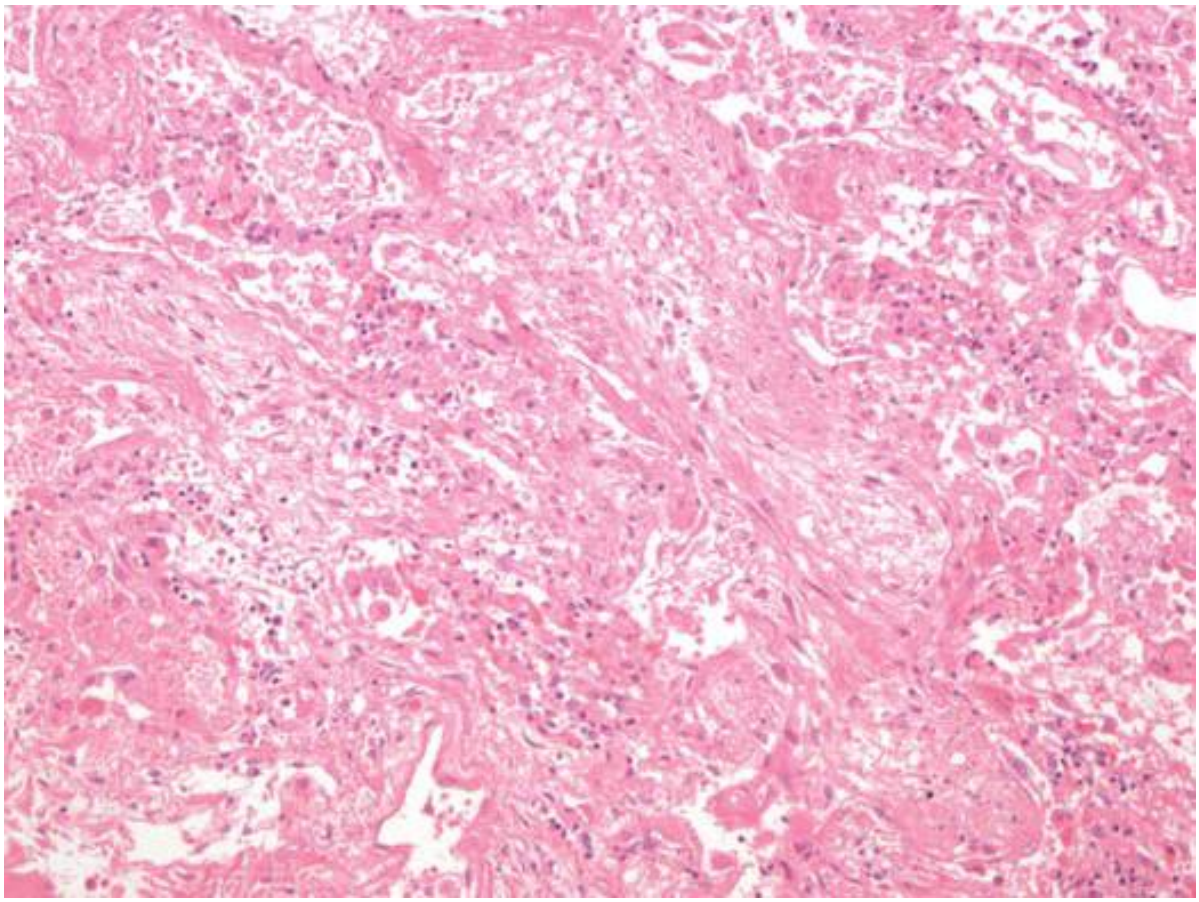


Figure 8. Fibro proliferative phase. The air spaces are filled with plugs of organizing immature fibroblastic tissue comprising myofibroblasts and loose matrix elements.

This process may be variable in its distribution and foci of organization may be seen admixed with persisting hyaline membranes and interstitial inflammatory cells. In association with this picture, there is

marked proliferation of type II alveolar cells along the alveolar walls (Figure 9). These cuboidal cells have a “hob-nail” appearance and usually feature a considerable degree of cytological atypia with pleomorphic nuclei and prominent nucleoli.

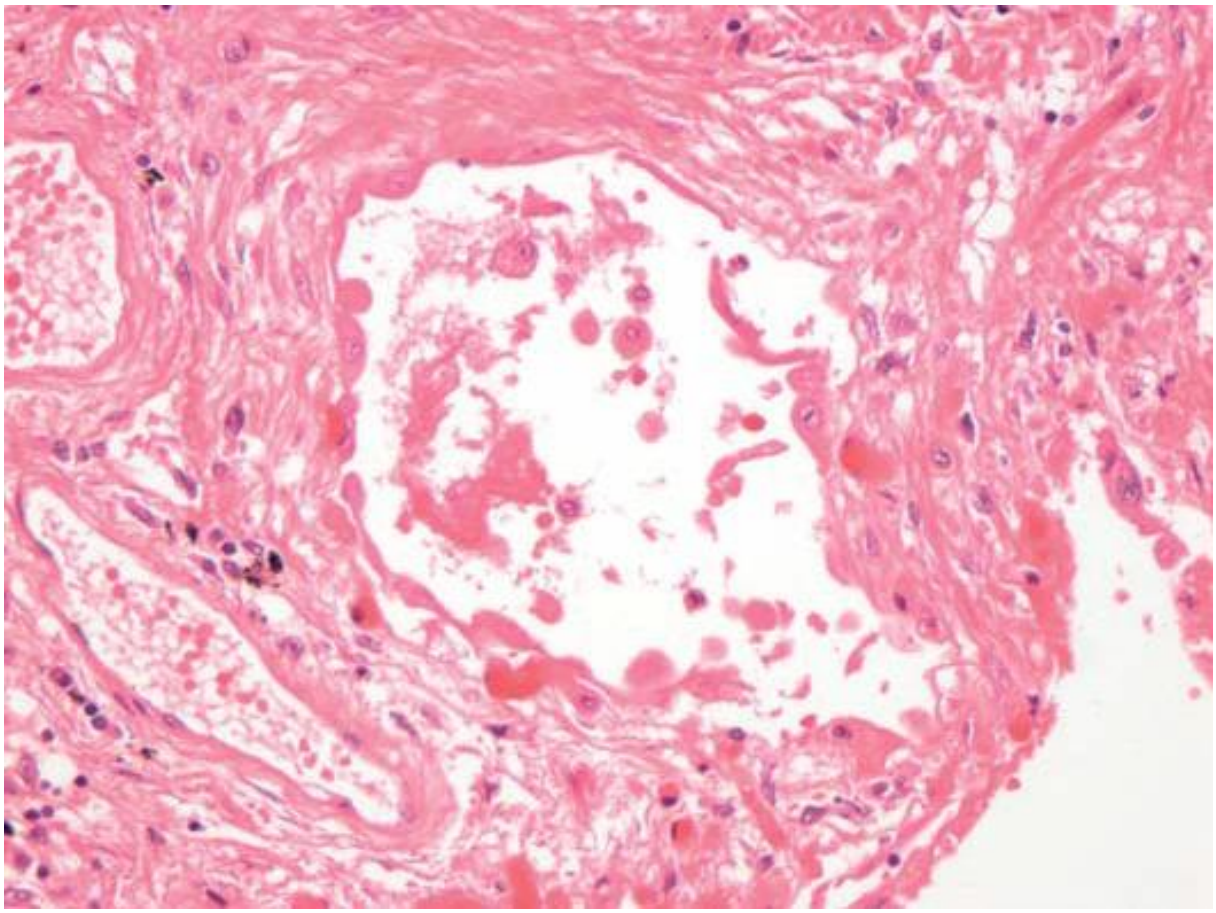


Figure 9. Type II alveolar epithelial cell hyperplasia

At the ultrastructural level, the basement membrane may be disrupted and there may be further evidence of endothelial injury and micro thrombi within alveolar capillaries. Myofibroblasts both proliferate within the interstitium and also migrate through the breaks in the basement membrane into the exudates within the alveolar spaces and ducts. This process results

in the development of airspace granulation tissue polyps. This is associated with secretion of extracellular matrix components, such as fibronectin and tenascin.

Increasing interstitial fibrosis, collapse of the alveolar architecture and progressive distortion of the lung architecture often follow (Figure 10).

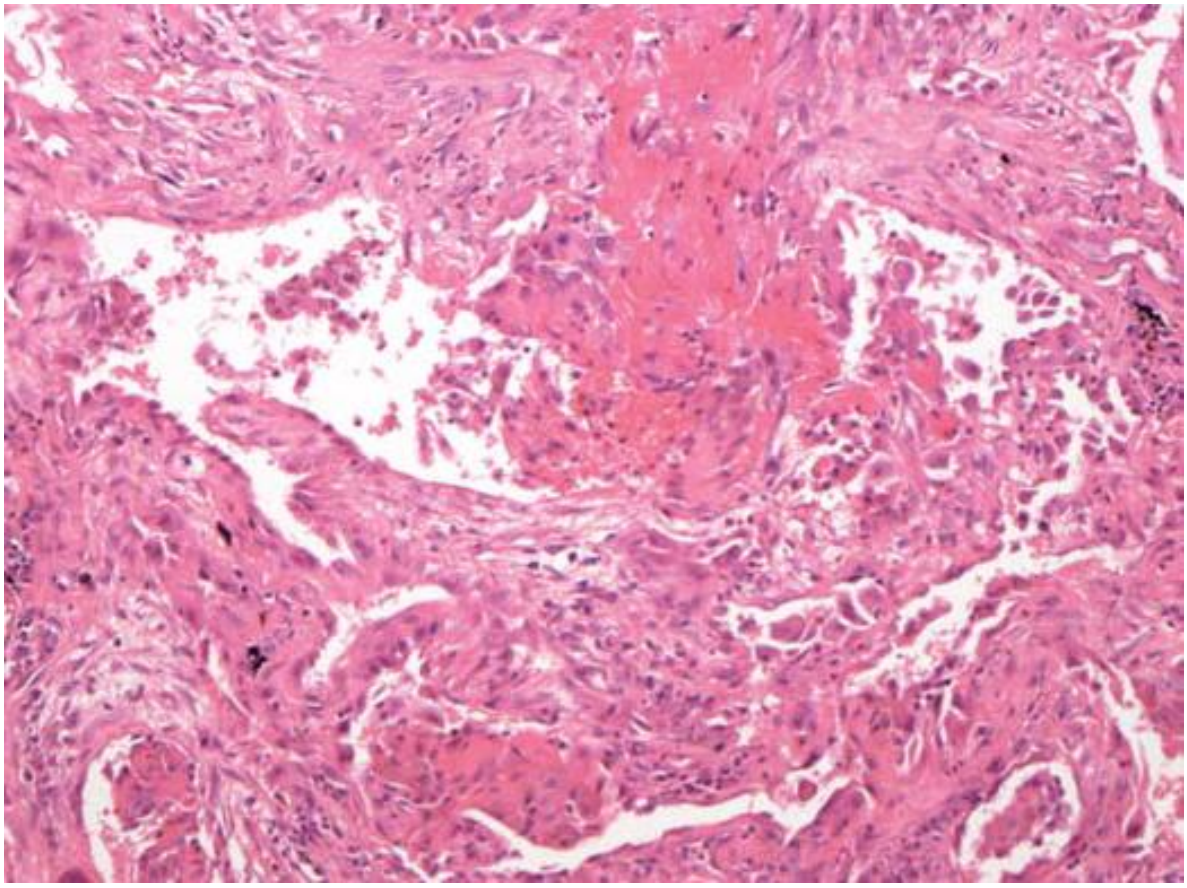


Figure 10. Fibroblastic exudates are incorporated into adjacent alveolar walls. This leads to pulmonary fibrosis

In patients surviving several weeks a variety of patterns of established fibrosis may be observed. In some, there may be a honeycomb pattern

although care needs to be taken to exclude a pre-existing usual interstitial pneumonia-pattern in such cases.

Others show extensive nodular fibrosis with residual cleft-like spaces lined by alveolar epithelial cells. Some develop a more diffuse pattern of alveolar wall thickening with better architectural preservation, the appearance of which may resemble the fibrotic nonspecific interstitial pneumonitis pattern (NSIP) (Figure 11).

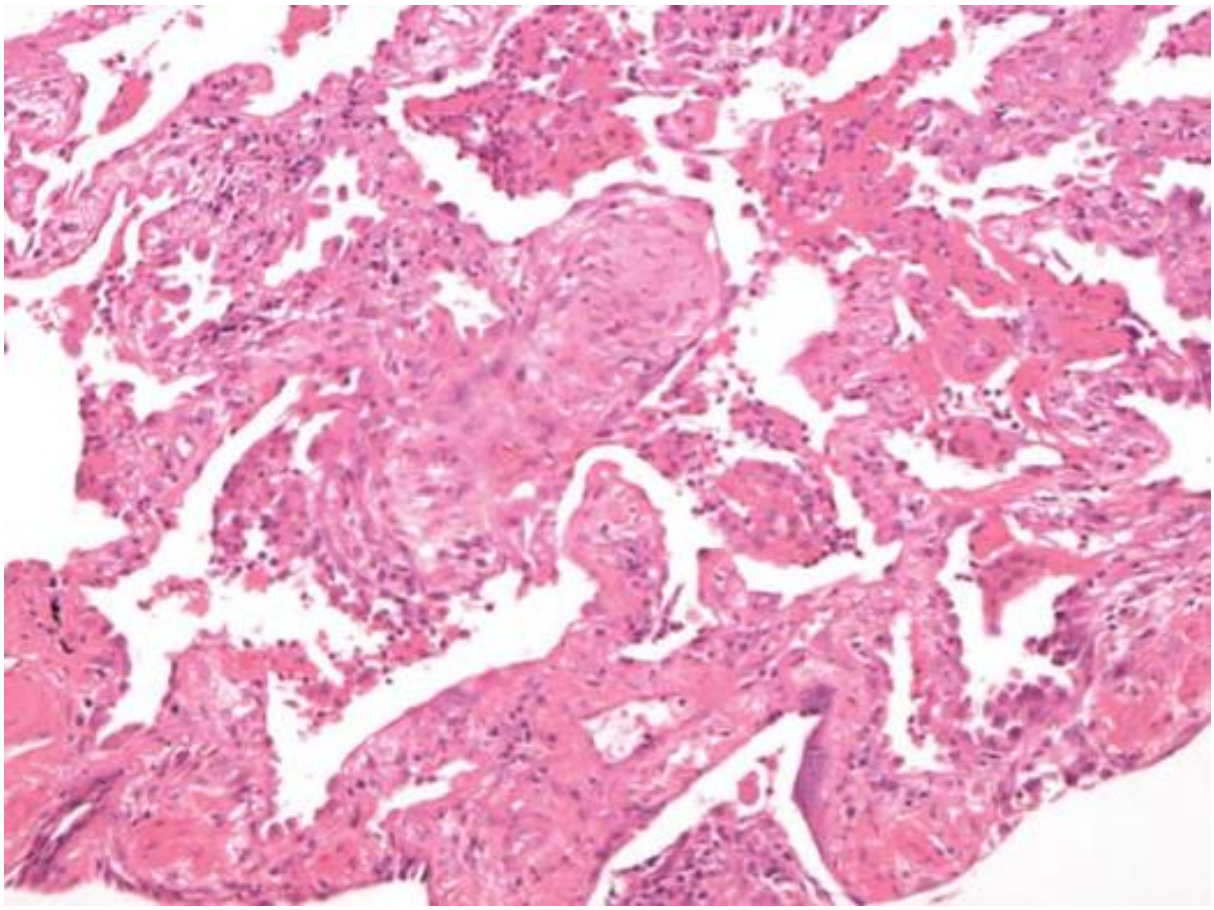


Figure 11. Lung fibrosis in a patient who had died following a prolonged ICU admission. A nonspecific pneumonia-like pattern is seen

An appropriate clinical history of previous severe lung injury is likely to distinguish these cases from idiopathic NSIP. There may be extensive squamous metaplasia and secondary acute inflammatory changes with neutrophils in cystic spaces. In the latter stages there is usually vascular remodelling with extensive medial hypertrophy of muscular pulmonary arteries, irregular intimal fibrosis in both arteries and veins, and arteriolization of pulmonary arterioles. Pulmonary veins also show marked intimal fibrosis, most likely secondary to the pulmonary fibrosis. Destruction of the pulmonary vascular bed leads to secondary pulmonary hypertension.

Resolution and patient survival may follow the proliferative phase. The factors responsible for the clearance of the immature organizing exudative process are poorly understood. Apoptosis of myofibroblasts combined with phagocytosis of the debris and immature extracellular matrix elements are suggested mechanisms. If resolution occurs early, before the development of fibrosis, then the lung architecture may return to near normal. Patients with more protracted courses are often left with variable degrees of established lung fibrosis and subsequent loss of respiratory function.

Clinical manifestations:

Kerosene mainly affects respiratory system and the central nervous system. Kerosene inhalation may cause headache, dizziness, drowsiness, incoordination and euphoria. The deterioration of the clinical condition may occur over the first two to three days and the resolution of symptoms will occur in three to six days[15].

Respiratory:

Aspiration into the lungs causes pneumonitis with choking, cough, wheeze, breathlessness, cyanosis and fever. Respiratory symptoms can remain mild or progress rapidly to the acute respiratory distress syndrome (ARDS) and respiratory failure. Ingestion causes nausea, vomiting. Aspiration into the lungs during ingestion and vomiting causes respiratory symptoms like gasping, choking, cough, wheeze, breathlessness. Chemical pneumonitis is the most serious pathological complication in the lung[18]. Kerosene induced chemical pneumonitis is usually characterized by respiratory distress in the form of tachypnea, fever, intercostal retractions, nasal flaring, grunting, cyanosis and hypoxemia[12].

In a study conducted in Chandigarh[12] it was found that the severity of aspiration pneumonia was higher in those with vomiting or who underwent lavage before admission. The symptoms appeared within 4 hours of ingestion. They also reported that hypoxemia on arrival, prior vomiting,

higher need for ventilation and ventilator associated complications were associated with poor outcome.

In a study of 200 cases with kerosene poisoning, 35% of cases developed respiratory involvement. They also reported that respiratory involvement appeared within one or two hours[17]

CNS manifestations:

CNS manifestations of kerosene poisoning are mainly due to the hypoxemia resulting from the pulmonary involvement. It ranges from headache, irritability, seizures, drowsiness to deep coma. It may be due to narcotic effects of Kerosene.

In a study of kerosene poisoning the CNS impairment seen in one third of the patients in the form of restlessness, stupor and convulsions[4].

Constitutional Disturbance:

Pyrexia is a common symptom in cases of kerosene poisoning. The pyrexia appears to be the direct result of kerosene absorption, in the absence of chemical pneumonitis. The pyrogenic action by kerosene was not severe enough to give rise to concern. But the duration of fever varied considerably and there was no apparent relation to the severity of illness and subsequent complications. For example in a study a two and half year old child who had

a temperature of 104.5F on admission didn't develop any chemical pneumonitis and was discharged well after 48 hrs[17]

Ocular exposure causes to the eyes causing an immediate stinging and burning sensation with lacrimation inspite of kerosene expected to be pH neutral.

On **dermal exposure** kerosene causes irritation and causes drying and cracking due to defatting action. There may be transient pain with erythema, blistering and superficial burns. Pneumonitis does not result from dermal absorption of kerosene.

The main hazard associated with kerosene is chemical pneumonitis, resulting from aspiration of vomitus following ingestion or inhalation of kerosene liquid or contaminated water. A rare complication of kerosene intoxication may be cardiac arrhythmia and ventricular fibrillation, attributed to increased myocardial sensitivity to endogenous catecholamines[20]

Lang et al.,(2008) found that 2% of cases ingesting hydrocarbons die every year in Kenya[21].

Balchandin et al., in a study of 200 cases reported one fatal case. That child presented to the hospital approximately two and half hours after ingestion and died one and half hours of admission in spite of ICU treatment. Autopsy of that child showed severe pulmonary oedema.

Investigations:

Chest radiography may initially be normal. The features of chemical pneumonitis occur in chest radiograph within 2-8 hrs of exposure in patients. Pneumatoceles and pleural effusions may occur. Fever and leucocytosis are common accompanying signs in patients with pneumonitis and don't necessarily imply bacterial superinfection.

Chest radiography:

Chest radiographs usually show bilateral alveolar infiltrates, though it is recognized that radiographic changes may lag behind profound hypoxemia by a few hours. The alveolar infiltrates reflect disruption of the barrier function of the alveolar – capillary membrane. The resultant congestion and depletion of surfactant lead to impaired lung compliance and alveolar atelectasis. As such, recruitment of congested/collapsed alveoli is fundamental to mechanical ventilation strategies aiming to improve ventilation-perfusion matching.

The extent of alveolar infiltration may vary greatly between patients, a wide variety of co-existence pathologies may be represented on the X-ray. It includes effects of barotrauma such as pneumothorax. The position of the patient (often supine) may make interpretation more difficult.

In a study of Hydrocarbon poisoning in children admitted to PICU in Chandigarh[12] reported following chest radiograph abnormality: Bilateral

lower lobe infiltrate in 34%, Right lower lobe infiltrate in 6%, left lower lobe infiltrates in 3%, pneumatoceles in 2% and Pneumothorax in 1%.

Baldachin et al., in a study reported that the X ray changes resembled those of non-segmental consolidation and atelectasis confined to the middle and lower zones of both lungs. This clearly indicates that aspiration is the primary cause of the pulmonary condition. Also the predominant auscultatory signs were found at the lung bases.

Abdel Raouf et al in a study found that 54% had normal X ray films. The remaining 46% had X ray findings like increased broncho vascular markings, hilar congestion, bilateral pneumonic patches.

Lifshitz et al., in a study of 274 children in children reported that 43% of children had chemical pneumonia in the form of interstitial pneumonitis and vomiting was significantly correlated with the development of pneumonia[14].

C T Scan:

Computed tomography (CT) scanning can add considerable information regarding the nature of alveolar infiltrates seen on a chest radiograph. CT generally reveals homogeneous and gravitationally dependent alveolar shadowing, often in a rather patchy distribution. The variable extent of parenchymal involvement and the gravitational dependence are worth keeping in mind.

CT scanning has recently provided valuable insights into the extremely variable degree to which patients' lung tissue can be successfully recruited for better oxygenation during mechanical ventilation. CT studies have also suggested that the structure and function of the lung varies markedly over time in ARDS, with progressive evolution of a more restrictive lung defect, development of microcystic bullae and a greater risk of pneumothorax.

Treatment

Because of the risk of aspiration emesis and lavage are contraindicated. Activated charcoal is not useful because it can induce vomiting and does not bind kerosene. In cases of co-ingestion is suspected, gastrointestinal decontamination would be warranted of toxic hydrocarbons like camphorated, halogenated and co-ingestion of heavy metals and pesticides. In such cases GI decontamination done after air way secured with cuffed endotracheal tube.

The skin should be decontaminated as soon as possible by removing the clothing and thoroughly washing the skin as the vapour inhalation and cutaneous absorption may occur for long time after exposure.

Treatment is generally supportive, consisting of oxygen, ventilator support and intravenous fluids. If chemical pneumonitis develops respiratory treatment is supportive. Stabilization of the airways is always the first

priority. Supplemental oxygen is given to children with face mask or oxygen hood. Early intubation, mechanical ventilation and use of positive end-expiratory pressure are indicated for children with inadequate oxygenation and who have severe respiratory distress or a decreased level of consciousness.

Standard mechanical ventilation, high-frequency ventilation, and ECMO have all been used to manage the respiratory failure and ARDS associated aspiration pneumonitis.

Routine prophylaxis with antibiotics is not necessary.

Prevention:

Parents should be educated as it is crucial in the prevention of accidental exposure. Advise the parents about labelling the harmful chemical and proper storage of chemicals. Parents should be taught of supervising their children when they are in high-risk areas like kitchen, garage and laundry rooms where usually the toxic substance are stored.

Prognostic indicator and scoring systems:

Gupta et al (1992)[22] Studied 95 children with kerosene poisoning over a period of three years. He studied the first seventy five patients retrospectively and included them into Internal group. For the rest twenty

five patients, he studied prospectively and included them into External group.

He divided the children in internal group into two groups based on the illness severity.

- **Group A:** Asymptomatic cases at 24 hours after admission
- **Group B:** Cases with either of the following:

i. Respiratory distress, fever or coma persisting for more than 24 hours

ii. Complications including myocarditis, encephalopathy or respiratory failure

iii. Death

These A and B groups were compared for different parameters like age, sex, quantity of kerosene ingestion, nutritional status, clinical features and radiograph findings.

Based on the severity of the illness and clinical features in the 70 patient in the internal group, he devised a weighted scoring system to determine the prognosis. This included

- Fever: absent 0, present 1
- Severe malnutrition: absent 0, present 1

- Respiratory distress: absent 0, present 2, with cyanosis 4
- Neurological symptoms: absent 0, present 2, with convulsions 4

So the score ranged from 0 to 10. Using discriminate function analysis he found out that those with a score of 4 or more is associated with prolonged hospital stay and complications. Those with the score equal to or more than 8 was found to have increased risk of dying. The validity of score was found to be 84%.

Jayashree et al.(2006)[12] had stated the predictors of poor outcome. They are history of prior lavage, hypoxemia on arrival, higher frequency of secondary pneumonia, higher need for ventilation and ventilator complications. They studied on children with hydrocarbon poisoning receiving intensive care treatment.

Abdel Raouf et al. (2012)[12] devised the Scoring system for hydrocarbon poisoning. They studied on 70 subjects of which 50 were classified into Test group and 20 were classified into Confirmatory group. In the test group they obtained data from some parameters examined in the subjects and were statistically studied and used to establish the scoring system. The subjects in confirmatory group were used to assess the validity of the scoring system.

They studied the clinical parameters. They classified them into four grades according to the clinical outcome observed in the test group. The four grades are Grade 1 contained subjects who showed complete cure and excellent prognosis, Grade 2 included the subjects who had chest complications in the form of chemical pneumonitis but had complete cure with treatment. Grade 3 had patients who needed ICU admissions and ventilator support and Grade 4 had patients who had died.

They found that following parameters affecting prognosis arranged according to severity Cyanosis, X ray, Duration of hospitalization, LFT, RFT, CNS manifestations, Pulmonary involvement, Quantity of kerosene ingested, WBC count, respiratory distress, Vomiting. They also found that the following parameters doesn't have statistically significance to affect the prognosis. They are fever, time passed after ingestion of hydrocarbon, age and sex.

They devised the scoring system using the parameters which can be assessed bedside and given score 0 to 3 according to their correlation with the prognosis based on the observations made in the test group.

The scoring system was applied for every patient in the confirmatory group and total scoring number was obtained for each patient. The total scoring number was interpreted as follows:

Score less than 5:

Excellent prognosis. Outpatient treatment would be enough

Score between 5 to 8:

Complete cure but have to be admitted to hospital

Score between 9 to 11:

Chest complication like chemical pneumonitis

Score between 12 to 15:

Bad prognosis by means of ICU admission, ventilator support and may be death.

They have found that the validity of the scoring system is 90%.

AIM OF THE STUDY

To predict the illness severity and outcome of children less than or equal to 12 years with kerosene ingestion admitted at Tirunelveli Medical College on admission using the Scoring System for Hydrocarbon poisoning.

Study design:

It is a hospital based observational study.

Methodology:

All children less than or equal to 12 years with history of kerosene oil ingestion admitted in Tirunelveli Medical college from April 2014 to August 2015 are evaluated with the scoring System for Hydrocarbon poisoning at the time of admission and serially every 2 hours upto 8 hours. The maximum score arrived is taken to assess the severity of outcome in the subjects.

The development of chemical pneumonitis associated with kerosene ingestion takes upto 8 hours. So the subjects are assessed with the scoring system every two hours upto 8 hours.

Inclusion criteria:

All children less than or equal to 12 years with history of kerosene oil ingestion admitted at Department of Paediatrics, Tirunelveli Medical College.

Exclusion criteria:

Children with mixed hydrocarbon ingestion or pure hydrocarbon ingestion other than Kerosene oil.

Children with known chronic respiratory illness.

Children with history of Central Nervous System, Cardiac or Renal diseases.

Study protocol:

After written consent from the parents the subjects are included in the study. The following details are obtained.

- Name, age, sex, residence
- Number of siblings
- Parent's education
- Usage of kerosene at home
- Container used to store kerosene
- Duration between ingestion and admission in hospital
- History of vomiting. If yes, whether it is spontaneous or induced
- Any treatment given before admission
- Past history of significant medical illness

Clinical examination is performed. Child was assessed for the following parameters

- Cyanosis
- SpO2 level
- CNS (Conscious / Drowsiness / Coma responding to verbal or painful stimuli / Not responding to verbal or painful stimuli)

- Pulmonary involvement (No lung signs / Wheeze / Crepitation and or diminished air entry)
- Respiratory distress (Cough, tachypnea, usage of accessory muscle)

The Total Scoring Number (TSN) is obtained at the time of admission and every 2 hours upto 8 hours

X - Ray was taken after 8 hours and assessed for the development lung complications.

Child was followed up for the duration of stay in the hospital, development of lung complications and need of ventilator support.

The final outcome obtained in terms of duration of stay, development of lung complications or death.

Scoring system for Hydrocarbon poisoning:

Parameter / Score	**0**	**1**	**2**	**3**
Cyanosis	Absent			Present
SpO_2 Level	>95%	81% - 95%	61% - 80%	Less than or equal to 60%
CNS	Fully conscious	Drowsiness	Coma responding to stimuli (Verbal or painful stimuli)	Deep coma (Not responding to verbal or painful stimuli)
Pulmonary involvement	Free chest	Wheezes	Crepitation and or diminished air entry	
Respiratory distress	Absent	Cough	Cough & tachypnea	Cough, tachypnea and or accessory muscle use
Vomiting	Absent	Present		

Total scoring number (TSN)

• (TSN) 0-4: excellent prognosis.

• (TSN) 5-8: complete cure.

• (TSN) 9–11: chest complications (chemical pneumonitis).

• (TSN) 12–15: ICU, ventilator use and may be death (bad prognosis).

Statistical analysis:

The different parameters are assessed in relation to outcome. Age distribution, sex distribution, relation between vomiting and total score.

The final outcome is correlated with the final score attained. The result is assessed in terms of correlation coefficient.

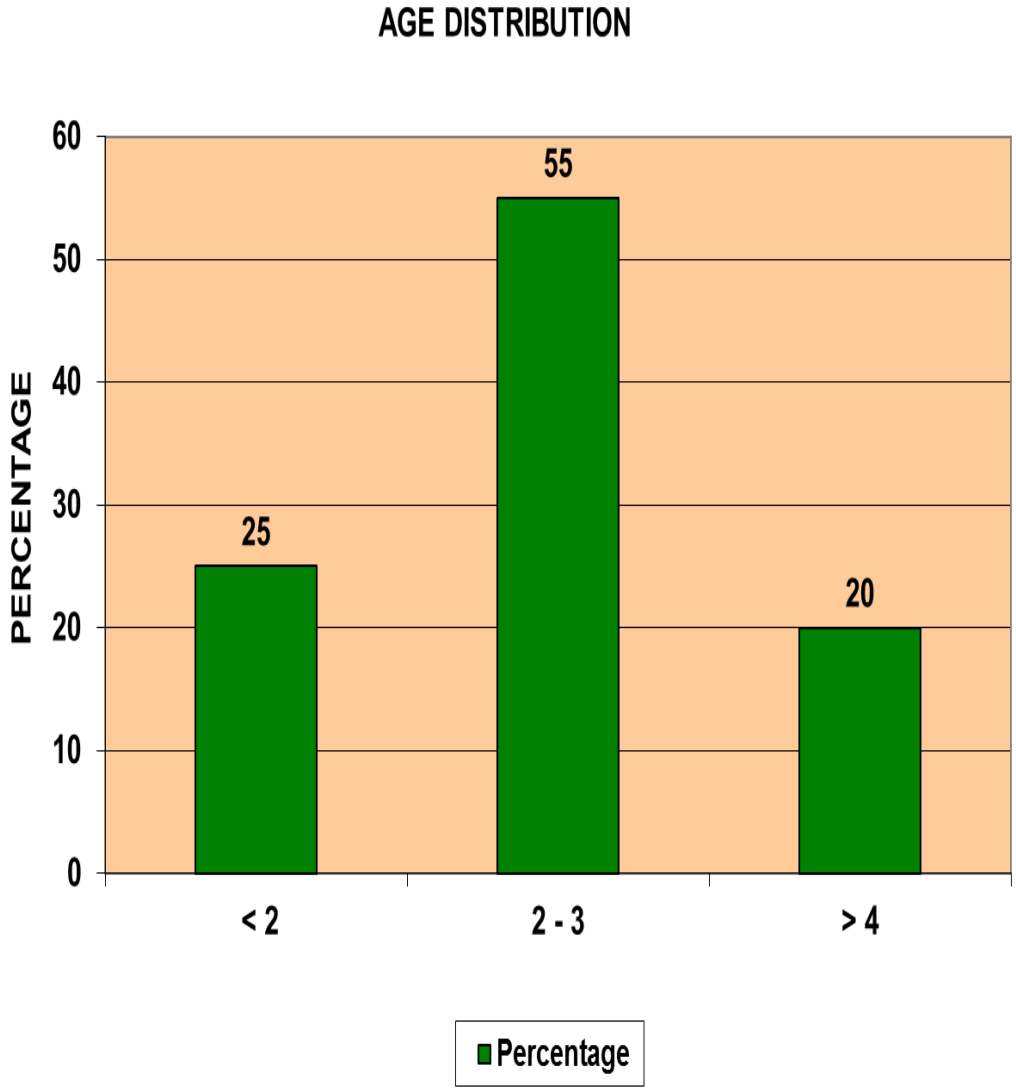
OBSERVATIONS AND RESULTS

40 subjects were included in the study. The following observation and results are obtained in the study group.

Age distribution:

Although the study group can include children upto 12 years, maximum age of children in the study was 6 years. The age ranged from 1 year to 6 year with mean of two and half years and the standard distribution of 1.25 years.

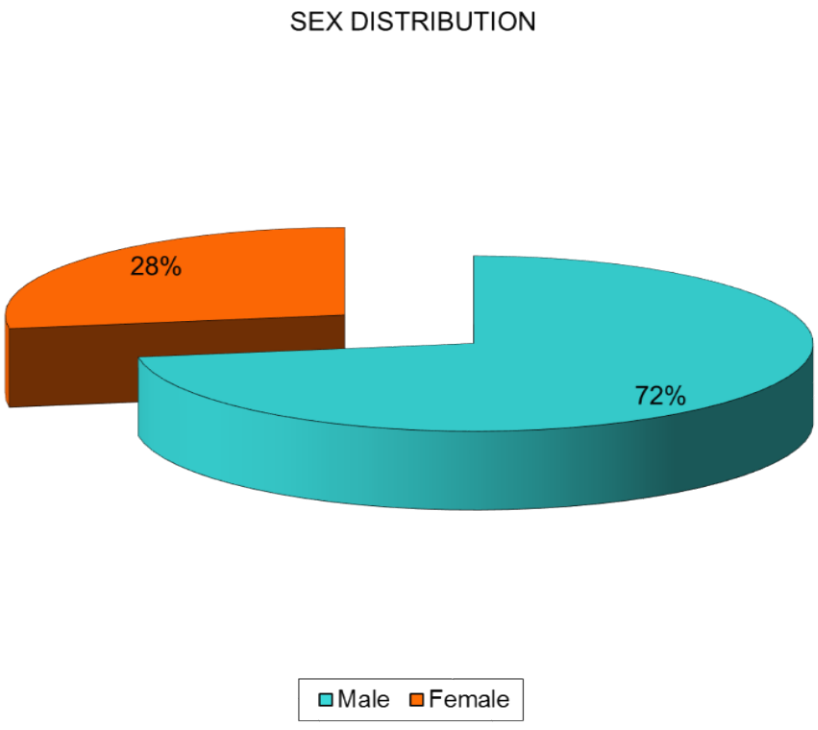
Age in years	**Number of cases**	**Percentage**
< 2	10	25%
2 - 3	22	55%
≥ 4	8	20%
Total	40	100%


AGE DISTRIBUTION
PERCENTAGE
60
50
40
30
20
10
0
25
55
20
< 2
2 - 3
> 4
Percentage

Sex distribution:

Among 40 subjects 29 children are boys and 11 are girls

Gender	Number of cases
Male	29
Female	11



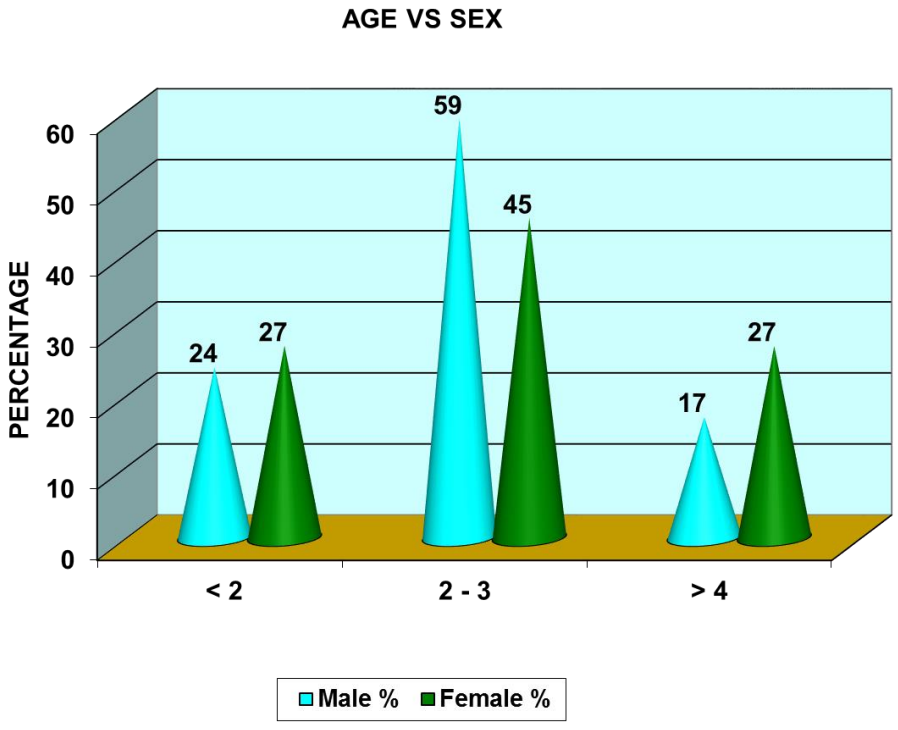
Age vs Sex distribution:

Among male children 24% are <2 years, 59% are 2 to 3 years and 17% are more than or equal to 4 years

Among female children 27% are less than 2 years, 45 % are 2 to 3 years and

27 % are more than or equal to 4 years

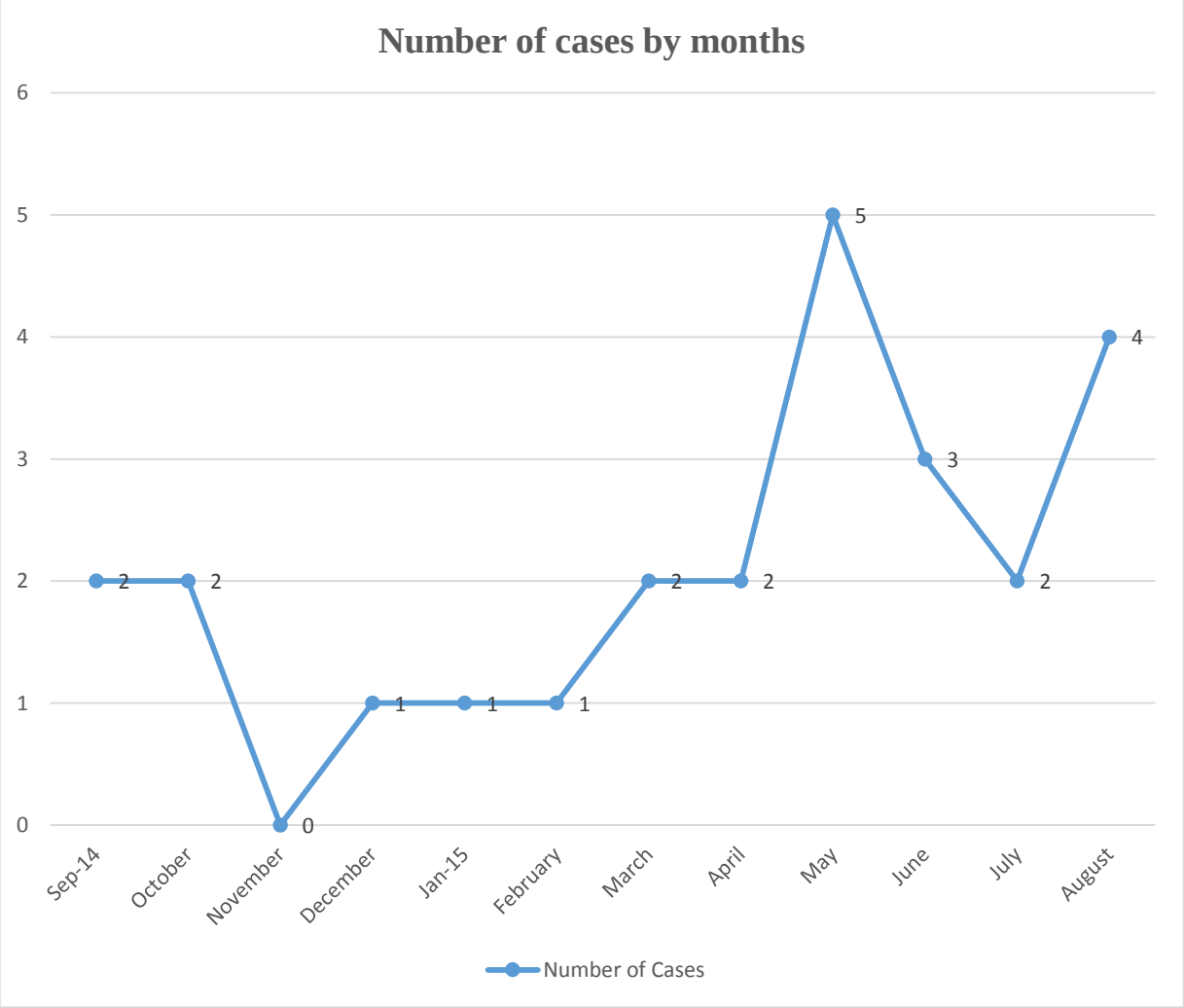
Age in years	Male	Female	Total
< 2	7	3	10
2 - 3	17	5	22
4 - 6	5	3	8
Total	29	11	40



Poisoning occurrence by months:

The study was done form April 2014 to August 2015. A total of 40 were included in the study during this period. To assess the frequency of kerosene poisoning by months throughout one year we have taken the occurrence from September 2014 to August 2015. 25 cases of poisoning occurred in that one year period as given below.

Month	**Number of Cases**
September 2014	2
October	2
November	0
December	1
January 2015	1
February	1
March	2
April	2
May	5
June	3
July	2
August	4



Out of 25 cases occurred over one year, 18 cases (72%) occurred during the summer months of April to September. 7 cases (28%) occurred during the months of October to March which come under monsoon and winter months

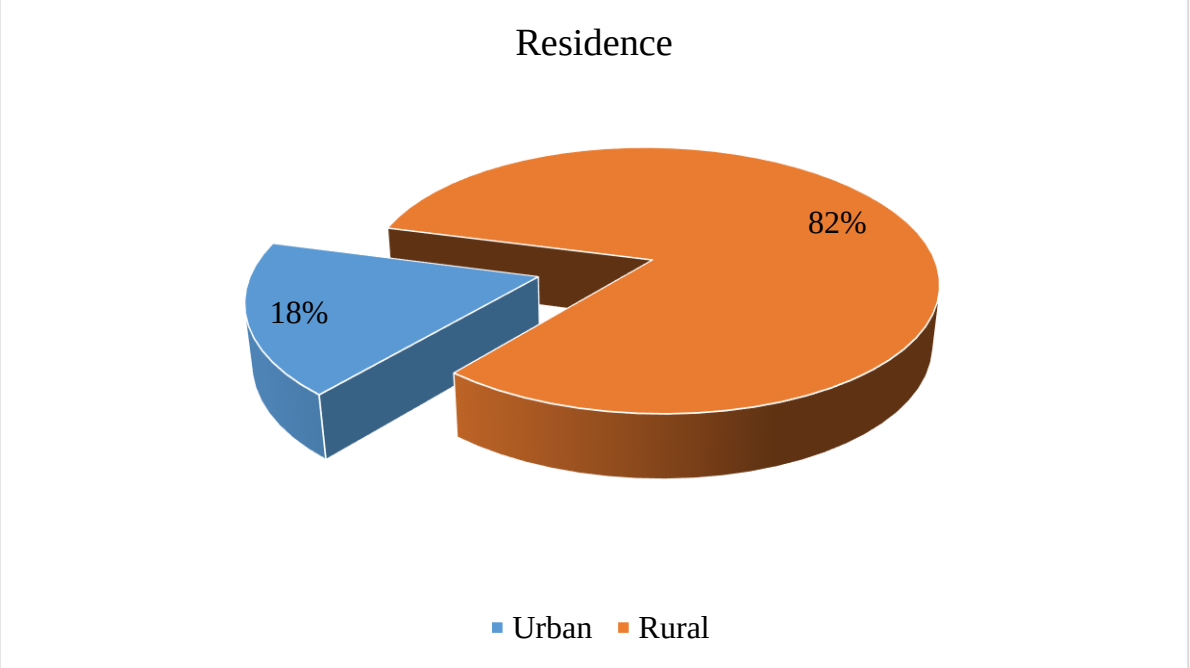
Residence:

Residence of children divided into rural and urban.

Those residing within the corporation and municipality are taken into urban population, and the rest including those residing in panchayat, village areas are taken into rural population.

Out of total 40 subjects 7 were from urban areas and the remaining 33 from rural areas

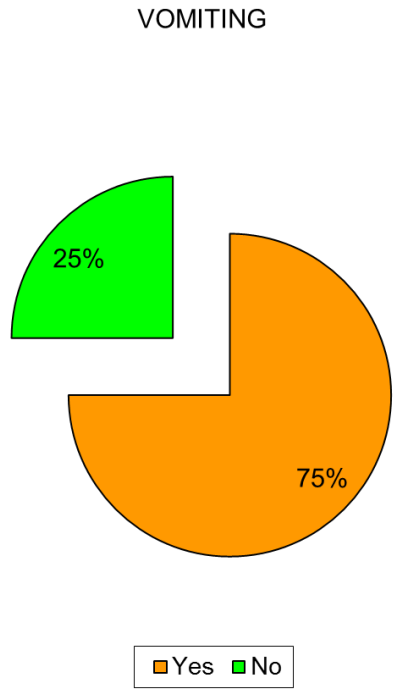
Residence	**Number of subjects**
Urban	7
Rural	33



Vomiting:

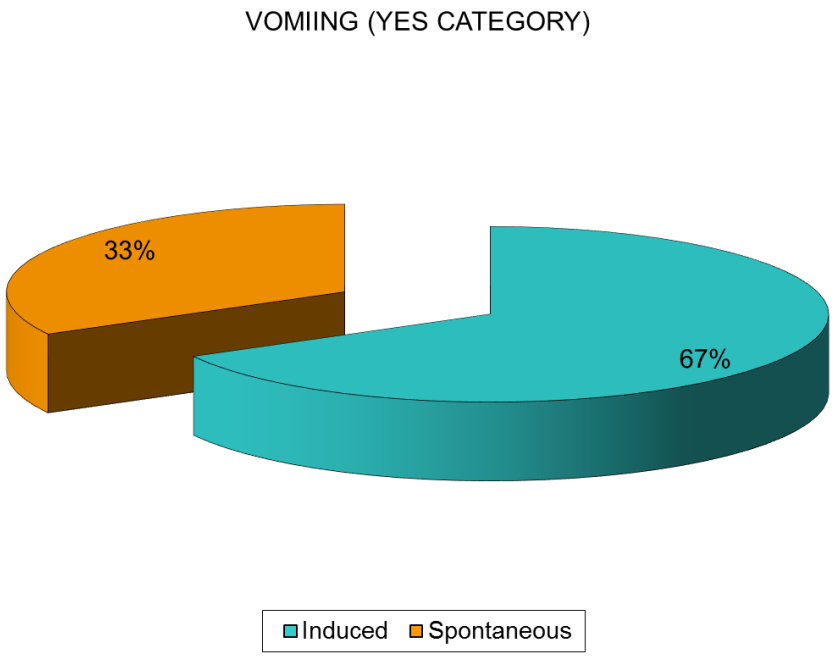
Vomiting is a major risk factor for the development of chemical pneumonitis as the risk of aspiration is high with vomiting. Among the 40 subjects 30 had vomiting.

Vomiting	**Number of cases**
Yes	30
No	10
Total	40



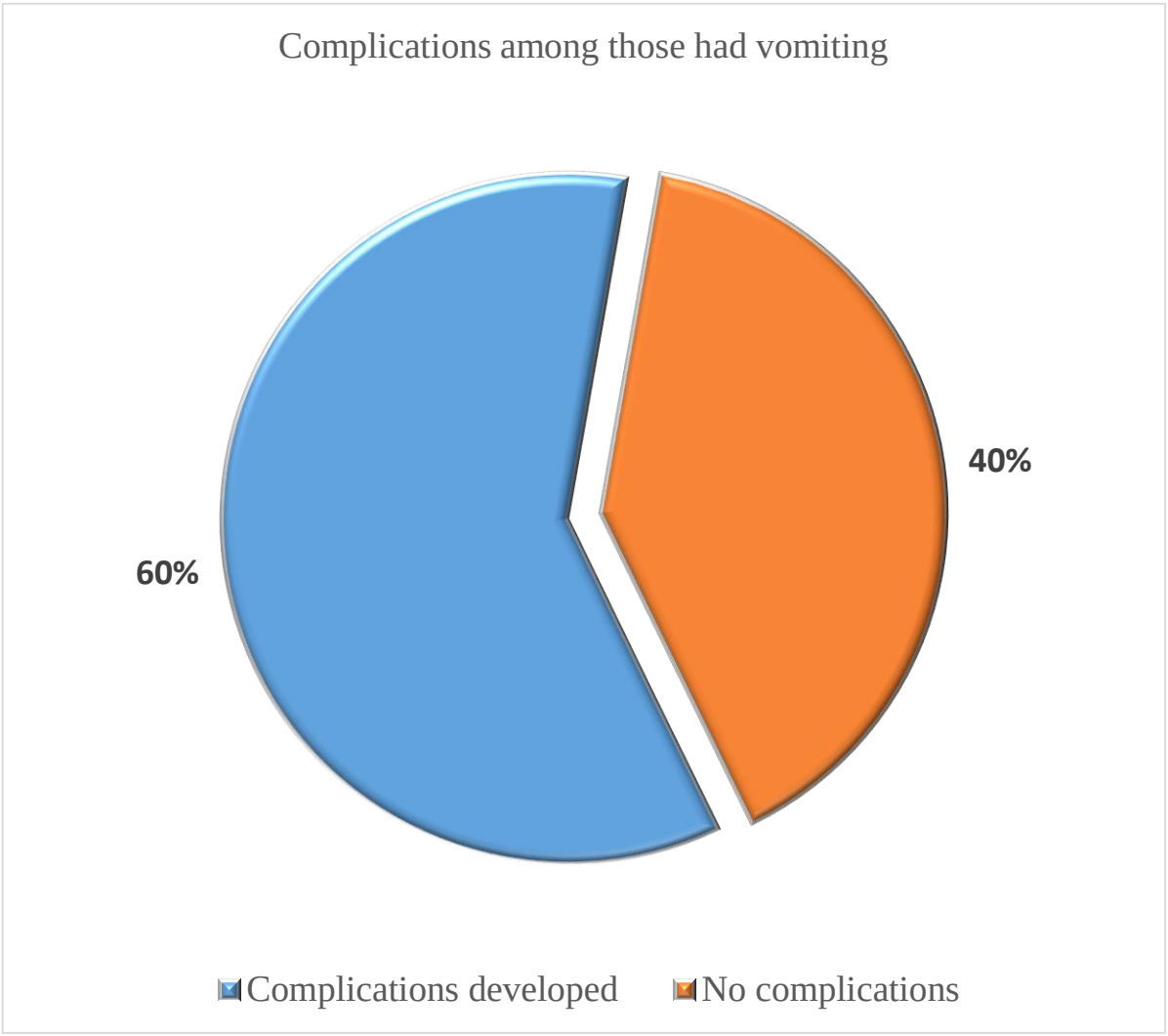
Among the 30 subjects who had vomiting, in 67% of subjects vomiting was induced by parents or care givers and the remaining 33% of subjects had spontaneous vomiting

Vomiting (Yes category)	**Number of cases**
Induced	20
Spontaneous	10
Total	30



Vomiting and development of complications:

30 subjects had vomiting, out of which 18 subjects had developed complications in terms of chemical pneumonitis and death. There was no complications who didn't have vomiting.



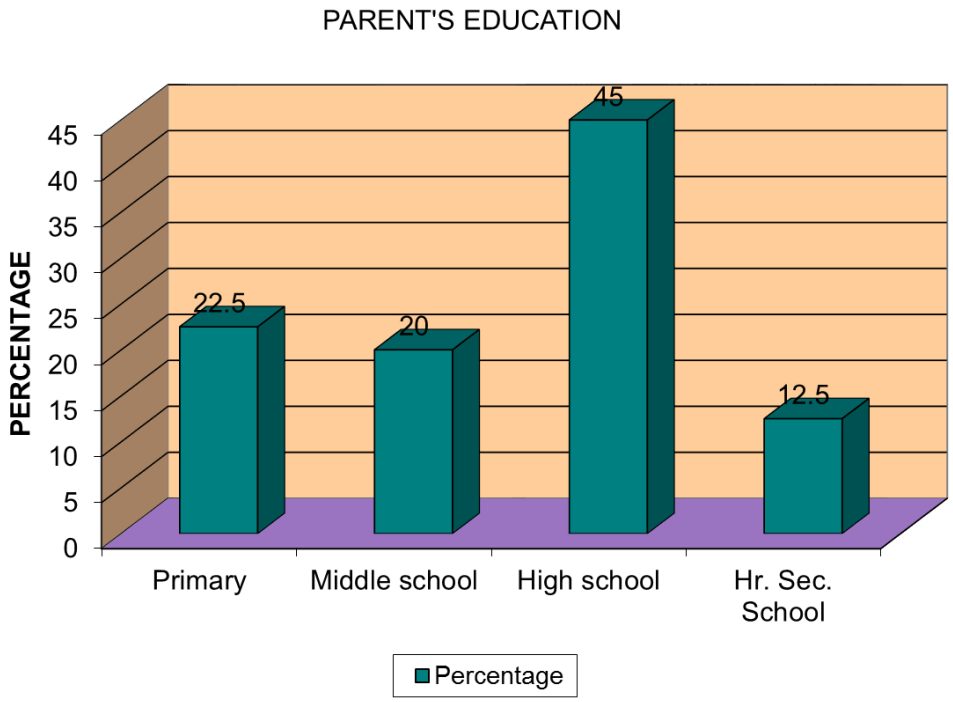
Parent's education:

Higher the qualification among the father and mother is taken for categorization. Parent's education is divided into four categories. Those studied upto 5th Standard are categorized to Primary school, 6th standard to 8th standard are categorized to middle school, 9th standard to 10th standard into High school and 11th and 12th standard into Higher secondary school.

There was no parents who are illiterate, graduates or diploma holders.

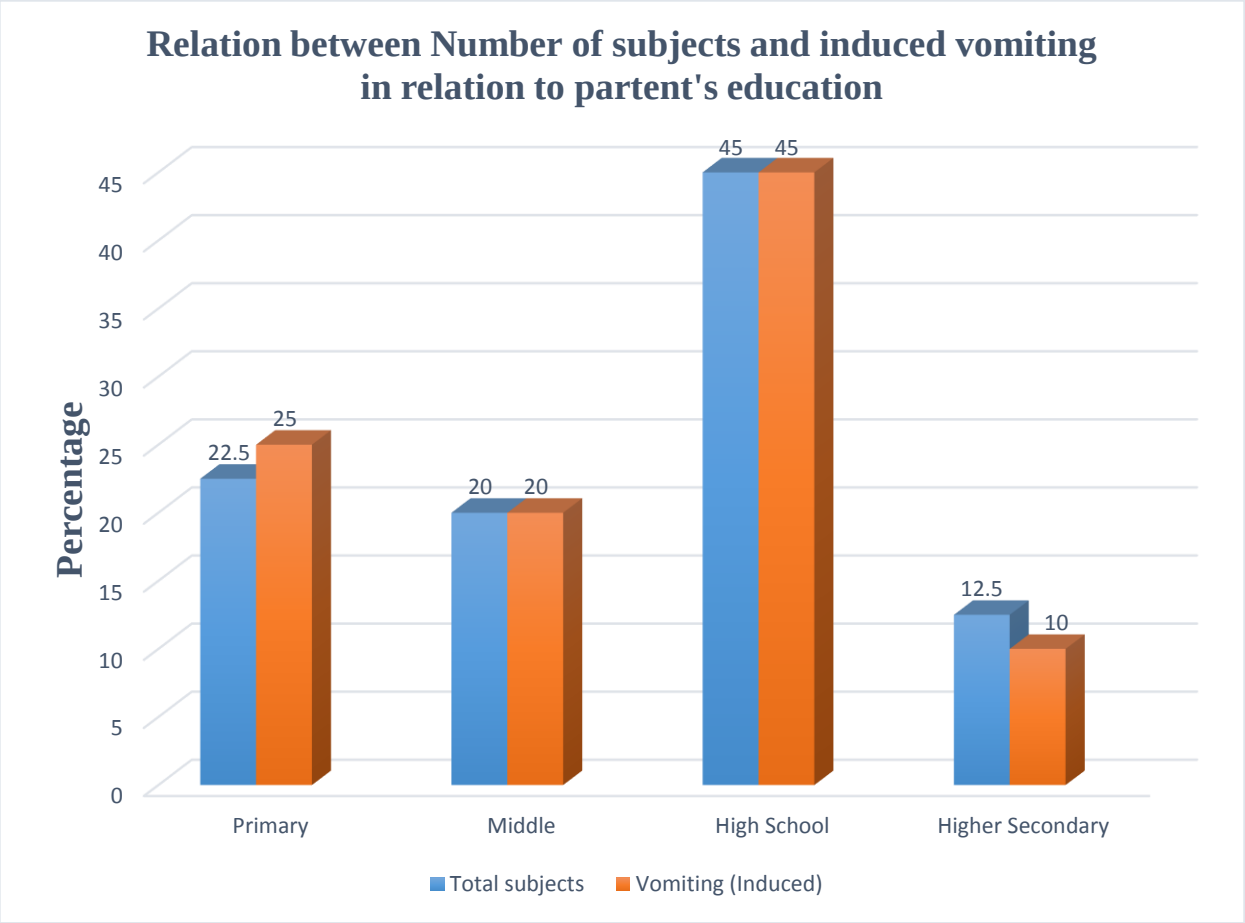
Parent's Education	**Number of cases**	**Percentage**
Primary school	9	22.5%
Middle school	8	20%
High school	18	45%
Higher Secondary School	5	12.5%
Total	40	100%

Parents of 45% of subjects were studied upto High school, 22.5% were completed primary schooling, 20% upto middle school and 12.5% were studied upto Higher Secondary.



Parent's education is analysed in relation to the children who had induced vomiting. Because by inducing vomiting child would have more chance of developing pulmonary complications. Expected is that those with higher education wouldn't induce vomiting. But the distribution of those who had induced vomiting among the parents education categorization was in relation to the distribution of the number of subjects.

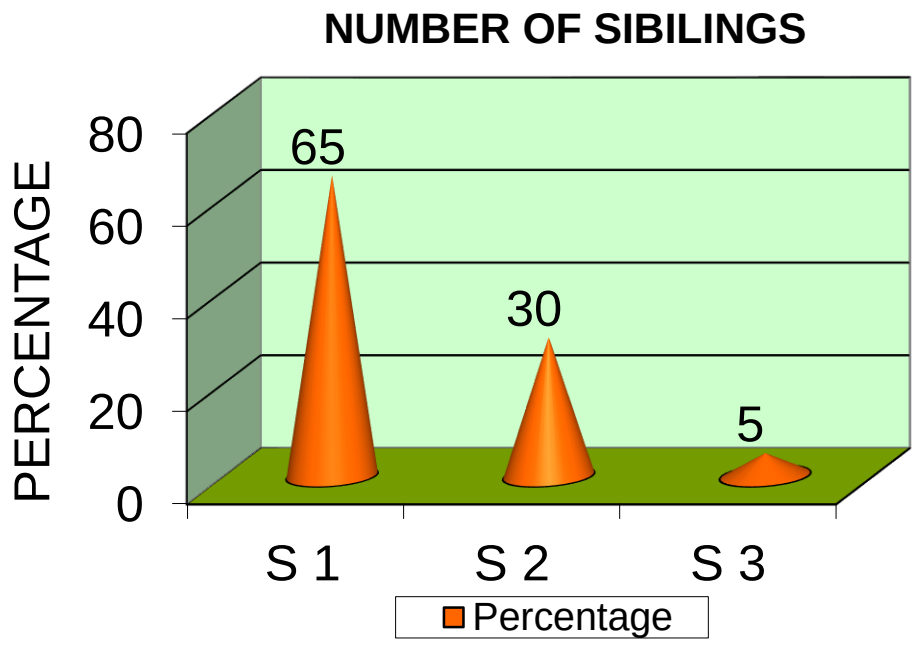
Parent's education	Total subjects (Percentage)	Vomiting (Induced) (Percentage)
Primary	22.5	25
Middle	20	20
High School	45	45
Higher Secondary	12.5	10



Number of Siblings:

The subjects were analysed in relation to number of siblings. 26 subjects had one sibling, 12 subjects had two siblings, 2 subjects had 3 siblings and there was no subjects with four or more siblings

Number of siblings	**Number of cases**
1	26
2	12
3	2
≥ 4	0
Total	40



Storage of kerosene:

The kerosene poisoning occurred at home in all the 40 cases.

Among the poisoning cases the purpose of kerosene at home and the container used to store the kerosene were elicited.

Among the 40 cases, kerosene used for cooking purpose in 26 cases (65%) and used for heating water at home in the remaining 14 (35%) cases.

In all cases the kerosene was stored in the kitchen.

The kerosene was stored in water bottles in 32 (80%) cases and in soft drink bottles in 8 cases (20%). In all cases the containers were placed in reachable places like on the floor or in the lower shelves of open cup-board.

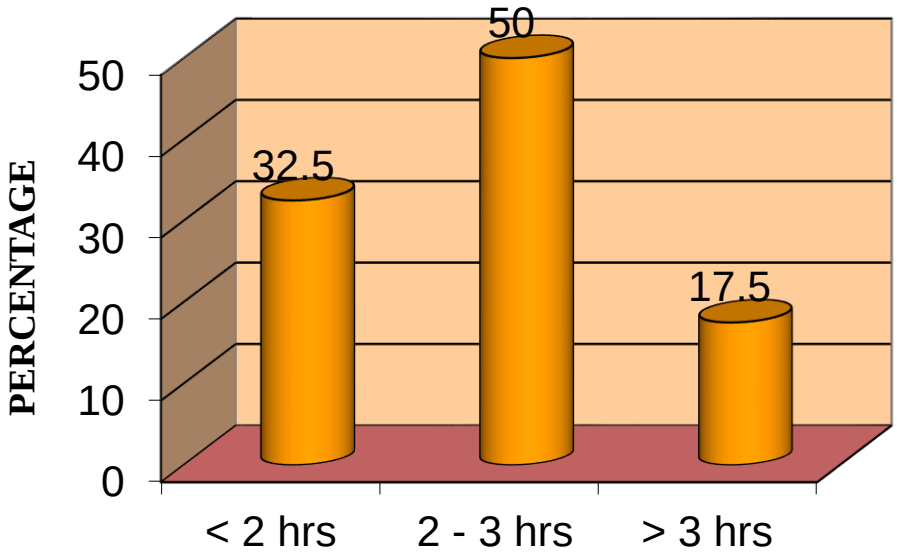
Duration between Kerosene ingestion and hospitalization:

Among the 40 total subjects 13 cases admitted in the hospital within 2 hours after kerosene ingestion. 20 subjects admitted in 2 to 3 hours and 7 subjects admitted after three hours of ingestion.

Duration between exposure and hospitalization ranged from half an hour to 13 hours with mean of two and half hours and standard deviation of two hours

Time passed since exposure	Number of cases
< 2 hrs	13
2 - 3 hrs	20
> 3 hrs	7

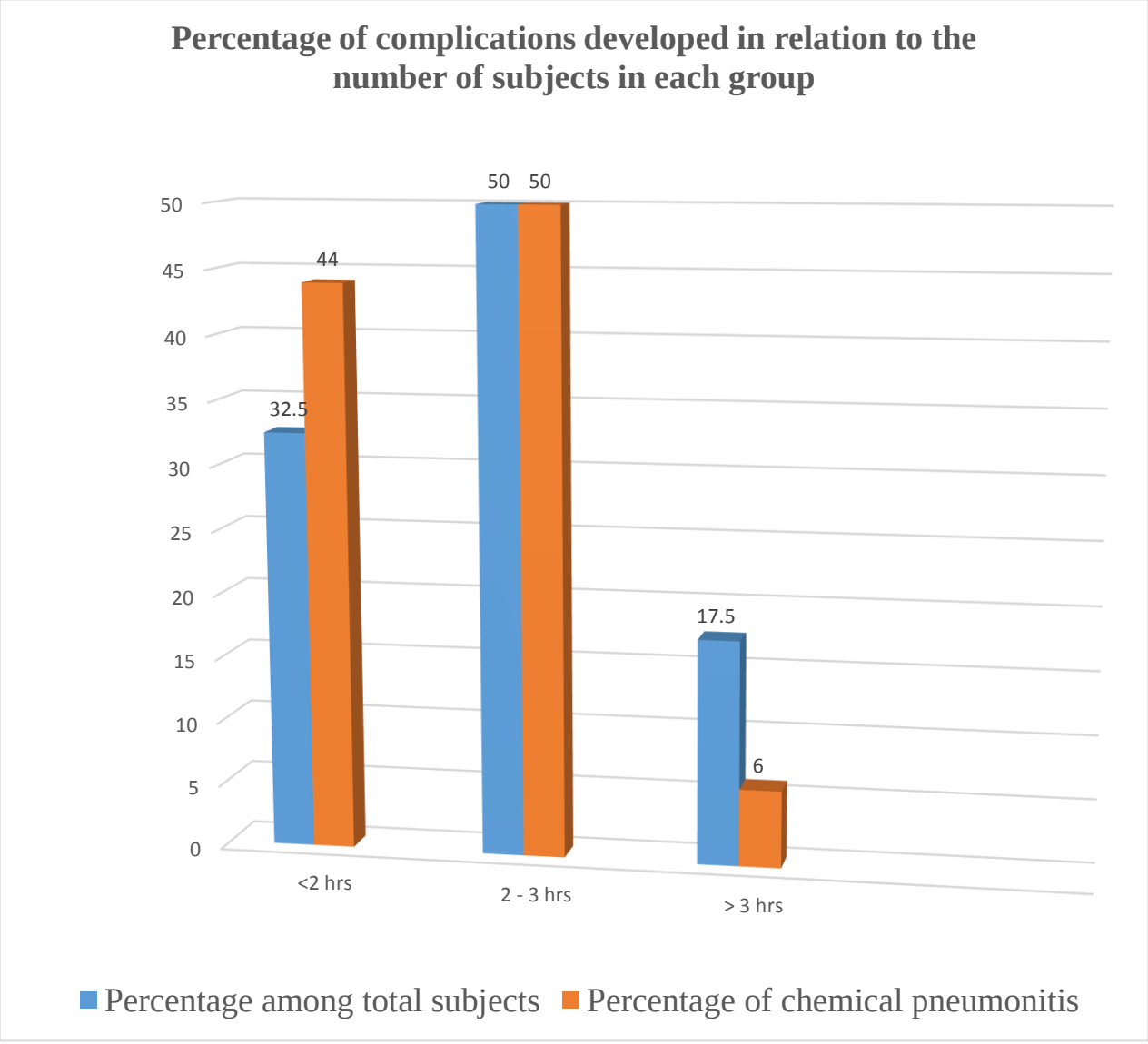
TIME PASSED SINCE EXPOSURE



The total number cases in each duration group is compared to number of cases who developed complications in terms of chemical pneumonitis and death.

It showed that those presented earlier were developed complications more in relation to those who presented earlier.

Time passed since exposure	Number of cases	Number of cases developed complications
< 2 hrs	13	8
2 - 3 hrs	20	9
> 3 hrs	7	1



Complications developed:

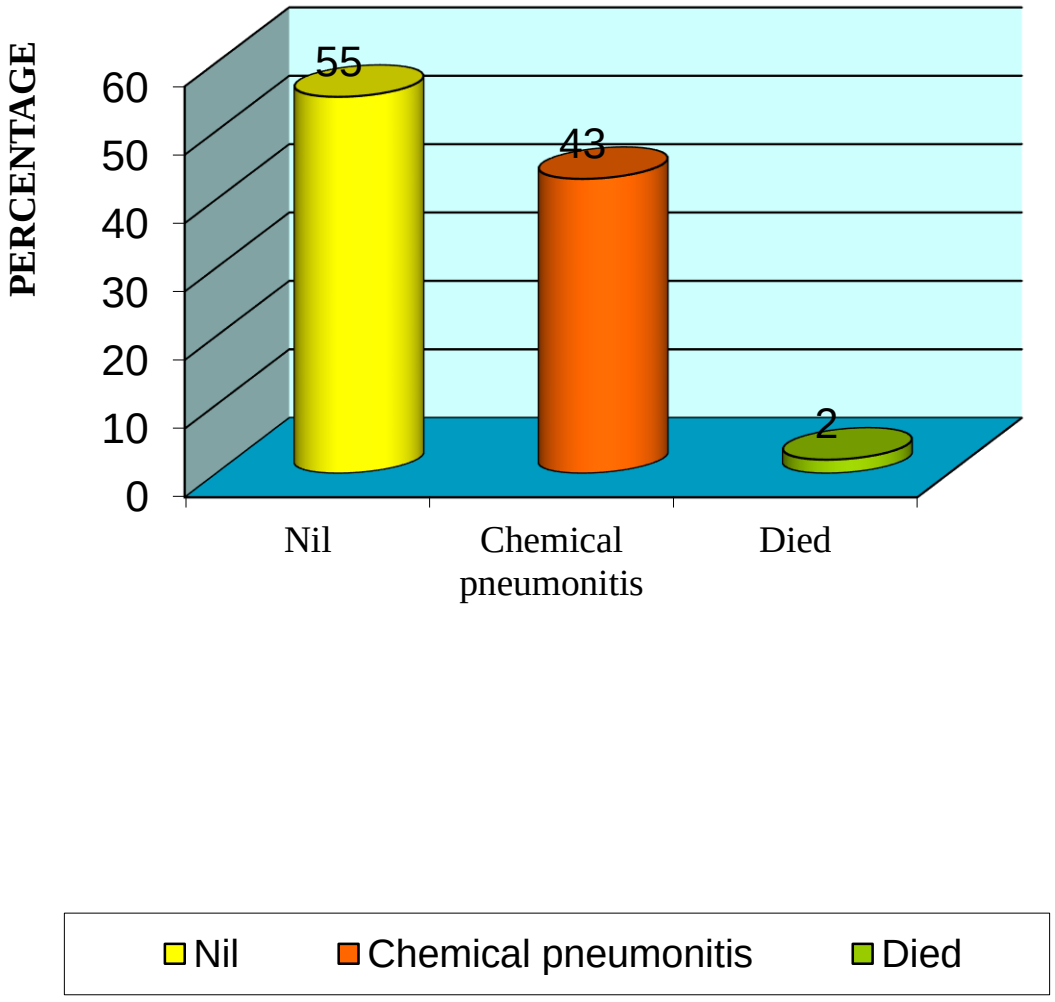
Complications of kerosene ingestion is analysed in terms of development of chemical pneumonitis with the X – Ray findings, ICU admissions, need of ventilator support and death.

Among the total 40 subjects 22 subjects developed no complications, 17 subjects developed Chemical pneumonitis and 1 subject needed ventilator support and subsequently died.

The subject who had died presented to the hospital 3 hours after ingestion and died within 3 hours of admission, i.e. within 6 hours after ingestion. That child was referred another hospital.

Complications	**Number of cases**
Nil	22
Chemical pneumonitis	17
Death	1
Total	40

COMPLICATIONS



Scoring system for Hydrocarbon poisoning:

Subjects were assessed by the scoring system for Hydrocarbon poisoning at the time of admission and then every two hours upto eight hours. The maximum score attained is taken for the study purpose.

Score were assessed by the following parameters:

- Cyanosis
- SpO_2 level
- CNS based on the consciousness
- Pulmonary involvement based on auscultatory findings
- Respiratory distress
- History of vomiting

Total score for the scoring system is 15. The total score is divided into 4 categories as Score 0 to 4, 5 to 8, 9 to 11 and 12 to 15 and following outcome was expected

- **Score 0 to 4:** Excellent prognosis in terms of short duration of admission and no complications
- **Score 5 to 8:** complete cure in terms of no complications developed
- **Score 9 to 11:** Development of chest complications (Chemical pneumonitis) and longer duration of stay

- **Score 12 to 15:** Bad prognosis in terms of ICU admission, ventilator use and may be death

The following observations obtained for the individual parameters of the Hydrocarbon poisoning score

Cyanosis:

Out of 40 subjects 39 subjects had absence of cyanosis (score 0) and one subject had cyanosis (Score 3).

Cyanosis (Score)	Number of cases	Percentage
0	39	97.5%
3	1	2.5%
Total	40	100%

SpO2 level:

19 subjects had SpO2 >95% (Score 0), 20 subjects had SpO2 81% to 95% (score 1) and 1 subject had SpO2 <60% (score 3). There was no subject with SpO2 61% to 80% (score 2)

SpO_2 level (score)	Number of cases	Percentage
0	19	47.5%
1	20	50%
2	0	0%
3	1	2.5%
Total	40	100%

CNS:

Out of 40 subjects, 8 subjects had no disturbance in consciousness (score 0), 16 subjects had drowsiness (score 1), 13 subjects had coma responding to verbal and painful stimuli (score 2) and 3 subjects had deep coma (score 3)

CNS (score)	Number of cases	Percentage
0	8	20%
1	16	40%
2	13	32.5%
3	3	7.5%
Total	40	100%

Pulmonary involvement:

Based on the auscultatory findings 14 subjects had wheeze (score 1) and 26 subjects had crepitation and or diminished air entry (score 2)

Pulmonary involvement (score)	Number of cases	Percentage
0	0	0%
1	14	35%
2	26	65%
Total	40	100%

Respiratory distress:

Based on respiratory distress 2 subjects had only cough (score 1), 20 subjects had cough and tachypnea (score 2) and 18 subjects had cough, tachypnea and accessory muscle use (score 3)

Respiratory distress (score)	Number of cases	Percentage
0	0	0%
1	2	5%
2	20	50%
3	18	45%
Total	40	100%

Vomiting:

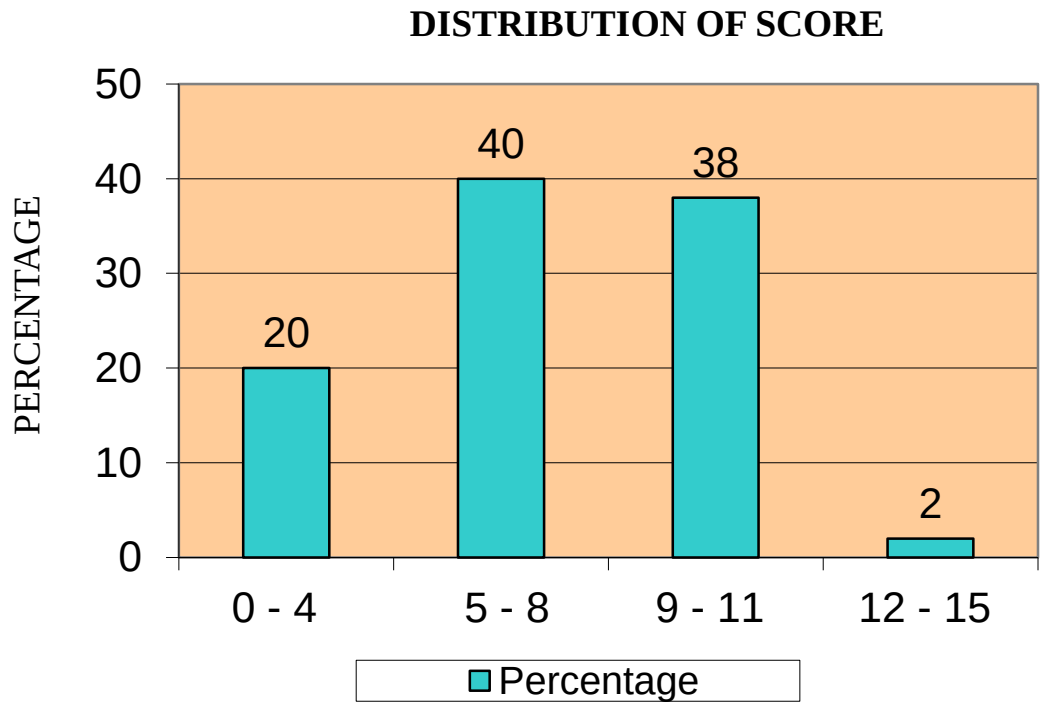
Among 40 subjects, 30 subjects had history of vomiting following kerosene ingestion (score 1) and 10 subjects had no vomiting (score 0)

Vomiting (Score)	Number of cases	Percentage
0	10	25%
1	30	75%
Total	40	100%

Distribution of score:

The subjects are categorised into four groups. 8 subjects had score 0 to 4, 16 subjects had score 5 to 8, 15 subjects had score 9 to 11 and 1 subject had score 12 to 15

Score	Number of cases
0 - 4	8
5 - 8	16
9 - 11	15
12 - 15	1



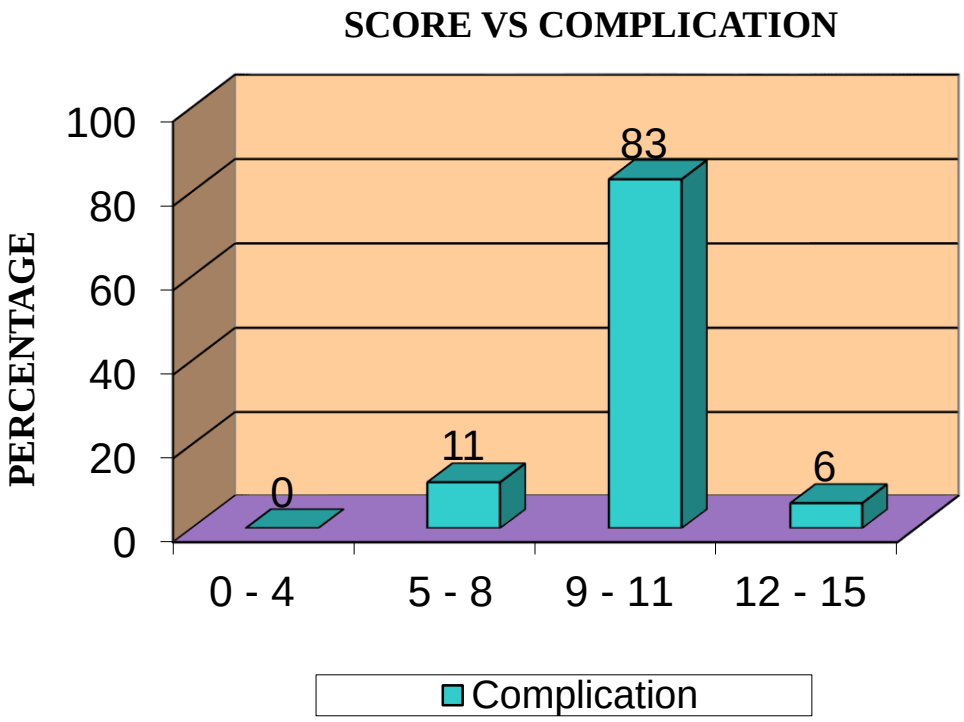
Complications developed:

Complications were assessed in terms of development of chemical pneumonitis, ICU admission, ventilator support and death. Totally 17 subjects developed chemical pneumonitis and one subject needed ventilator support and subsequently died.

The number of complications in each category of score: There was no complication in 0 to 4 score category, 2 subjects developed chemical pneumonitis in score 5 to 8 category, 15 subjects developed chemical pneumonitis in score 9 to 11 category of which 2 subjects needed ICU treatment and one subject had died in the score 12 to 15 category

Score	Complications	Ventilator support
0 – 4	0	No
5 – 8	2	No
9 - 11	15	No
12 - 15	1	yes

11% of subjects who developed complication was in score 5 to 8 category, 83% of subjects who developed complication was in score 9 to 11 category and 6% of subjects who developed complication was in score 12 to 15 category.

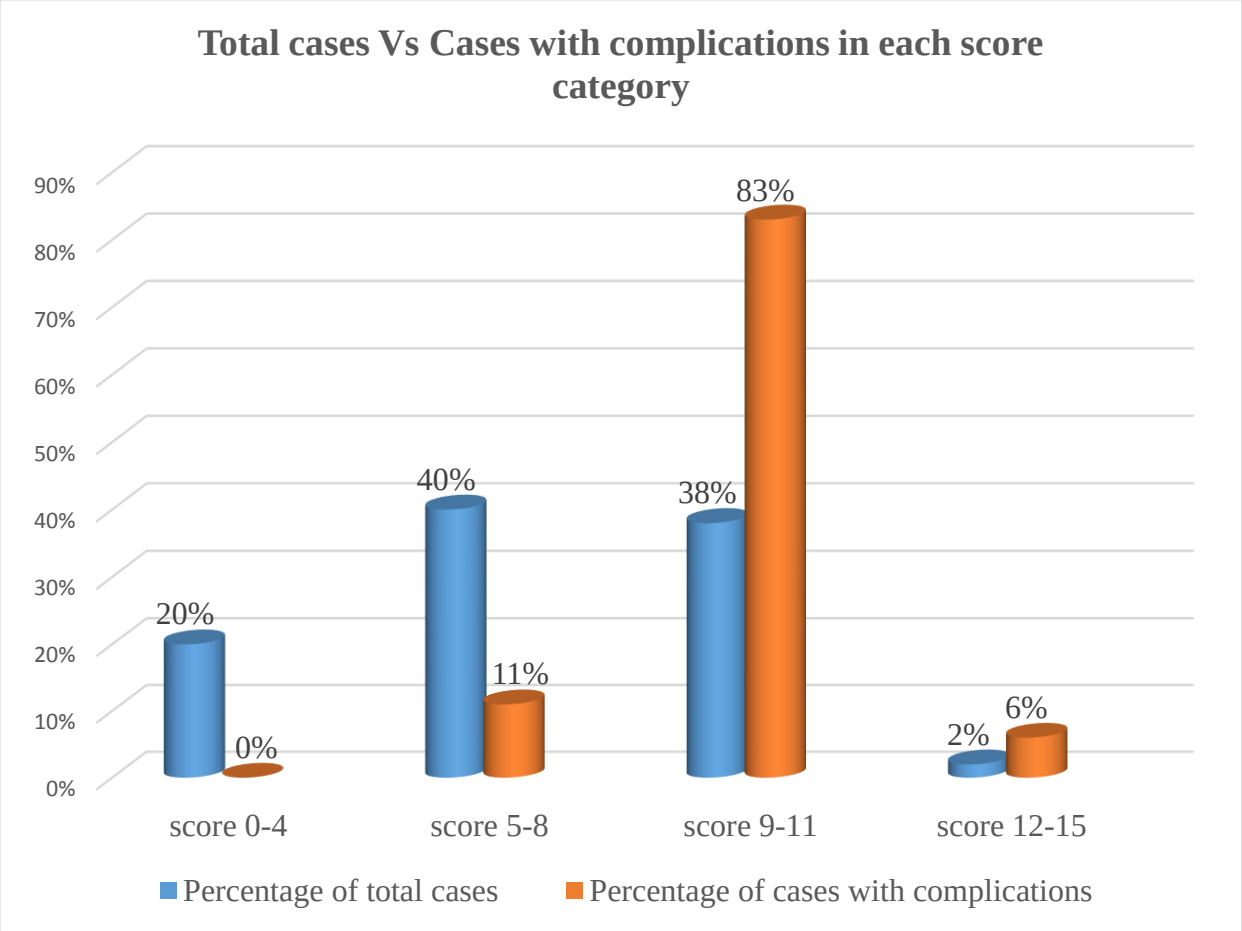


On comparing number of subjects and those developed complications in each score category following result was obtained.

- Score 0-4 had 20% of cases, but none of them developed complications
- Score 5-8 had 40% of cases and had 11% of cases who developed complications
- Score 9-11 had 38% of cases and had 83% of cases who developed complications

- Score 12 -15 had 2% of cases and had 6% of cases who developed complications

Score	Number of cases	Number of cases with Complications
0-4	8	0
5-8	16	2
9-11	15	15
12-15	1	1

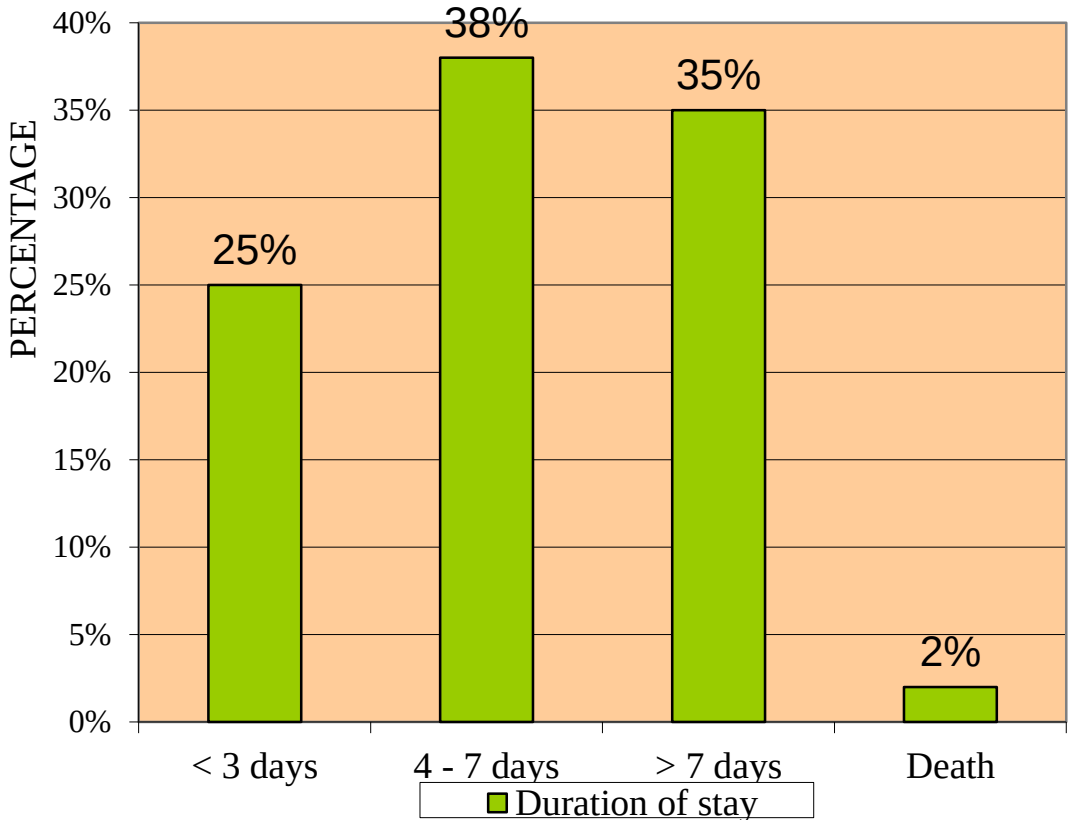


Outcome distribution:

Outcome is assessed by the duration of In-patient treatment and death. The duration of hospital stay is divided into 4 categories as less than or equal to 3 days, 4 to 7 days and more than 7 days. The number of cases in each group is as below

Duration of In-patient treatment	Number of cases
≤ 3 days	10
4 - 7 days	15
> 7 days	14
Death	1

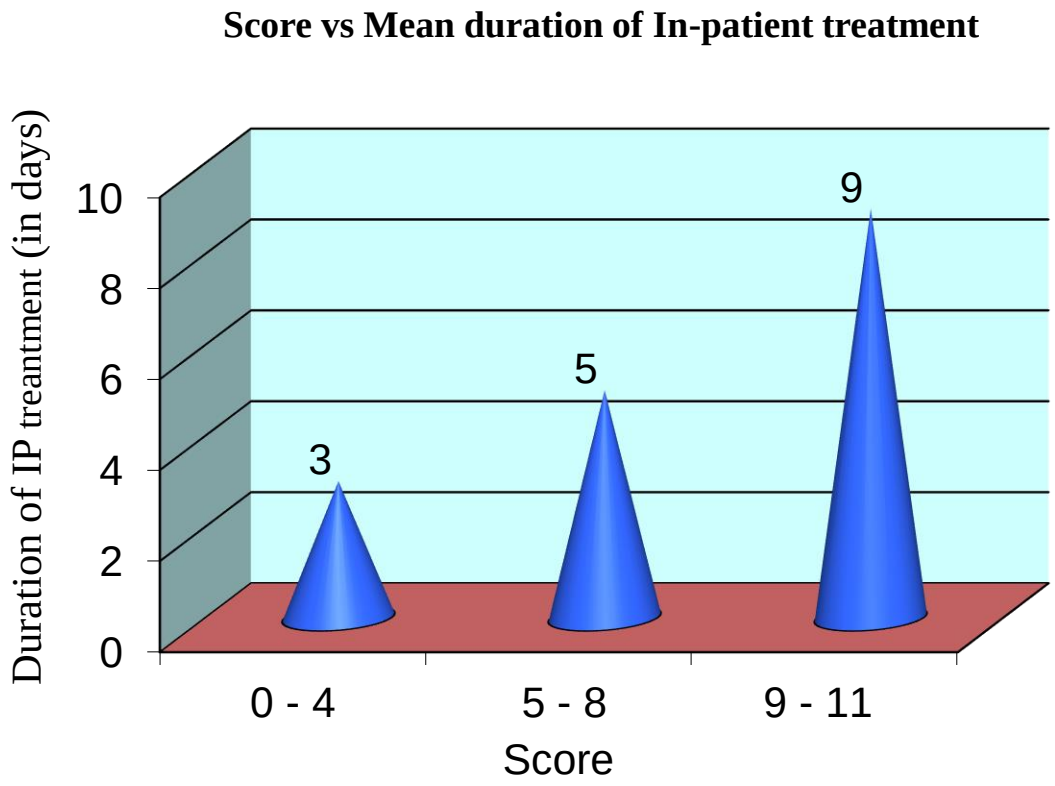
OUTCOME DISTRIBUTON



The duration of In-patient (IP) treatment is each score category is given below

- Score 0-4: The duration of IP treatment had ranged from 2 days to 3 days with mean of 3 days (corrected to full number without decimal)
- Score 5-8: The duration of IP treatment had ranged from 4 days to 8 days with mean of 5 days
- Score 9-11: The duration of IP treatment had ranged from 6 days to 11 days with mean of 9 days

- Score 12-15: The one subject in this category had died in 3 hours of admission



Statistical analysis:

The duration of hospitalization is compared to each score categories and the correlation between these two values is assessed by Correlation coefficient.

Correlation coefficient obtained is 0.849

A correlation coefficient of -1 to 0 means Negative correlation and 0 to +1 means positive correlation. With this value of 0.849 means very high correlation between the score values and the duration of hospitalization and development of complications and death.

DISCUSSION

As discussed in the review of literature accidental poisoning of kerosene is common in children in developing countries like India. Based on the study the following inference were obtained.

Age:

The poisoning occurred mostly in young children with range of one year to 6 years with a mean of two and half years. 80% of children in the study were below 3 years of age.

As discussed earlier the reason for this may be the immature development of olfactory functions in young children. They tend to easily attracted by the colour of the kerosene which is blue in colour in government subsidised supply which is the common source of kerosene for domestic usage in India. In all cases the kerosene was stored in water bottles or soft drink bottles and kept in reachable places at home, particularly in kitchen

The poisoning occurred in 72% of cases in summer months of April to September as the children gets thirsty often and tend to drink the kerosene thinking that it is a water or soft drinks because of the container used.

Sex distribution:

In 72% of cases the poisoning occurred in boys.

On comparing age Vs sex distribution poisoning occurred at highest in boys in 2 to 3 years age group. In all age groups boys are commonly affected.

The reason behind male preponderance may be the higher degree of hyperactivity behaviour among boys[23]

Residence:

82% of children admitted with kerosene ingestion was from rural areas as the kerosene usage is more common in rural population compared to urban population. Kerosene is also used for initiation of fire while cooking with woods in rural areas.

Vomiting:

Vomiting occurred in 75% of cases. Among the cases with history of vomiting 67% were induced by the parents and care givers and the remaining 33% were spontaneous. Aspiration and subsequent development of chemical pneumonitis and other complications are common in vomiting. Among those cases who had vomiting 60% developed complications. There was no complications in children who didn't have vomiting.

Parent's education:

On comparing the parent's education background most of the parent's studied upto high school (45%). Among the parents mother had higher qualification compared to father.

The parent's education is compared to those had vomiting induced by the caregivers. It showed no difference in relation to parent's education. This probably due to parents have little knowledge that the risk of aspiration increases with vomiting. Another possible reason is that vomiting was induced initially by the grandparents.

Quantity of ingestion:

The quantity of kerosene ingestion hadn't taken to assess the outcome as the parents and care givers could not clearly tell the amount of ingestion. Moreover apart from the amount of ingestion, the quantity of kerosene get aspirated produces the complications.

Duration between poisoning and hospitalisation:

Children with poisoning brought to the hospital with a mean of two and half hours and 82% of cases brought to the hospital within 3 hours of exposure.

On comparing the duration between poisoning and hospitalisation with the development of complications, it showed higher percentage of score

and developing complication in those presented earlier. It is due to those with higher respiratory distress and CNS findings were brought to this tertiary care centre directly and those presented late were with less respiratory distress taken to nearby hospitals and later referred here.

Complications developed:

18 (45%) subjects developed complications of which 17 persons had chemical pneumonitis and 1 subject need ventilator support and subsequently died.

Scoring system for hydrocarbon poisoning:

The subjects were assessed by Scoring system for hydrocarbon poisoning on admission and every 2 hours upto 8 hours. Maximum score was attained at the end of 8 hours in all cases. The scoring system could be assessed bedside.

Among the manifestation of symptoms and signs, Respiratory involvement was more common than CNS involvement. The scoring system also has two parameters for the respiratory manifestations while single parameter for CNS manifestation to give more weightage to respiratory manifestations. The parameter ‘Pulmonary involvement’ is based on auscultatory findings and the parameter ‘Respiratory distress’ deals with the respiratory rate and accessory muscle use.

The score was compared with the complications developed during hospitalization. It was found that higher proportion of developing complications in the score categories 9 to 11 and 12 to 15. Only one subject within the category of score 12 to 15 had died.

Also the duration of hospitalization was compared with the score. The mean duration of hospitalization was 3 days with score 0 to 4, 5 days with score 5 to 8 and 9 days with score 9 to11. So this scoring system is very useful in predicting the illness severity and clinical outcome with kerosene poisoning.

The statistical analysis showed a correlation coefficient of 0.849 which is a very high correlation. Maximum value of correlation coefficient is 1.0

CONCLUSION

The Scoring system for hydrocarbon poisoning can be applied bedside and is useful in assessing the clinical severity and outcome of children with kerosene poisoning.

Accidental kerosene poisoning is common among the children in developing countries like India. Young children less than 3 years and boys are commonly affected. Poisoning occurs commonly during summer.

Kerosene poisoning is common among rural population because of wide usage there compared to urban population.

Vomiting is an important risk factor for the development of chemical pneumonitis and the duration of hospitalization.

Common presentation with kerosene ingestion is the respiratory distress and second common presentation is with CNS manifestations.

Parents education in important in prevention of accidental poisoning of kerosene.

BIBLIOGRAPHY

1. Poisoning in children: Indian scenario. Datta AK, Seth A, Goyal PK, et al. Indian J Pediatr 1998; 65: 365-370.
2. Litovitz T. Hydrocarbon ingestions. Entechnology 1983; 62:142-147.
3. HYDROCARBON AND RELATED COMPOUNDS POISONING, Utpal Kant Singh, Prasad R, Gaurav A, Indian Journal of Practical Pediatrics 2009; 11(1) : 16
4. "Hydrocarbon poisoning in children: A 5 year retrospective study". Lifshitz, M.; Sofer, S. and Gorodischer, R. (2003): Wilderness and Environmental Medicine, 14:78-82.
5. "Paraffin (kerosene) poisoning in under five children : A problem of developing countries". Tshiamo, W. (2009) : International Journal of Nursing Practice, 15: 140-144.
6. "The study of etiological and demographic characteristics of acute household accidental poisoning in children - a consecutive case series study from Pakistan". B Manzar, N.; Saad, S. M. A.; Manzar, B. and Fatima, S. S. (2010): MC pediatrics, 10: 28-31.
7. "An intervention to reduce kerosene related burns and poisoning in low income south African communities". David, S. C.; Dehran, S.; Jennifer, S.; Azor, H. S. K. and Phumla, H. (2009) Health Psychology, 28(4): 493-500.

8. Common childhood poisonings and their outcome in a tertiary care centre in Delhi. Brata Ghosh V, Jhamb U, Singhal R, Krishnan R., Indian J Pediatr.2013 Jun;80(6):516-8.
9. Toxicological profiles for fuel oils. Risher, J. F. and Rhodes, S. W. (1995). US Department of Health and Human Services
10. Accidental kerosene ingestion: a 3-year prospective study. Dudin, A. A., Rambaud-Cousson, A., Thalji, A., Jubeh, II, Ahmad, H. M. and Libdeh, B. A. (1991). Ann Trop Paediatr 11, 155-61.
11. Accidental kerosene poisoning in infants and children. Santhanakrishnan, B. R. and Chithra, S. (1978). Indian J Pediatr 45, 265-73
12. "Predictors of outcome in children with hydrocarbon poisoning receiving intensive care". Jayashree, M.; Singhi, S. and Gupta, A. (2006): Indian Pediatric Journal, 43: 715-719.
13. Clinical profile of children with kerosene aspiration. Venkatesh C, Sriram P, Adhisivam B, Mahadevan S., Trop Doct. 2011 Jul;41(3):179-80.
14. Hydrocarbon poisoning in children: a 5-year retrospective study, Lifshitz M1, Sofer S, Gorodischer R, Wilderness Environ Med. 2003 Summer;14(2):78-82.
15. Kerosene oil poisoning in children : A Hospital-based prospective study in Sri Lanka, G.N. Lucas, Indian J Pediatr 1994; 61 : 683-687

16. A proposed new scoring system for Hydrocarbon poisoning. Ahmed Abdel Raouf et al., Mansoura J. Forensic Med. Clin. Toxicol. Vol. XX, No. 1, Janurary 2012
17. Clinical and Therapeutic Aspects of Kerosene Poisoning: A series of 200 cases. B.J. Baldachin, R.N. Melmed, BMJ, 1964, 2, 28-30
18. "Kerosene poisoning in childhood : A 6 year prospective study at the Princess Rahmat teaching hospital". Shotar, A. M. (2005) : Neuroendocrinolgy Letters, 26 (6): 835-838
19. Spencer's pathology of the lung, Philop Hasleton, Douglas B. Flieder, Sixth edition
20. Miscellaneous hydrocarbon solvents. Bebarta, V. and DeWitt, C. (2004). Clin Occup Environ Med 4, 455-79
21. "Accidental paraffin poisoning in Kenya children". Lang, T.; Thuo, N. and Akech, S. (2008): Tropical Medicine Internal Health, 13:845-847
22. Prognostic score for kerosene oil poisoning. Gupta P, Singh RP, Murali MV, Sharma PP. Indian Pediatr. 1992 Sep;29(9):1109-12.
23. Sex and age differences in attention-deficit/hyperactivity disorder symptoms and diagnoses. Ramtekkar UP, Reiersen AM, Todorov AA, Todd RD. J Am Acad Child Adolesc Psychiatry. 2010 Mar;49(3):217-28.e1-3.
24. Indian J Pediatr. 2013 Jun;80(6):516-8. doi: 10.1007/s12098-012-0879-5. Epub 2012 Sep 2.

25. Kerosene poisoning in childhood: a 6-year prospective study at the Princess Rahmat Teaching Hospital. Shotar AM, Neuro Endocrinol Lett. 2005 Dec;26(6):835-8.

S.No	Name	Age (in years)	Sex	In patient (IP) Number	Residence	Parents education	Number of siblings	Past H/O any medical illness	Duration between exposure and admission	H/O Vomiting	Sc
											On admission
1	Bharathi	3	M	15514/14	Rural	Middle	2	Nil	2hr	yes (spontaneous)	6
2	Joseph	2	M	17874/14	Urban	High school	1	Nil	1hr	Yes (induced)	4
3	Nivesh	4	M	19668/14	Rural	Primary	3	Nil	1.5hr	Yes (induced)	6
4	Sarathy	2	M	21276/14	Rural	High school	1	Nil	2hr	Yes (induced)	6
5	Jebaraj	1.5	M	22155/14	Rural	Primary	1	Nil	3hr	yes (spontaneous)	6
6	Isakkivel	2	M	25698/14	Rural	High school	1	Nil	4hr	no	2

7	Parthibha	1.5	M	28799/14	Rural	Higher secondary	1	Nil	2hr	yes (spontaneous)	7
8	Velmurugan	1.5	M	32566/14	Rural	Primary	1	Nil	1hr	yes (spontaneous)	6
9	Santhiya	5	F	33043/14	Rural	Higher secondary	2	Nil	2 hr	yes (spontaneous)	6
10	Velpaunraj	5	M	36176/14	Rural	Middle	3	Nil	1 hr	no	2
11	Vishnu	1.25	M	48031/14	Urban	High school	2	Nil`	1/2 hr	no	3
12	Jebaraj	1.5	M	50340/14	Rural	High school	2	Nil	1 hr	Yes (induced)	5
13	Mutheesh	1.5	M	51301/14	Urban	High school	2	Nil	1 hr	yes (induced)	6

14	Esakkiraja	3	M	55785/14	Rural	Middle	1	Nil	1.5hr	Yes (induced)	5
15	Vansha	2	M	60024/14	Rural	High school	2	Nil	2hr	no	4
16	Akinas	5	M	72684/14	Rural	Primary	1	Nil	2hr	Yes (induced)	5
17	Sahasra	1	F	1153/15	Rural	Middle	1	Nil	4hr	no	5
18	Aravinth	3	M	10401/15	Rural	Middle	1	Nil	2hr	yes (spontaneous)	6
19	Sujith Gowtham	2	M	10924/15	Rural	Primary	1	Nil	3hr	yes (spontaneous)	14
20	Kishore	4	M	11163/15	Rural	High school	1	Nil	3hr	Yes (induced)	5
21	Pranav	4	M	16570/15	Urban	High school	2	Nil	1hr	Yes (induced)	4
22	Karthik	3	M	18498/15	Rural	Primary	1	Nil	2hr	No	3

23	Surya narayanan	2	M	23702/15	Rural	Higher secondary	1	Nil	4hr	Yes (induced)	6
24	Selva subashini	1.5	F	24051/15	Urban	High school	1	Nil	2hr	Yes (induced)	5
25	Esakki karthi	2	M	25631/15	Rural	High school	1	Nil	1hr	Yes (induced)	7
26	Makesh	2	M	27679/15	Rural	High school	1	Nil	2hr	yes (spontaneous)	7
27	Kannan	3	M	31119/15	Rural	Middle	2	Nil	3hr	Yes (induced)	6
28	Indhusudha	2	F	31782/15	Rural	Higher secondary	1	Nil	3hr	no	3
29	Keerthiga	2	F	32188/15	Rural	High school	1	Nil	2hr	yes (spontaneous)	4
30	Keerthavan	1.5	M	34240/15	Rural	Higher secondary	1	Nil	2hr	Yes (induced)	3

31	Parthiban	3	M	37825/15	Urban	Primary	2	Nil	2hr	Yes (induced)	4
32	Palaniammal	6	F	38512/15	Rural	Primary	2	Nil	13hr	Yes (induced)	9
33	Inslika	1.5	F	41434/15	Rural	High school	1	Nil	1hr	Yes (induced)	6
34	Muthulakshmi	4	F	44238/15	Rural	High school	1	Nil	2hr	no	4
35	Dhansika	2	F	49465/15	Rural	High school	1	Nil	3hr	no	6
36	Ajitha	3	F	50378/15	Rural	High school	2	Nil	4hr	no	2
37	Sankar selvam	2	M	51279/15	Urban	Middle	1	Nil	1hr	Yes (Induced)	4
38	Sudalai muthu	2	M	51456/15	Rural	High school	1	Nil	4hr	yes (spontaneous)	5
39	Srimathi	2	F	55087/15	Rural	Middle	1	Nil	4hr	Yes (induced)	6
40	Abinesh	3	M	55650/15	Rural	Primary	2	Nil	1.5hr	Yes (induced)	6

ore by duration of exposure				Final score									
2 hr	4 hr	6 hr	8 hr	Cyanosis	SpO2 level	CNS	Pulmonary involvement	Respiratory distress	Vomiting	Total score	Complications	ICU treatment	Mechanical Ventillation
6	6	8	9	0	1	2	2	3	1	9	Chemical pneumonitis	No	No
4	5	6	6	0	1	1	1	2	1	6	Nil	No	No
6	8	9	9	0	1	2	2	3	1	9	Chemical Pneumonitis	No	No
6	6	8	9	0	1	2	2	3	1	9	Chemical Pneumonitis	No	No
	6	6	6	0	0	1	2	2	1	6	Nil	No	No
	2	2	2	0	0	0	1	1	0	2	nil	No	No

7	8	10	10	0	1	3	2	3	1	10	Chemical Pneumonitis	yes (1 day)	No
6	6	8	8	0	1	1	2	3	1	8	Chemical pneumonitis	No	No
6	8	9	9	0	1	2	2	3	1	9	Chemical pneumonitis	No	No
2	2	2	2	0	0	0	1	1	0	2	Nil	No	No
3	3	4	4	0	0	1	1	2	0	4	Nil	No	No
5	7	8	9	0	1	2	2	3	1	9	Chemical pneumonitis	No	No
6	8	9	9	0	1	2	2	3	1	9	Chemical pneumonitis	No	No

5	7	9	9	0	1	2	2	3	1	9	Chemical Pneumonitis	No	No
4	5	6	6	0	1	1	2	2	0	6	Nil	No	No
5	5	7	9	0	1	2	2	3	1	9	Chemical pneumonitis	No	No
	5	5	5	0	0	1	2	2	0	5	Nil	No	No
6	9	9	9	0	1	2	2	3	1	9	Chemical pneumonitis	No	No
	14	15		3	3	3	2	3	1	15	Died	yes	Yes
	5	5	5	0	0	0	2	2	1	5	Nil	No	No
4	4	4	4	0	0	0	1	2	1	4	Nil	No	No
3	3	3	3	0	0	0	1	2	0	3	Nil	No	No

	6	6	6	0	1	1	1	2	1	6	Nil	No	No
5	5	5	5	0	0	1	1	2	1	5	Nil	No	No
8	9	10	10	0	1	3	2	3	1	10	Chemical Pneumonitis	yes (2 days)	No
7	7	7	7	0	1	1	2	2	1	7	Chemical pneumonitis	No	No
	8	9	9	0	1	2	2	3	1	9	Chemical Pneumonitis	No	No
	3	3	3	0	0	0	1	2	0	3	Nil	No	No
4	4	5	5	0	0	1	1	2	1	5	NIl	No	No
3	4	5	5	0	0	1	1	2	1	5	Nil	No	No

4	4	4	5	0	0	1	1	2	1	5	Nil	No	No
				0	1	2	2	3	1	9	Chemical pneumonitis	No	No
6	8	9	9	0	1	2	2	3	1	9	Chemical Pneumonitis	No	No
4	4	5	5	0	0	1	2	2	0	5	Nil	No	No
	6	6	6	0	0	1	2	3	0	6	Nil	No	No
	2	3	3	0	0	0	1	2	0	3	Nil	No	No
4	4	4	4	0	0	0	1	2	1	4	Nil	No	No
	5	5	6	0	0	1	2	2	1	6	Nil	No	No
	6	6	6	0	0	1	2	2	1	6	NIl	No	No
6	7	9	9	0	1	2	2	3	1	9	Chemical pneumonitis	No	No

outcome (Duration of Stay)
8 days
5 days
8 days
8 days
6 days
2 days

11 days
8 days
7 days
3 days
3 days
9 days
8 days

9 days
5 days
8 days
3 days
6 days
Died in 3 hrs of admission
3 days
3 days
3 days

4 days
4 days
11 days
7 days
8 days
3 days
4 days
4 days

4 days
10 days
8 days
4 days
4 days
3 days
3 days
5 days
5 days
10 days

PROFOMA

Name

Age/Sex

Residence

Number of siblings

Parents Education

Usage of Kerosene at home

Place of Storage

Container used

Time passed since exposure

History of Vomiting following ingestion

(Spontaneous/induced, frequency, Blood streaked)

Any treatment given before admission

Past history of significant medical illness

Parameters in Scoring system

a. Cyanosis (Present/Absent)
b. SpO_2 level
c. CNS (Consciousness)
d. Pulmonary involvement (Wheeze/Crepitation and diminished air entry)
e. Respiratory distress (Cough, tachypnea, accessory muscle use)
f. Vomiting(Present/Absent)

Total score:

- On admission
- At 2hr
- At 4hr
- At 6hr
- At 8hr

X ray findings

Duration of hospitalization

ICU treatment / Ventilator support

Outcome: (Discharge / Death)