ASSESSMENT OF PREVALENCE AND MANAGEMENT OF ACUTE ORGANOPHOSPHATE POISONING AT KENYATTA NATIONAL HOSPITAL AND SELECTED RURAL HOSPITALS IN CENTRAL PROVINCE, KENYA - JAN 2004 TO DECEMBER 2006

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DECLARATION

This is my original work and has not been done or presented for diploma or degree in any institution.

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ABSTRACT

Background: Widespread use of organophosphate pesticides in rural agricultural communities has significantly contributed to increased incidences of acute organophosphate poisoning. The ease of organophosphate pesticide to degrade rapidly has made them more attractive alternative to resistant organ chlorine pesticides.

Objective: To investigate the prevalence and management of acute organophosphate poisoning in Kenyatta National Hospital and hospitals in neighboring agricultural suburbs.

Methods: The study was a cross-sectional study with retrospective review of medical records of 1564 cases of poisoning for three years between January 2004 to December 2007 at Kenyatta National Hospital (1215 cases), Nyeri Provincial General Hospital (255 cases) and Kiambu District Hospital (94 cases). The study further administered questionnaires assessing knowledge of healthcare providers on poisoning.

Results: Out of the 1564 cases studied, 18.1% was organophosphate poisoning and it was the highest prevalence compared to other poisonings. Organophosphate poisoning prevalence was 17.9% at Kenyatta National Hospital, 12.9% at Nyeri Provincial General Hospital and 35.1% at Kiambu District Hospital. Suicide contributed 68.2% of organophosphate poisoning while accidental poisoning accounted for 12% and homicide the lowest at 7.4%. Management of organophosphate poisoning using atropine was generally high (92.6%) and the use of oximes was relatively low (23.7%). Outcome of poisoning differed according to whether atropine alone or both atropine and oximes were used. Overall, 14.2% of the patients managed with atropine compared to 20.5% that were managed with both atropine and oximes died.

Generally, 62.2% of the healthcare providers had low level of knowledge on management of poisoning and specifically organophosphate poisoning and only 5.4% reported high level of knowledge. 43.1% of the healthcare providers attended training for poisoning management more than 6 years ago while another 31.1% were trained more than 3 years ago.

Conclusions: Organophosphate poisoning was more prevalent than other poisonings and suicide was the major among the circumstances that lead to poisoning. The use of oximes seemed to result in poor outcome as compared to outcome when atropine alone is used. In addition, the level of knowledge of healthcare providers was low and the continuous professional development programme on management of poisoning was unavailable.

DEDICATION

To my late father who were it not for him, I wouldn't have done pharmacy.

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CHAPTER ONE

1.0 INTRODUCTION

Deliberate self poisoning has reached epidemic proportions in parts of the developing world, where the toxicity of available poisons and sparse medical facilities ensure a high fatality rate [1]. Many deaths are due to organophosphorous pesticides and occur in young, economically active age groups [2].

Fatality rates of 20% are common and WHO has estimated that 200,000 people die each year from pesticide poisoning [3]. The accuracy of these figures is however keenly debated [4]. Unfortunately, the widespread use of organophosphate pesticides in the developing world's agricultural communities makes reduction of death by primary prevention a difficult task. Organophosphate pesticides tend to degrade rapidly on exposure to sunlight, air and soil though small amounts can persist and end up in food and drinking water. This has made them an attractive alternative to the resistant organochlorine pesticides such as DDT, aldrin and dieldrin.

Organophosphates are rapidly absorbed from skin, respiratory, and gastrointestinal routes. Toxicity varies depending on the route and degree of exposure as well as on the inherent toxicity of the specific compound. The mortality rate of organophosphate poisoning is high. Early diagnosis and appropriate treatment is often life saving.

Organophosphate poisoning usually occurs in one of three general circumstances: accidental ingestion by children, suicide attempts, or exposure in farm workers. The signs and symptoms of organophosphate poisoning result from accumulation of acetylcholine at the muscarinic, nicotinic, and central cholinergic receptor sites. Over stimulation at the muscarinic receptors produces hyperactivity of the parasympathetic nervous system producing the following signs and symptoms: salivation, lacrimation, urination, diarrhea, gastrointestinal cramps and emesis, papillary constriction, bradycardia and broncho-constriction. Over stimulation of nicotinic receptors (sympathetic and motor) leads to tachycardia, hypertension, hyperglycaemia, muscle fasciculations and paralysis particulary of respiratory muscle. Central nervous system effects vary from headache, slurred speech,

ataxia, seizures and coma. Death often results from central respiratory centre depression. Emergency department diagnosis of organophosphate toxicity depends on:

- History of exposure or high index of suspicion
- Signs and symptoms of cholinesterase inhibition
- Improvement of the signs and symptoms after administration of atropine with or without pralidoxime
- Inhibition of cholinesterase activity in blood.

The most classic presentation is a comatose patient who is diaphoretic with pinpoint pupils, diarrhea, excessive bronchial secretions and muscle fasciculations. However, if only the nicotinic and CNS effects are manifest, the diagnosis can be difficult. Many organophosphates have a characteristic garlic like odour that can provide an initial clue to the diagnosis.

In management of organophosphate poisoning attention to ABCs (Airways, Breathing, Circulation) is always the first priority both in the field and in the emergency department. Patients with altered mental status should receive intravenous naloxon, thiamine and dextrose. The other key aspects of general management of poisoning should be applied:

- Prevention of poison absorption (gastric lavage, chemical adsorption – activated charcoal)
- Enhancement of poison elimination
- Prevention of re-exposure

If signs and symptoms of poisoning with organophosphate are present, antidotal therapy with atropine must begin immediately. Atropine acts by competitively blocking acetylcholine at muscarinic receptors there by reversing the excessive parasympathetic stimulation. It has no effect on nicotinic receptors, and therefore will not reverse the muscle weakness or the sympathetic effect.

After atropine has been administered the second antidote, pralidoxime (2-PAM) should be given to reverse nicotinic effects. Pralidoxime specifically reactivates the cholinesterase that has been phospholylated by the organophosphate.

Complications of organophosphate poisoning include persistent central nervous system (CNS) effects (i.e. irritability, fatigue, impaired memory, depression and psychosis) and peripheral neuropathies (weakness, parasthesia, ataxia and chronic pain).

In untreated severely poisoned patients, death usually occurs within 24 hours and may be much more rapid with nerve agents. Most patients treated quickly and optimally, fully recover within10 days. If treatment is delayed 24 hours or inadequate, recovery may take months and neurological complications may be permanent.

1.1 GENERAL OBJECTIVE

To investigate the prevalence and management of organophosphate poisoning in Kenyatta National Hospital and hospitals in neighboring agricultural suburbs.

1.2 SPECIFIC OBJECTIVES

1. To determine the prevalence of organophosphate poisoning in Kenyatta National Hospital and hospitals in rural agricultural areas of Central Province.
2. To determine the circumstances of organophosphate poisoning in the three hospitals.
3. To determine the proportion of use of atropine and oximes in management of acute organophosphate poisoning and the outcome.
4. To assess the knowledge of organophosphate poisoning among health providers working in hospitals in rural agricultural areas of Central Province.

CHAPTER TWO

2.0 LITERATURE REVIEW

Organophosphates and carbamates are two of the most commonly used classes of insecticides. Both compounds are inhibitors of carboxylic esters hydroxylases, including acetyl cholinesterase (AchE) and Pseudocholinesterase. AChE is found in the human nervous system and plays a crucial role in controlling neurotransmission at the synapses.

Mechanism of Organophosphate Toxicity

Acetylcholine is a neurotransmitter at the neuromuscular junction, pre and postganglionic parasympathetic synapses. When the nerve terminal is stimulated by an action potential, acetylcholine is released into the synapse and binds its postsynaptic receptors. Acetylcholine immediately hydrolyses to prevent continuous stimulation of the receptors, which would lead to eventual paralysis of the synapse.

Inhibition of acetyl cholinesterase enzyme results in accumulation of acetylcholine at synapses and excessive stimulation of autonomic nervous system. This over stimulation produces the main toxic effects of organophosphates and carbamates.

Carbamates cause reversible inhibition of acetyl cholinesterase enzyme as opposed to the irreversible inhibition caused by organophosphates. The carbamylated enzyme can degrade spontaneously within minutes to hours to reactivate the enzyme. Therefore carbamates poisoning usually resolves within 24 – 48 hours. In contrast, organophosphates and acetyl cholinesterase enzyme form a phosphorylated complex that degrades over days to weeks. However, within 24 – 48 hours after phosphorylation, the enzyme complex loses an alkyl group in a process called aging, after which the enzyme can no longer spontaneously regenerate. New enzyme molecules must then be produced for physiologic activity to return.

Circumstances of poisoning

Information regarding the cause of poisoning is often superficial and speculative.
Poisoning circumstances can be classified as:

A. Deliberate

This can occur as self-poisoning where a person intends to commit suicide or as homicide where a poison is administered to an individual with the aim of harming them.

B. Accidental

Accidental poisoning occurs often in children. The individual is not aware of the toxic effects of the poison. More often than not it occurs when poison is transferred from their container into others e.g. soda bottles.

Clinical phases of organophosphate poisoning.

Following classical OP poisoning, three well defined clinical phases are seen: initial acute cholinergic crisis, the intermediate syndrome and delayed polyneuropathy (OPIDN) [7]. In addition, OPs on chronic exposure affect several of the physiological systems, which include central nervous system, neuromuscular junctions, cardiovascular system, metabolic and endocrine systems including reproduction. These effects have been reported both in humans and animals [8].

Acute cholinergic crisis

Organophosphates being inhibitors of esterases particularly the AChE, lead to acute cholinergic crisis in the initial phase. Accumulation of Acetylcholine occurs at nerve endings, as AChE is inhibited, leading to initial stimulation and eventually exhaustion of cholinergic synapses. The clinical findings are thereby a mixture of muscrinic effects resulting from the excitation of postganglionic parasympathetic activity, nicotinic effects resulting from accumulation of Acetylcholine (ACh) at neuromuscular junctions and consequent depolarisation and central nervous system effects causing initial excitation and subsequent inhibition of all CNS activity. Actual picture depends upon the balance between the nicotinic and muscrinic stimulation.

The muscrinic symptoms are diarrhoea, lacrimation, salivation, bronchorrhoea, bronchospasm, bradycardia, urination and miosis. However, depending upon the balance between the nicotinic and the muscrinic effects, patient may have hypertension and tachycardia occurring due to nicotinic actions rather than hypotension and bradycardia. The nicotinic receptors activated during acute intoxication lead to muscle paralysis. Fasciculation's may be seen and are a reliable sign of poisoning. Progression of paralysis may occur and the muscles of respiration may get affected. The mechanism of action of paralysis is depolarisation and desensitisation block induced by acetylcholine at the neuromuscular junctions. Severe intoxication may cause emotional irritability, mental obtundation, cognitive impairment, coma and convulsions because of CNS effects. In the cholinergic phase, paralysis usually passes off within 48-72 hours but complete clinical

recovery from all the effects may take up to a week after exposure to these compounds.

Intermediate syndrome

The term intermediate syndrome is derived from the fact that it arises between the period of early cholinergic syndrome and the late onset peripheral neuropathy. Its incidence in different studies has been reported to be between 20-68% [9]. This syndrome has been shown to be commonly associated with organophosphorous compunds like diazinon, dimethoate, methylparathion, methamidaphos, monocrotophos, fenthion and ethylparathion. It develops 12-96 hours after exposure and reflects a prolonged action of acetylcholine on the nicotinic receptors and is characterised by muscular weakness in the ocular, neck, bulbar, proximal limb and respiratory muscles. Occasionally, dystonic posturing may be observed and respiratory muscle weakness may be the first clue to the onset of this syndrome. The sensory functions characteristically remain normal and full recovery is evident in 4-18 days. Prolonged suppression of the enzyme acetyl cholinesterase is seen during this stage and metabolites of the parent compound may be demonstrable in the urine.

Organophosphate induced delayed polyneuropathy (OPIDN)

OPIDN is common following exposure to OPCs which have weak anticholinesterase activity e.g. triorthocresylphosphate. However, following exposure to the presently available OPCs which have strong anticholinesterase activity, it is distinctly uncommon [10]. OPIDN sets in after a period of 7-21 days of exposure and causes significant morbidity. The earliest symptoms to be seen are paraesthaesias and calf pain. Weakness initially appears initially in the distal leg muscles causing foot drop, followed by small muscles of the hands. Later it may extend proximally and even involve the truncal muscles. Gait ataxia is disproportionate to the motor and sensory loss. The cranial nerves and the autonomic nervous system are not involved. Deep tendon jerks are absent. The natural history of this neuropathy has revealed that it is subacute in onset in contrast to other toxic axonopathies, with a slow progression over 2 weeks. Clinical involvement of the corticospinal tracts and the dorsal columns becomes apparent when the peripheral neuropathy improves. The prognosis of patients with mild neuropathy is good but those with severe neuropathy are usually left with persistent deficits i.e. claw hand, foot drop, persistent atrophy, spasticity and ataxia.

The occurrence of OPIDN appears to follow the phosphorylation and subsequent ageing of an enzyme in axons called as neuropathy target esterase [10, 12]. Although the function of this

enzyme is not clear yet it is present in the brain, spinal cord and the peripheral nervous system. Animal experiments have shown that inhibition of the neuropathy-target esterase in the spinal cord produces only a spinal syndrome and not a peripheral neuropathy. For neuropathy to occur, ageing of the enzyme must take place and this involves cleavage of the lateral side chain from the phosphorylated neuropathy-target esterase and occurs in the axon and not the neuron cell body. These molecular events are then followed by characteristic changes in peripheral nerves, including the degeneration of predominantly long axons, with loss of myelin, and Schwan cell proliferation and macrophage accumulation in nerves. Neuropathy only develops with compounds which are able to inhibit as well as age the neuropathy-target esterase enzyme.

Chronic organophosphate induced neuropsychiatric disorder (COPIND)

Follow-up studies of individuals who have been exposed to high levels of organophosphorous compounds have shown that certain neurobehavioural changes may develop in them, which have been termed together as COPIND [13]. These effects include, drowsiness, confusion, lethargy, anxiety, emotional lability, depression, fatigue and irritability. Many of the studies of long term effects of high-dose organophosphorous compound exposure, are limited by the non-specific nature of these symptoms and by the low sensitivity and specificity of the neuropsychological scoring systems. On the other hand, some of these symptoms could be attributed to the sequelae of convulsions, anoxia, respiratory failure and cardiac arrhythmias that these patients might have suffered during the acute cholinergic syndrome. Savage et al [14] compared 100 matched pairs of individuals with previous organophosphorous poisoning to matched controls, and showed abnormalities in psychometric testing and motor reflexes. Rosenstock et al [15] studied agricultural workers who had a single episode of organophosphorous poisoning two years earlier. They demonstrated impaired neuropsychological testing and problems with visual memory, visuomotor speed, sequencing, problem solving and motor steadiness and dexterity. Chronic neuropsychiatric disorders like anxiety, depression, problems with memory and concentration have been described in workers exposed to organophosphorous compounds. In addition, dystonic reactions, schizophrenia, cog-wheel rigidity, choreoathetosis and electroencephalographical changes have been reported on high-dose exposure. These extrapyramidal symptoms are thought to be due to the inhibition of the acetylcholinesterase in the human extrapyramidal area. Psychosis, delirium, aggression, hallucination and depression, may also be seen during recovery from the cholinergic syndrome. Other types of delayed neurobehavioural effects are seen amongst people exposed to low dose of organophosphorous compounds for prolonged periods. Levin

et al [16] found a high level of anxiety in commercial sprayers of insecticides but not in farmers. Behan et al [17] observed that clinical features of psychological syndromes occurring after chronic exposure to organophosphorous compounds had great similarity to chronic fatigue syndrome.

GENERAL MANAGEMENT OF POISONED PATIENTS

Continuous observation, re-evaluation and supportive and symptomatic treatment are the cornerstones of the management of the poisoned patient. Emergency care begins with maintaining the airway, supporting respiration, and instituting cardiovascular resuscitation where necessary. Loss of airway or inadequate ventilation is the most common cause of serious morbidity or death in poisoning.

The object of treatment is to prevent and/or limit absorption of the poisonous substance and to maintain vital functions, such as ventilation and circulation. Once life support measures have been addressed, attention should be given to other aspects such as the identification of the poison, specific treatment and enhancement of elimination. In the management strategy, distinction should be drawn between exposures to poisonous substances and true poisonings, since the one does not necessarily lead to the other.

Terminating exposure

Skin: Absorption of poison through the skin should be limited by immediate removal of the contaminated clothing followed by washing with copious quantities of soap and water.

Emesis: Stimulation of the pharynx to induce emesis is safe provided that the patient is fully conscious and no contra-indications to the procedure exist. Induction of emesis by this means is not always effective but efficacy may be improved by giving the patient a glass of tepid water to drink before the attempt. Saline emetics should not be used, since they may cause hypernatraemia.

The use of **ipecacuanha** is discouraged, since there is no evidence in the literature to indicate that the potential benefits of its use outweigh the risks. Furthermore, **ipecacuanha** may produce persistent vomiting, diarrhoea, lethargy and drowsiness, effects which may complicate making an accurate diagnosis.

Table 2.1: Summary of Symptoms and signs of organophosphorus poisoning

Muscarinic receptors	Nicotinic receptors	Central receptors
Cardiovascular • Bradycardia • Hypotension **Respiratory** • Rhinorrhoea • Bronchorrhoea • Bronchospasm • Cough **Gastrointestinal** • Nausea/vomiting • Increased salivation • Abdominal cramps • Diarrhoea • Faecal incontinence **Genitourinary** • Urinary continence **Eyes** • Blurred vision • Increased lacrimation • Miosis **Glands** • Excessive salivation	**Cardiovascular** • Tachycardia • Hypertension **Musculoskeletal** • Weakness • Fasciculations • Cramps • Paralysis	**General effects** • Anxiety • Restlessness • Ataxia • Convulsions • Insomnia • Dysarthria • Tremors • Coma • Absent reflexes • Respiratory depression • Circulatory collapse

Gastric lavage: Lavage should not be used routinely in the management of oral exposure to poisonous substances. There is no evidence that its use improves outcome, and it may even cause significant morbidity. Unfortunately, this procedure is often used as a means of 'punishment' of a patient who has taken an overdose. There is no convincing clinical evidence that lavage, later than 1 hour after ingestion of a poisonous substance, is of therapeutic value. Lavage should only be considered where a patient has ingested a large amount of a poison with high inherent toxicity, if it can be performed within 1 hour of ingestion. It should be borne in mind that absorption of a poisonous substance may be enhanced by lavage; the large volumes of water increase dissolution of the toxic compound and may even flush it into the duodenum. It is recommended that a large bore orogastric tube/hose, 32 – 40 F in adults, and 16 – 28 F in children, be used if solid materials, such as tablets, capsules, and others have been ingested. In patients at risk of aspiration (e.g. patients with CNS depression or predisposed to seizures), airway protection is essential before gastric emptying is attempted.

Activated charcoal: Charcoal adsorbs (binds) poisons, thereby limiting their absorption. The greatest benefit is achieved if the charcoal is administered within two hours of ingestion of a poisonous substance. Activated charcoal is inert and generally safe and easy to use. Large doses may occasionally cause constipation. The usual single stat dose is 1 – 1.5 g/kg (50 – 100 g for an adult and 15 – 30 g for a child) given as an aqueous slurry, with 250 – 500 ml of water, i.e., one glass of water for every 30 – 50 g of charcoal. Activated charcoal is contraindicated after ingestion of a corrosive substance or when immediate endoscopy is to be undertaken. Water-soluble substances are not effectively adsorbed by activated charcoal. These include acids and alkalis, alcohols (including ethylene glycol and methanol), heavy metals (such as iron), arsenic, lithium and potassium salts. Contrary to what would be expected, charcoal does not effectively bind paraffin and related substances.

Cathartics: The use of cathartics (e.g. sorbitol), with or without activated charcoal, are not recommended for gastrointestinal decontamination since they may induce fluid and electrolyte disturbances, particularly in children.

Symptomatic and supportive care

Apart from specific measures directed at the poison itself (emesis, charcoal, antidotes, etc), careful ongoing observation and frequent re-evaluation of the patient form the cornerstones of management and care. Appropriate supportive care and effective relief of symptoms should not be neglected. Emergency procedures should be directed at maintaining a patent airway,

providing respiratory support, and effecting cardiovascular resuscitation if and when necessary. The loss of an effective airway and inadequate ventilation are the most common causes of serious morbidity and death in poisoning.

In comatose patients reliable **venous access** should be established and urine flow should be monitored.

Hypoventilation: Inadequate ventilation should be prevented by ensuring an adequate airway with immediate access to suction equipment, oxygen, and mechanical ventilation. Most poisons that depress consciousness also impair respiration. An obstructed airway needs immediate attention: dentures and oral secretions should be removed, an oral airway tube should be inserted, and the jaw should be extended forwards with the patient turned onto the left side (left lateral position).

Hypoglycaemia: Adequate blood glucose levels should be ensured in the comatose patient. If hypoglycaemia is present, 50 ml of 50% dextrose solution should be administered intravenously (adult dose). Hypoglycaemia should be suspected in poisoning/overdose with oral hypoglycaemics, salicylates and ethanol. Dextrose solution (50%) should not be administered before low blood glucose levels have been confirmed by a bedside finger prick blood glucose test. The empiric administration of high glucose concentrations should be avoided in patients at risk of central ischemia, e.g. patients with raised intracranial pressure, poor cerebral perfusion, cardiac output abnormalities, severe systemic hypotension, and in patients receiving CPR.

Volume depletion: Loss of fluid secondary to vomiting, diarrhoea and sweating is common and losses should be corrected.

Hypotension: Irrespective of the cause, e.g., volume depletion or venous pooling, central venous pressure monitoring should be resorted to for accurate assessment of fluid requirements.

Cardiac conduction and rhythm defects: Dysrhythmias and conduction defects may occur in acute poisoning with various substances. ECG monitoring is advisable and attention should be given to aggravating factors such as acidosis, hypoxia and electrolyte/fluid disturbances. Treatment will depend on the toxin involved and the type of dysrhythmia.

Seizures: Seizures that are self limiting and of short duration do not require immediate anticonvulsive therapy. Diazepam, given intravenously, should be administered if convulsions are protracted or recur. Phenytoin is an effective alternative agent. For the treatment of status epilepticus, unresponsive to the usual measures, thiopentone and muscle relaxants may be indicated (with appropriate respiratory support).

Hypothermia (< 35°C) may develop in comatose patients and may be missed by the unwary clinician. A low reading rectal thermometer should be used to record the temperature whenever hypothermia is suspected [18].

Specific management of organophosphate poisoning

The initial steps are clear. As the poison can be absorbed from the intact skin, it is necessary to remove any soiled clothing and wash the skin if there is evidence of contamination. A stomach wash is required and this may profitably be repeated after 2-3 hours as the drug is secreted back in the stomach, and to remove any residue not fully removed. As organophosphate phosphorylates the esteratic site of the enzyme Cholinesterase (AChE), it is unable to hydrolyze Acetylcholine. This accumulates at receptors & produces muscarinic and nicotinic signs. Muscarinic signs such as meiosis, diarrhea, vomiting, sweating, bronchial secretions are usually first to appear, and are treated with atropine. Nicotinic signs usually appear later, and do not respond to atropine. Though large dose of atropine are given in some units, 3-10 mg may be the loading dose, depending on severity. Once atropinized, a maintenance type dose at 1-3 mg 1/2 hourly is usually sufficient. Atropinization is assessed by a combination of signs including pupils, pulse rate, pulmonary secretions and mental state. It is not desirable to use any one criterion alone, because cases are seen where the pupils do not dilate or pulse does not become fast in spite of adequate doses. The use of atropine also tends to cause depressed reflexes to reappear, as the internuncial neurons in the cord are also muscarinic.

Use of Oximes such as pralidoxime (P2AM). P2AM is generally given at a dose of 1 gm 4 to 6 hourly. Oximes displace the organophosphates from the acetylchoine esterases and bind to the enzyme itself. Later it disassociates and AChE is reactivated. After animal studies, Johnson et al [19] believe that the effective dose is one which causes a blood level of 4 ng/ml. This could be achieved in an adult human by a dose of approximately 12 gms per day.

P2AM is not effective after the combination of pesticides and AChE has aged, and therefore is best given in the first 36- 48 hrs.

The problem with oximes is that its use has not been validated in humans in a controlled clinical trial. Use of oximes in OP poisoning remains conflicting and controversial. From the randomized, controlled trials it appears that they have no effect in moderate and severe poisoning and do more harm than good. Pralidoxime is very expensive, and unstable in aqueous solution. The treatment options are anticholinergic drugs and assisted ventilation, which is often needed.

2.1 STUDY JUSTIFICATION

According to preliminary report [5] acute organophosphate poisoning contributed 25% of all poisoning at Kenyatta National Hospital. This causes a significant burden to state in terms of resources and loss of workforce.

There is need to investigate the circumstances under which acute organophosphate poisoning occurs, the prevalence of poisoning according to sex, age and management of acute organophosphate poisoning. This will be aimed at coming up with information that will guide policy development for management of acute organophosphate poisoning in terms of control, prevention, preparedness and standard operating procedures in management of acute organophosphate poisoning. This is expected to reduce the incidence of organophosphate poisoning and improve management outcome.5 management outcome of acute poisoning.

CHAPTER THREE

3.0 METHODOLOGY

3.1 Study sites

The study was conducted in Kenyatta National Hospital, Nyeri Provincial General Hospital and Kiambu District Hospital between September 2007 and May 2008. Kenyatta National Hospital is a national referral hospital located in the city of Nairobi, Kenya. Both Nyeri PGH and Kiambu DH are in Central Province and located about 180 km and 15 km north of Nairobi respectively.

3.2 Study population

The study targeted all cases of poisoning treated in the three hospitals between January 2004 and December 2006.

3.3 Study design

The study utilized cross-sectional study design/survey.

3.4 Sample size and sampling procedure

3.4.1. Sampling poisoning cases

Poisoning was compared between Kenyatta National Hospital and three hospitals that were picked by convenience method. The inclusion criteria were that they were supposed to be in high potential agricultural areas of central Kenya. Nyeri Provincial Hospital, Thika District Hospital and Kiambu District Hospital were selected as study hospitals. The whole population of poisoning treated in the three hospitals between January 2004 and December 2006 was utilized in the study. The total sample was 1564 patients that included 1215 patients at KNH, 255 patients at Nyeri PGH and 94 patients at Kiambu DH. The research could not take place at Thika District Hospital despite having the green light because at the time of research the medical records department was relocating to new premises and could not avail the required patient files.

3.4.2 Sampling of healthcare providers for assessment of knowledge on poisoning

The sample size for assessment of knowledge among the healthcare providers was reached as described below.

Nyeri Provincial General Hospital

The following information was availed:

Cadre	Number
• Medical Officers	13
• Pharmacists	9
• Pharmaceutical Technologists	0
• Clinical Officers	22
• Nurses	269

In each category the sample was recruited by getting at least 10 – 20% of the total number of staff in each category putting into account inclusion criteria:

- Health workers chosen must fit into the predetermined category of health care providers.
- Health worker must have worked in hospital for six months before the study.
- Health worker must have worked in the outpatient (casualty) or in medical wards for at least one year.

A sample of pharmacist and medical officers who met the inclusion criteria were chosen by convenience method:

- 4 pharmacist were recruited representing 4/92 = 44%
- 7 Medical Officers 7/13 = 54%

There were no pharmaceutical technologists working at Nyeri Provincial General Hospital at the time of study. For clinical officers only those not working on specialized areas were recruited and 7 were recruited representing 7/22 = 32%. The Nursing Officers the nurses were recruited from the following areas.

Table 3.1: Recruitment of nursing officers

	Total Number of Nurses in the Unit	Nurses recruited
Casualty	19	4
Medical Wards	27	3
Pediatric Ward	12	3
ICU	12	3

Therefore the total population of nurses identified for study was 70 and the sample recruited was 16 representing 16/70 = 23%. Once the sample size was identified the sample was recruited by convenience and questionnaire administered.

Kiambu District Hospital

The same approach used in Kiambu DH as for Nyeri Provincial General Hospital in recruiting the sample. For nurses a total of one hundred and ninety seven (197) nurses work at Kiambu District Hospital. Out of this a total of 61 nurses meet the inclusion criteria and a total of 20 nurses were recruited. Ten from casualty and 10 from the medical wards.

The following was the sample recruited.

Table 3.2: Overall recruitment of healthcare providers

Category	Total Number	Number Recruited	Number recruited %
Medical Officers	13	5	38%
Pharmacists	5	5	100%
Pharmaceutical Technologists	2	2	100%
Clinical Officers	8	15	53%
Nurses	61	20	33%

Then the questionnaire (Annex III) was administered to each healthcare providers recruited.

3.5 Data collection procedure

3.5.1 Review of Medical Records of Cases of Organophosphate poisoning at Kenyatta National Hospital (KNH) Jan 2004 – Dec 2006

The medical records staff were requested to avail all the files for poisoning for the period of Jan 2004 – Dec 2006 through a letter of Chairman Department of Pharmaceutics and Pharmacy Practice, School of Pharmacy, Nairobi University (Annex VI). After the payment of pre-requisite fees the project was given a green light and the patient files were availed by the medical records department.

Two tools were used to extract data from the patient files. One tool was to collect poisoning resulting from organophosphate (**Annex I**) and the following data was collected. Patient biodata, the poison details, management of acute poisoning and the outcome. The other tool (**Annex II**) targeting poisoning other than organophosphate and the following information was collected, patient biodata, type of poison, circumstances and outcome.

3.5.2 Review of Medical Records of Cases of organophosphate poisoning in Central Province rural agricultural areas

Each hospital was visited and permission sought from hospital administration using authority letter to carry research from Kenyatta National Hospital ethical committee (annex V) and an introduction letter of the researcher from Faculty of Pharmacy University of Nairobi. Permission was granted by each of the two hospitals.

In Nyeri Provincial General Hospital and Kiambu District Hospital the medical records departments of respective hospitals were visited and requested to avail patient files of cases of poisoning for period Jan 2004 – Dec 2006.

Two tools were used to extract information from the patient files. One specifically targeted poisoning with organophosphate (Annex I) and helped collect the following information, patient biodata, the poison details, circumstances, management of organophosphate and outcome.

The other data collection tool (Annex II) was used to collect information on poisoning other than organophosphate. The following information was collected; patient biodata, poison, circumstance and outcome.

3.5.3 Assessment of knowledge of organophosphate poisoning among the healthcare providers in rural hospitals in Central Province

Nyeri Provincial General Hospital and Kiambu District Hospital were visited separately and a questionnaire administered (Annex III) to collect data on the knowledge of organophosphate poisoning of the health care providers. The different categories of health care providers sampled and who consented to participate in the study were interviewed using structured questionnaires by the researcher.

3.6 DATA MANAGEMENT

3.6.1 Data Coding

All the data collected on organophosphate poisoning, other poisonings and the knowledge of the healthcare providers were coded appropriately to facilitate data entry into computer for analysis.

For assessment of knowledge of organophosphate poisoning among healthcare providers the respondent were scored on how they answered the following questions in the questionnaire:

Table 3.3: Scores for knowledge questions

QUESTION NUMBER	MAXIMUM SCORES
1	4
2	5
3	5
4	5
5	6

The knowledge levels were categorized as follows:

Table 3.4: Score categories showing the cumulative levels of knowledge

SCORE	KNOWLEDGE LEVEL
18 - 25	High knowledge
10 – 17	Medium knowledge
1 – 9	Low knowledge
0	No knowledge

3.6.2 Data Analysis

The coded data was entered into the computer and then cleaned/edited before analysis was performed using Statistical Package for Social Sciences (SPSS) Version 11.5. The categorical variables such sex, age group, prevalence of poisons, circumstances of poisoning, management of poisoning, outcome and knowledge levels were analyzed and presented using proportions. The differences between specific variables were tested for statistical significance using chi-square (X2) test. Similarly, continuous variables such as age, dosages and frequency of administration of drugs were analyzed and presented as mean or median where appropriate. All tests were performed at 5% significance level or 95% confidence limit.

3.7 STUDY LIMITATIONS

The study relied on information obtained from hospital records on cases of poisonings that were treated in the three hospitals. Generally, the patients' history was very scanty mostly in Nyeri PGH and Kiambu DH and the most lacking histories include: social and occupational history, history of previous illness, family history and treatment history. But generally, the records for KNH were very good.

CHAPTER FOUR

4.0 RESULTS

4.1 BASELINE CHARACTERISTICS

The study reviewed the records of 1564 patients that sought treatment for poisoning at Kenyatta National Hospital (1215 patients), Nyeri Provincial General Hospital (255 patients) and Kiambu District Hospital (94 patients) between 2004 and 2006. The sex distribution of the study population was 60% male and 40% female and with a median age of 22 years (Range: 0.17-85 years). The male population had a median age of 24 years (0.17 – 84 years) and the female 20 years (0.58 – 85 years) showing a statistically significant difference (P=0.00) between the two groups in terms of age.

4.2 ORGANOPHOSPHATE POISONING

4.2.1 Characteristics of patient presenting with acute organophosphate poisoning

Among those treated with acute organophosphate poisoning, 69.3% and 30.7% were males and females respectively with an overall median age of 25 years (Range: 5 months to 85 years). Similarly, specific data from hospitals recorded a high proportion of males involved in poisoning and was highest in Kiambu (81.8%) compared to Nyeri PGH (75.8%) and KNH (66.4%). The median age of the patients in the three hospitals were 25 years (Range: 2.5 – 65 years), 26 years (Range: 1 – 85 years) and 25 years (Range: 5 months – 60 years) respectively.

Table 4.1 below shows the descriptive characteristics of organophosphate poisoning.

Table 4.1: Organophosphate poisoning

Characteristics	Overall Count (%)	KNH Count (%)	Nyeri PGH Count (%)	Kiambu DH Count (%)
Patient's sex				
Male	196 (69.3)	144 (66.4)	25 (75.8)	27 (81.8)
Female	87 (30.7)	73 (33.6)	8 (24.2)	6 (18.2)
Poison name				
Diazinon	123 (43.5)	110 (50.7)	4 (12.1)	9 (27.3)
Acaricide	21 (7.4)	17 (7.8)	2 (6.1)	2 (6.1)
Diamethoate	5 (1.8)	2 (0.9)	3 (9.1)	0 (0)
Malathion	123 (4.6)	2 (0.9)	7 (21.2)	4 (12.1)
Others	121 (42.8)	86 (39.6)	17 (51.5)	18 (54.5)

4.2.2 Organophosphate chemicals employed in the poisoning

Among the organophosphate poisons recognized while treating patients for poisoning, the commonly encountered poisons included diazinon, acaricides and malathion. Some

poisonings were only identified as caused by organophosphates but the specific poisons were not identified.

Generally, as indicated in Table 4.1 above, diazinon contributed 43.5% of organophosphate poisoning which was the highest compared to 7.4%, 4.6% and 1.8% of acaricides, malathion and diamethoate respectively. However, other unidentified organophosphates contributed a considerable overall proportion of 42.8% of the poisons. The overall proportion of diazinon poisoning seemed to increase, though statistically insignificant, from 37.1% in 2004 to 45.2% in 2005 and then 49.4% in 2006.

Diazinon was still leading at 50.7% at KNH and the unidentified organophosphates contributing 39.6%. Notably, unidentified organophosphate poisons were highest in Nyeri PGH and Kiambu DH and was estimated at 51.5% and 54.5% respectively. Conversely, malathion in Nyeri PGH contributed more poisoning (21.2%) than diazinon (12.1%) and the trend of malathion poisoning doubled from 12.5% in 2004, 23.5% in 2004 and then 25% in 2006. Similarly, diazinon in Kiambu contributed a considerably higher proportion of poisoning (27.3%) compared to acaricides and malathion.

4.2.3 Prevalence of organophosphate poisoning

Out of the studied 1564 cases of poisoning, 283 cases (18.1%) were due to organophosphate poisoning accounting for the highest proportion of poisoning in the three hospitals. Zinc phosphide contributed the second highest at 15.3% and other notable poisons included kerosene (11.8%), food poisoning (8.2%), prescription drugs (6.9%), sedatives (6.8%) and amitrax (6.6%). The rest of the proportion was contributed by other poisons as indicated in Table 4.2 below.

Similarly, organophosphate poisoning prevalence was 17.9% and 35.1% in KNH and Kiambu DH respectively and was still high compared to the rest of the poisoning. However, Nyeri PGH that recorded 12.9% organophosphate poisoning prevalence differed with the other hospitals and amitrax contributed the highest proportion (21.2%) followed by kerosene (16.9%) in the hospital.

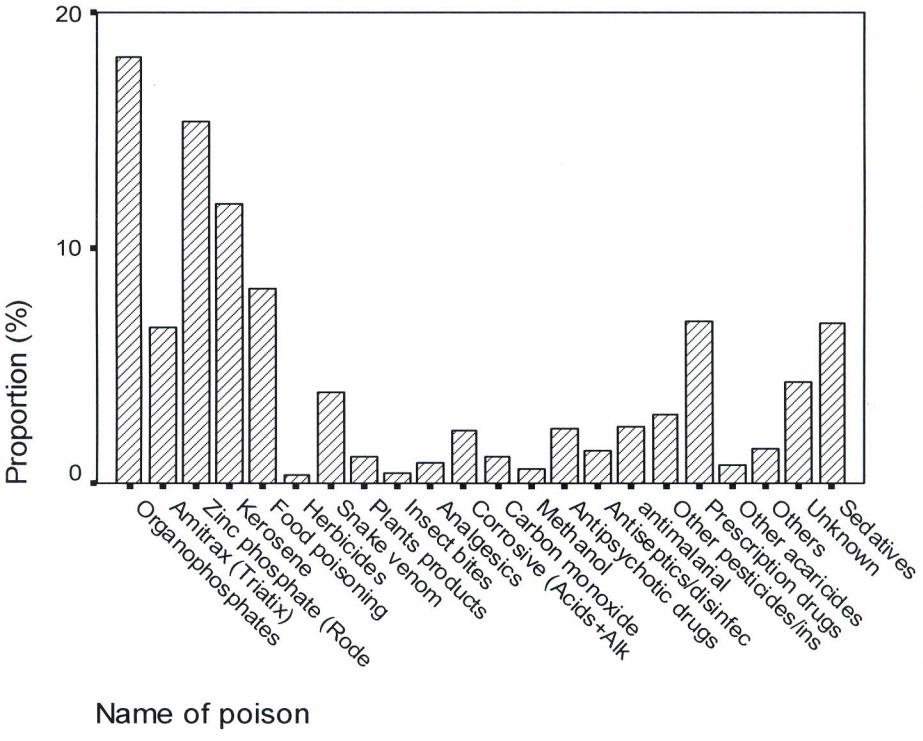
Annually, organophosphate poisoning was not significantly different over the three years studied. In 2004 the prevalence stood at 19.7% with a slight reduction in 2005 and 2006 standing at 17% and 17.6% respectively. The contribution of each poison to poisoning as was

Table 4.2: Prevalence of poisoning

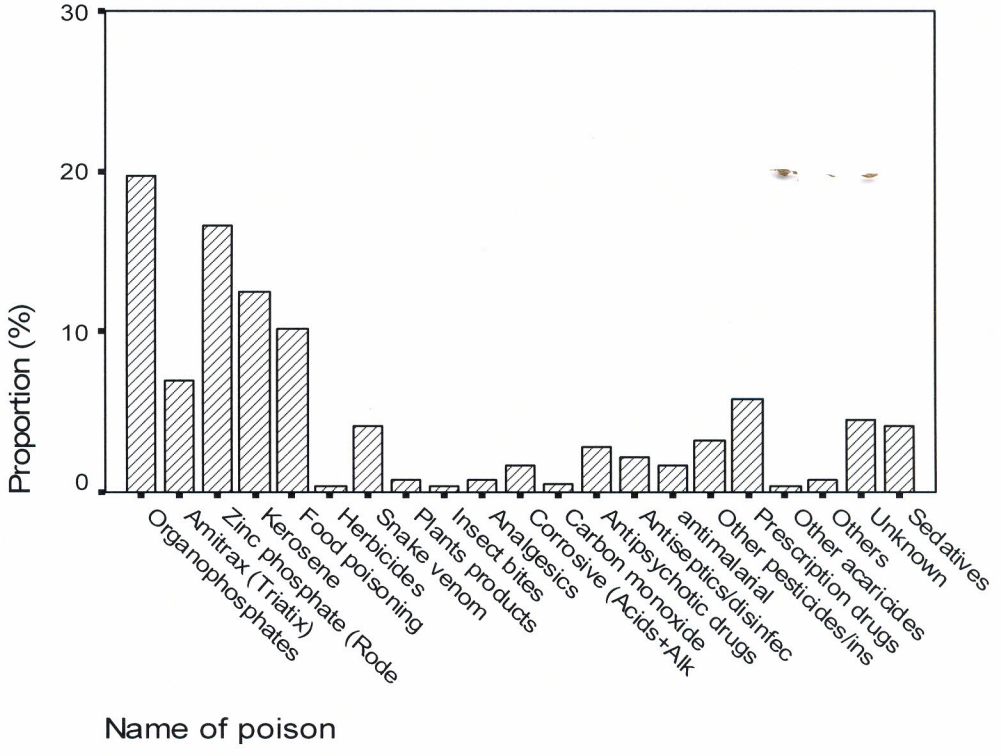
Type of poison	Overall Count (%)	KNH Count (%)	Nyeri PGH Count (%)	Kiambu DH Count (%)
Organophosphates	283 (18.1)	217 (17.9)	33 (12.9)	33 (35.1)
Amitrax (Triatix)	103 (6.6)	45 (3.7)	54 (21.2)	4 (4.3)
Zinc phosphide (Rodenticide)	240 (15.3)	190 (15.6)	28 (11.0)	22 (23.4)
Kerosene	185 (11.8)	137 (11.3)	43 (16.9)	5 (5.3)
Food poisoning	129 (8.2)	105 (8.6)	21 (8.2)	3 (3.2)
Herbicides	6 (0.4)	1 (0.1)	4 (1.6)	1 (1.1)
Snake venom	61 (3.9)	59 (4.9)	2 (0.8)	
Plants products	17 (1.1)	15 (1.2)	1 (0.4)	1 (1.1)
Insect bites	7 (0.4)	5 (0.4)	2 (0.8)	2 (2.1)
Analgesics	14 (0.9)	12 (1.0)	2 (0.8)	
Corrosive (Acids+Alkali)	35 (2.2)	31 (2.6)	2 (0.8)	
Carbon monoxide	18 (1.2)	17 (1.4)	1 (0.4)	
Methanol	9 (0.6)	9 (0.7)		
Antipsychotic drugs	36 (2.3)	27 (2.2)	8 (3.1)	1 (1.1)
Antiseptics/disinfectants	22 (1.4)	19 (1.6)	3 (1.2)	
Antimalarial	37 (2.4)	34 (2.8)	3 (1.2)	
Other pesticides /insecticides	46 (2.9)	20 (1.6)	23 (9.0)	3 (3.2)
Prescription drugs	108 (6.9)	97 (8.0)	6 (2.4)	5 (5.3)
Other acaricides	12 (0.8)	5 (0.4)	7 (2.7)	
Others	23 (1.5)	19 (1.6)	2 (0.8)	2 (2.1)
Unknown	67 (4.3)	45 (3.7)	10 (3.9)	12 (12.8)
Sedatives	106 (6.8)	106 (8.7)		
Total	**1564 (100.0)**	**1215 (100.0)**	**255 (100.0)**	**94 (100.0)**

recorded showed a uniform pattern over the three years studied. As illustrated by figure 4.1 (a), (b), (c) and (d), organophosphate poisoning was highest among all poisons and zinc phosphide contributed the second highest portion. Then kerosene and food poisoning were also significant poisons in a pattern that was repeated over the 3 years studied. As illustrated by figure 4.1 (e), (f) and (g), the proportions organophosphate and zinc phosphide were high in both KNH and Kiambu and a difference was recorded only in Nyeri PGH.

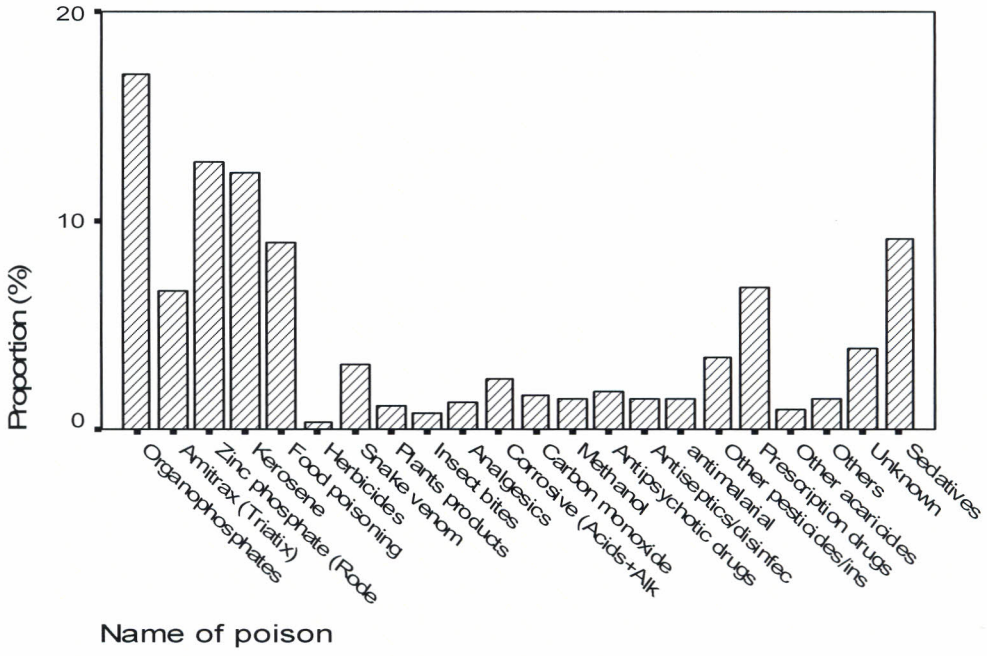
Figure 4.1
(a): Overall proportions of cases of poisoning treated



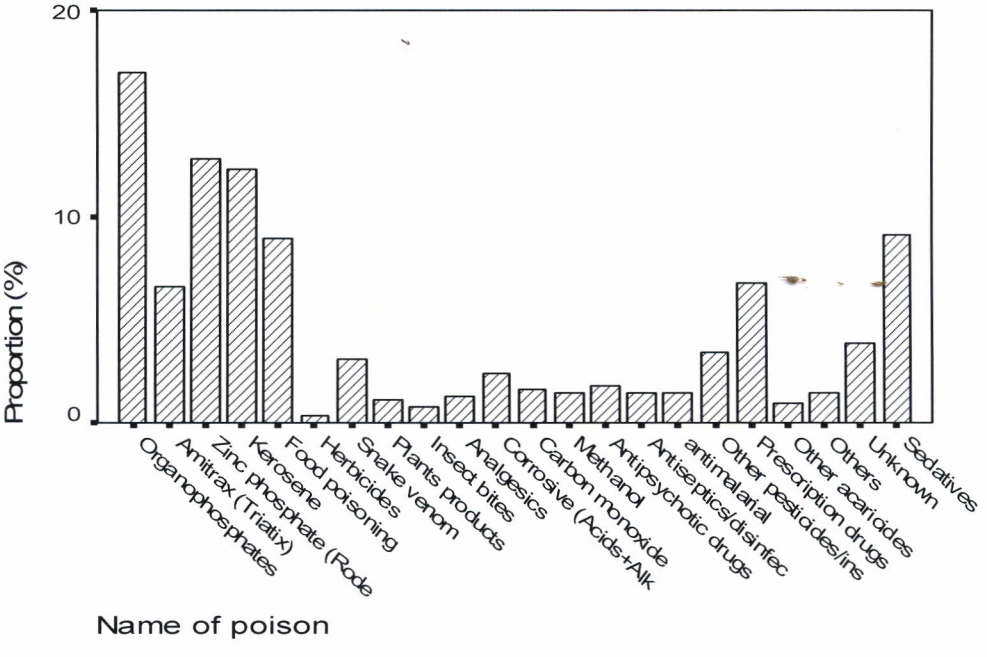
(b) Proportions of cases of poisoning in 2004



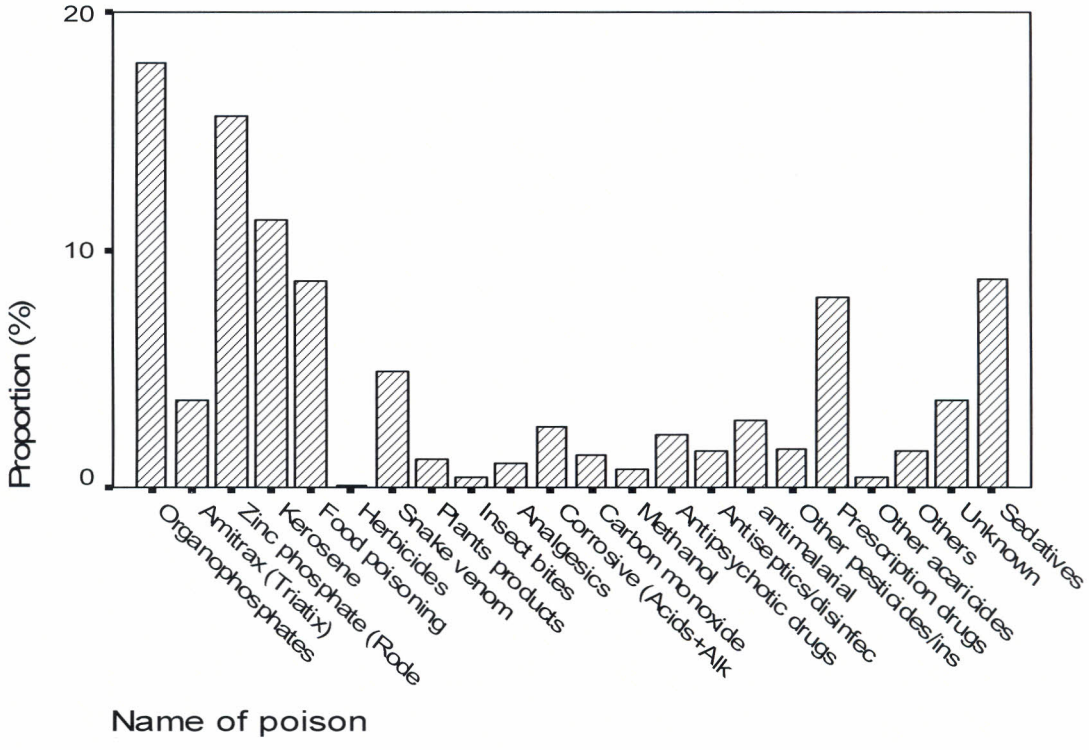
(c) Proportions of cases of poisoning treated in 2005



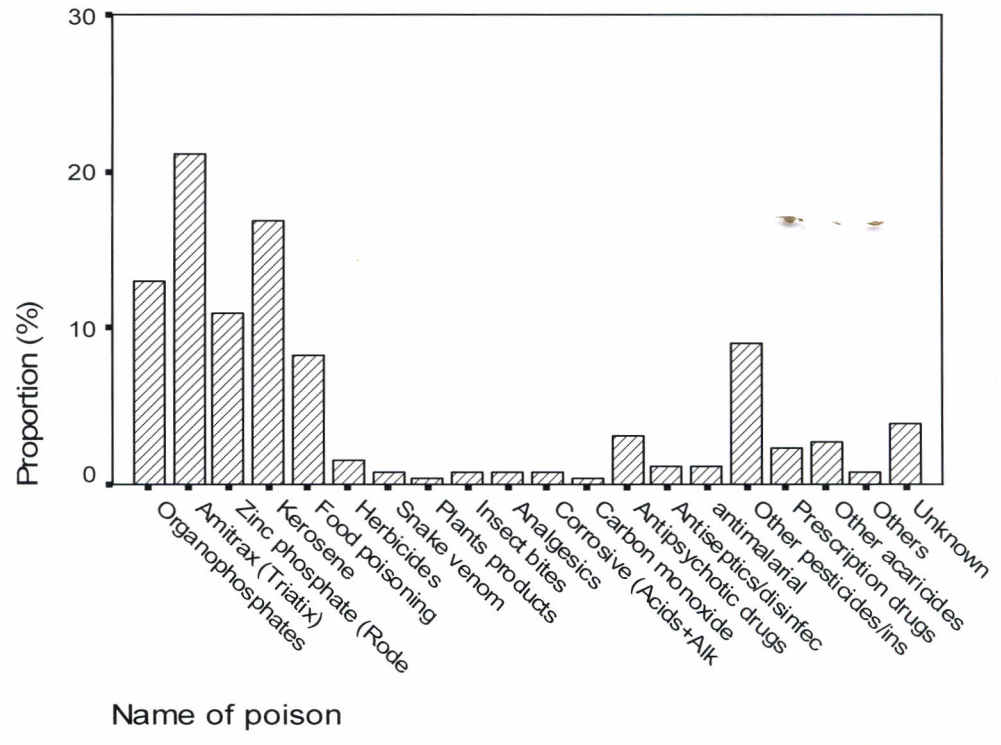
(d) Proportions of cases of poisoning treated in 2006



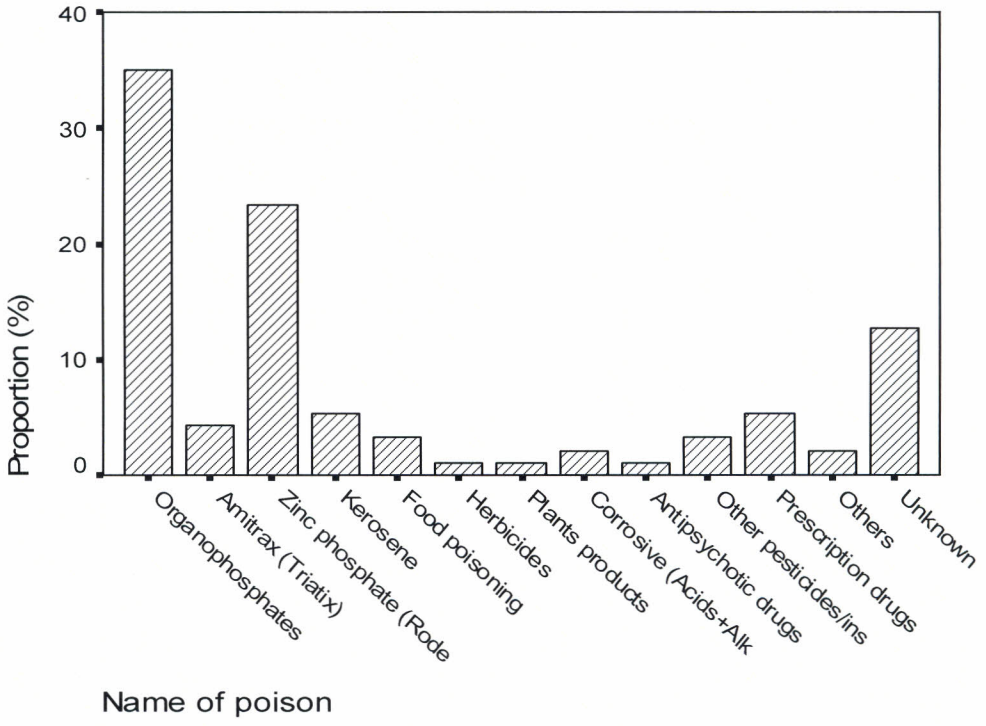
(e) Proportions of cases of poisoning treated in KNH



(f) Proportions of cases of poisoning treated in Nyeri PGH



(g) Proportions of cases of poisoning treated in Kiambu DH



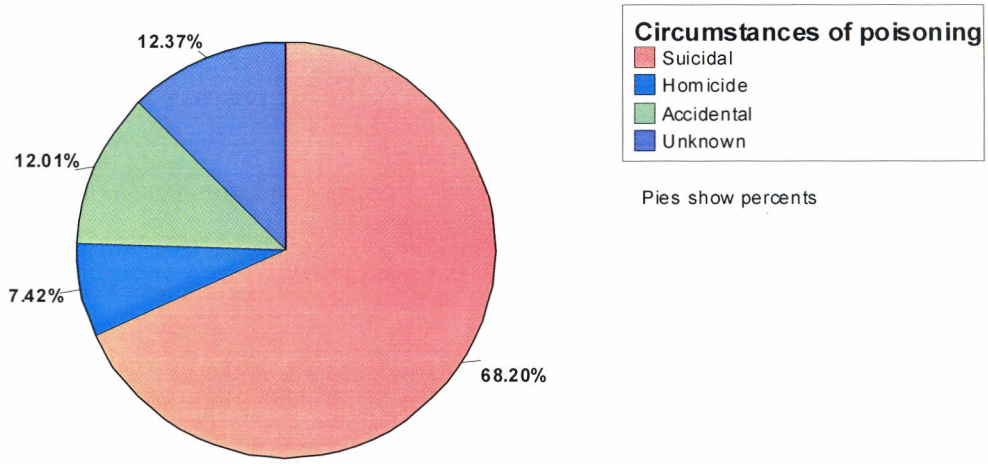
4.2.4 Circumstances of organophosphate poisoning

Table 4.3: Circumstances of organophosphate poisoning

Characteristics	Overall Count (%)	KNH Count (%)	Nyeri PGH Count (%)	Kiambu DH Count (%)
Circumstances of poisoning				
Suicidal	193 (68.2)	148 (68.2)	25 (75.8)	20 (60.6)
Homicide	21 (7.4)	20 (9.2)	1 (3.0)	0 (0)
Accidental	34 (12.0)	29 (13.4)	2 (6.1)	3 (9.1)
Unknown	35 (12.4)	20 (9.2)	5 (15.2)	10 (30.3)

The circumstances that surrounded poisoning included mainly suicide, accidents and homicide. As indicated in Table 4.3 and illustrated by figure 4.2 below, 68.2% of overall organophosphate poisoning was due to suicide while homicide contributed the lowest proportion of 7.4% and accidental poisoning accounted for 12%. Similarly, specific data for each hospital concurred that suicide is the main contributor of poisoning giving proportions of 68.2%, 75.8% and 60.6% at KNH, Nyeri PGH and Kiambu DH respectively. Homicide was lowest in all the three hospitals with no case reported at Kiambu DH in the three studied years. However, there was an overall 12.4% portion of poisoning that occurred under unknown circumstances.

Figure 4.2: Circumstances of organophosphate poisoning



4.2.5 Management of organophosphate poisoning

Most patients that presented with organophosphate poisoning received general management (ABCD) from the hospitals. This involved airway support (removal of vomitus, mucus secretions and ensuring ventilation), breathing support (mechanical breathing), circulation support (monitoring blood pressure and heart rate at regular intervals and giving of intravenous fluids) and giving dextrose for comatose patients and determination of blood sugar levels where possible. Overall, as shown in Table 4.4, 96.1% were given general management and equally high proportions were obtained for KNH (98.6%), Nyeri PGH (93.9%) and (Kiambu DH) 81.8%.

Management of poisoning included removal and inactivation of unabsorbed poisons and removal of absorbed poisons. Unabsorbed poisons were removed in 75.3% of all organophosphate poisoning and the most preferred method was removal of poisons from GIT accounting for 88.3%. Removal of poison from skin was low accounting for only 3.3%. The removal of poison from both GIT and contaminated skin was also low at 6.6%. KNH, Nyeri PGH and Kiambu DH also reported 74.7%, 90.9% and 63.6% cases whose unabsorbed poisons were removed respectively and in all the cases the choice method was removal from the GIT (85.2%, 100% and 95.2% respectively for the three hospitals). Only 11 (3.9%) cases of the overall 283 cases of poisoning received inactivation of unabsorbed poisons. Inactivation using activated charcoal was used in 10 cases while only 1 case washing was used. In addition, removal of absorbed poison was low with only 33 (11.7%) cases overall with 63.9% undergoing induction of diuresis and 21.2% urine alkalization. In overall 68.9% of the organophosphate poisoning was managed using atropine alone, 23.7% used both atropine and oximes and 7.4% used neither atropine nor oximes.

Table 4.4: Management and outcome of organophosphate poisoning

Characteristics	Overall Count (%)	KNH Count (%)	Nyeri PGH Count (%)	Kiambu DH Count (%)
General management (ABCD)				
Yes	272 (96.1)	214 (98.6)	31 (93.9)	27 (81.8)
No	11 (3.9)	3 (1.4)	2 (6.1)	6 (18.2)
Removal of unabsorbed poison				
Yes	213 (75.3)	162 (74.7)	30 (90.9)	21 (63.6)
No	70 (24.7)	55 (25.3)	3 (9.1)	12 (36.4)
Method of removal of unabsorbed poison				
	7 (3.3)	6 (3.7)		1 (4.8)
Skin	188 (88.3)	138 (85.2)	30 (100)	20 (95.2)
GIT	14 (6.6)	14 (8.6)		
Both	4 (1.9)	4 (2.5)		
Others				
Inactivation of unabsorbed poison				
Yes	11 (3.9)	7 (3.2)	2 (6.1)	2 (6.1)
No	272 (96.1)	210 (96.8)	31 (93.9)	31 (93.9)
Method of inactivation of unabsorbed poison				
Skin (washing)	1 (9.1)	7 (100)	2 (100)	1 (50.0)
GIT (activated charcoal)	10 (90.9)			1 (50.0)
Removal of absorbed poison				
Yes	33 (11.7)	19 (8.8)	6 (18.2)	8 (24.2)
No	250 (88.3)	198 (91.2)	27 (81.8)	25 (75.8)
Method of removal of absorbed poison				
Urine alkalinisation	7 (21.2)	7 (36.8)		
Induction of diuresis	21 (63.6)	8 (42.1)	6 (100)	7 (87.5)
Both	1 (3.0)	4 (21.1)		1 (12.5)
Others	4 (12.1)			
Use of atropine				
Yes	262 (92.6)	207 (95.4)	27 (81.8)	28 (84.8)
No	21 (7.4)	10 (4.6)	6 (18.2)	5 (15.2)
Use of loading dose				
Yes	71 (27.1)	51 (24.6)	5 (18.5)	15 (53.6)
No	191 (72.9)	156 (75.4)	22 (81.5)	13 (46.4)
Route of atropine administration				
IV	118 (45.0)	110 (53.1)	4 (14.8)	4 (14.3)
IM	5 (1.9)	3 (1.4)	1 (3.7)	1 (3.6)
SC	4 (1.5)	3 (1.4)	0 (0)	1 (3.6)
Infusion	7 (2.7)	4 (1.9)	2 (7.4)	1 (3.6)
Not indicated	128 (48.9)	87 (42.0)	20 (74.1)	21 (75.0)
Use of oximes				
Yes	67 (23.7)	64 (29.5)		3 (9.1)
No	216 (76.3)	153 (70.5)	33 (100)	30 (90.9)
Reason for no oximes				
Not prescribed	201 (93.1)	142 (92.8)	33 (100)	26 (86.7)
out of stock	9 (4.2)	9 (5.9)		0 (0)
Unknown	6 (2.8)	2 (1.3)		4 (13.3)
Route of oximes administration				
IV	37 (55.2)	37 (57.8)	-	
Not indicated	30 (44.8)	27 (42.2)		3 (100)
Outcome of poisoning				
Well and discharged	242 (85.5)	188 (86.6)	27 (81.8)	27 (81.8)
Died	41 (14.5)	29 (13.4)	6 (18.2)	6 (18.2)

4.2.6 Use of atropine

Generally, as indicated in Table 4.4 above, 92.6% of cases of organophosphate poisoning were managed using atropine. This was similarly high in all the hospitals reporting 95.2%, 81.8% and 84.8% in KNH, Nyeri PGH and Kiambu DH respectively. A small proportion of the cases (27.1%) were started with a loading dose of atropine that ranged from 0.1 - 2.4 mg (median of 1.2 mg). the maintenance dose of atropine among those that used it and whose data on dosage was available reported a median dose of 1.2 mg that was given after a median time of 30 minutes. The dosages were different for three hospitals with KNH and Nyeri PGH recording similar median loading dose of 2.0mg while Kiambu reported a lower dose of median 0.6 mg. The maintenance doses were also different between the hospitals with KNH, Nyeri PGH and Kiambu DH reporting a median of 1.2 mg, 2.0 mg and 0.6 mg respectively. While the median frequency of administration for KNH and Nyeri PGH, both reported 30 minutes, Kiambu reported a lower time interval of 15 minutes between atropine administrations. Forty five (45%) of atropine administration was done intravenously and the other alternative routes of administration were intramuscular (1.9%), subcutaneous (1.5%), infusion (2.7%) and those with route not indicated formed 48.9%.

4.2.6.1 Atropine usage in KNH

A total of 217 cases of organophosphate poisoning were treated at KNH. Out of all the patients, 207 were put on atropine and the doses were indicated in 197 of the cases while the frequency of atropine administration was indicated in 184 patients.

For children aged 0 – 5 years, the dosages of atropine varied from 0.1 to 2mg. the most widely used dosage for this age group was 0.3mg with 32.1% usage followed by 0.5mg with 25% usage. The other dosage was 0.1mg with 10.7% usage. For children the recommended dosage is 0.02mg to 0.05mg/kg body weight every 10 to 15 minutes. This translates to 0.2mg to 0.7mg every 10 to 15 minutes. For children more than 14 years and adults, the dosages varied from 0.5mg to 1g (given by infusion). The most common dosage in this group was 2mg contributing 41.9% followed by 1.2mg contributing to 17.5%, 1mg contributing 17.5% and 0.6mg contributing another 17.5%. The 1g dosage was administered as infusion. The recommended adult dose is 2mg to 3mg intravenously every 10 to 15 minutes.

The frequency of atropine administration at KNH varied from every 5 minutes to every 30 minutes with infusion running for one hour. The frequency widely used was every 15 minutes contributing 52.8% followed by every 30 minutes contributing 29.6% and followed by every 20 minutes contributing 9.2%.

Table 4.5: Atropine dosage and age group at KNH

Atropine dose in mg	AGE GROUP							Total
	0-5 years	6-13 years	14-20 years	21-30 years	31-40 years	41-50 years	51 years and above	
0.10	3	0	0	0	0	0	0	3
0.20	1	0	0	1	0	0	0	2
0.24	1	0	0	0	0	0	0	1
0.30	9	0	0	0	0	0	0	9
0.35	1	0	0	0	0	0	0	1
0.50	7	1	0	0	1	0	0	9
0.60	1	1	5	18	2	2	1	30
0.70	1	0	0	0	0	0	0	1
0.75	1	0	0	0	0	0	0	1
0.95	1	0	0	0	0	0	0	1
1.00	0	2	4	17	4	3	0	30
1.20	1	0	4	16	5	2	1	29
1.50	0	0	1	0	0	0	0	1
1.80	0	0	0	1	1	0	0	2
2.00	1	0	10	30	12	8	7	68
4.00	0	0	0	3	0	0	0	3
8.00	0	0	0	2	0	0	0	2
36.00	0	0	0	0	0	1	0	1
1000.00	0	0	1	1	1	0	0	3
Total	28	4	25	89	26	16	9	197

Table 4.6: Atropine dosage and frequency of administration at KNH

Atropine dose in mg	Frequency					Total
	Every 5 mins	Every 15 mins	Every 20 mins	Every 30 mis	Hourly or more	
0.10	0	0	1	1	1	3
0.20	0	1	1	0	0	2
0.24	0	0	0	0	1	1
0.30	0	4	0	1	3	8
0.35	0	1	0	0	0	1
0.50	0	7	0	1	1	9
0.60	2	13	2	5	5	27
0.70	0	1	0	0	0	1
0.75	0	1	0	0	0	1
1.00	0	11	2	11	5	29
1.20	1	8	2	7	7	25
1.50	0	0	0	1	0	1
1.80	0	0	0	2	0	2
2.00	0	28	4	21	13	66
4.00	0	0	0	0	3	3
8.00	0	0	0	0	1	1
36.00	0	0	0	0	1	1
1000.00	0	0	1	1	1	3
Total	3	75	13	51	42	184

4.2.6.2 Atropine usage in Nyeri PGH

A total of 33 cases of organophosphate poisoning were treated at Nyeri PGH over the study period. Twenty seven (27) of them were put on atropine and the dosages of atropine were indicated in 20 patients while only 16 cases had the frequency of atropine administration indicated.

For the age group of 14 and above, the dosages of atropine administered ranged from 1mg – 4mg. the highest encountered dosage was of 2mg contributing 64.7% followed by 1mg contributing 23.5%. The dosages of atropine were on the lower recommended dosage levels.

The frequency of atropine dosage administration ranged from every 15 to 30 minutes for bolus injection with infusion taking one hour. The most commonly applied frequency of administration in Nyeri was every 30 minutes contributing 66.7% followed by every 15 minutes contributing 26.7%. This frequency is in the normal range but skewed to the upper limit.

Table 4.7: Atropine dosage and age group at Nyeri PGH

Atropine dose in mg	AGE GROUP					Total
	14-20 years	21-30 years	31-40 years	41-50 years	51 years and above	
1.00	2	1	0	0	1	4
1.20	0	0	0	0	1	1
2.00	1	5	0	1	4	11
4.00	0	0	0	0	1	1
12.00	0	0	1	0	0	1
12.50	1	1	0	0	0	2
Total	4	7	1	1	7	20

Table 4.8: Atropine dosage and frequency of administration at Nyeri PGH

Atropine dose in mg	Frequency				Total
	Every 15 mins	Every 20 mins	Every 30 mis	Hourly or more	
1.00	2	0	1	1	4
1.20	1	0	0	0	1
2.00	1	1	8	0	10
4.00	0	0	1	0	1
Total	4	1	10	1	16

recording 100% non-prescription. In addition, the route of administration of oximes in the cases that were indicated was intravenous.

4.2.7.1 Oximes doses and frequency of administration

The doses of oximes administered ranged from 2mg to 2000mg with frequency of administration varying s follows:

- Loading doses – 39 patients
- Every 15 minutes – 2 patients
- Every 30 minutes – 2 patients
- Every 1 hour – 2 patients
- Every 2 hours – 1 patient
- Every 4 hours – 1 patient
- Every 6 hours – 2 patients
- Every 8 hours – 2 patients
- Every 12 hours – 4 patients
- Every 24 hours – 3 patients

From the above scenario, it shows there is no standardized administration of oximes in both KNH and Kiambu DH.

Table 4.11: Oximes doses and the frequency of administration

Oximes doses in mg	Frequency of oxime use (hours)										Total
	Stat	Quarter hourly	Half hourly	Hourly	Two hourly	Four hourly	Six hourly	Eight hourly	Twelve hourly	Daily	
2.00	4	0	0	0	0	0	0	0	0	0	4
30.00	1	0	0	0	0	0	0	0	0	0	1
120.00	0	0	0	0	1	0	0	0	0	0	1
140.00	1	0	0	0	0	0	0	0	0	0	1
150.00	1	0	0	0	0	0	0	0	1	0	2
200.00	2	0	0	1	0	0	0	0	0	0	3
240.00	0	0	0	1	0	0	0	0	0	0	1
250.00	3	0	0	0	0	0	0	0	0	0	3
300.00	0	0	0	0	0	0	0	1	1	1	3
350.00	2	0	0	0	0	0	0	0	0	0	2
400.00	0	0	0	0	0	0	0	1	0	0	1
410.00	1	0	0	0	0	0	0	0	0	0	1
450.00	1	0	0	0	0	0	0	0	0	0	1
500.00	3	0	0	0	0	1	0	0	0	1	5
600.00	1	0	0	0	0	0	0	0	0	0	1
750.00	1	0	0	0	0	0	0	0	0	0	1
1000.00	6	2	1	0	0	0	0	0	0	0	9
1200.00	2	0	0	0	0	0	1	0	0	0	3
1500.00	1	0	0	0	0	0	0	0	0	0	1
2000.00	9	0	1	0	0	0	1	0	2	1	14
Total	39	2	2	2	1	1	2	2	4	3	58

4.2.8 Outcome of organophosphate poisoning after management with atropine and oximes

As indicated in Table 4.12 below, outcome of poisoning seemed to differ between the patients that were managed using atropine alone and those that used both atropine and oximes. Overall, 14.2% of the patients managed with atropine compared to 20.5% that were managed with both atropine and oximes died. Similarly, the data from each hospital reported the same pattern where at KNH, 13.4% of those that used atropine compared to 20% of those that used both drugs died and Kiambu DH reported 19.4% and 33.3% respectively. Only Nyeri PGH did not administer oximes and the data for those that used atropine showed 15% deaths.

Table 4.12: Outcome after use of atropine alone or both atropine and oximes

	OUTCOME	
	Atropine	**Atropine & Oximes**
Overall		
Well & discharged	266 (85.8)	62 (79.5)
Died	44 (14.2)	16 (20.5)
KNH		
Well & discharged	207 (86.6)	60 (80.0)
Died	32 (13.4)	15 (20.0)
Nyeri PGH		
Well & discharged	34 (85.0)	0
Died	6 (15.0)	0
Kiambu DH		
Well & discharged	25 (80.6)	2 (66.7)
Died	(6) 19.4	1 (33.3)

The differences according to the treatment given are illustrated by the figures 4.3, 4.4, and 4.5 below for overall, KNH and Kiambu DH respectively.

Figure 4.3: Overall outcome of poisoning after management with atropine and oximes

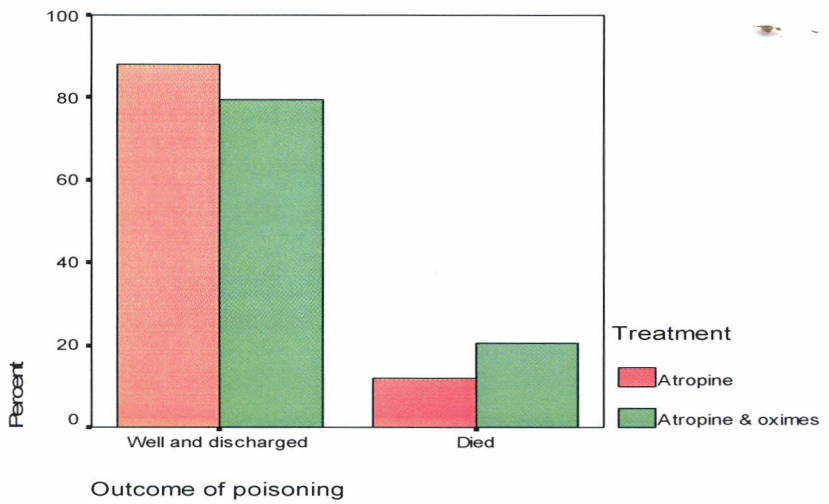


Figure 4.4: Outcome of poisoning at KNH after management with atropine and oximes

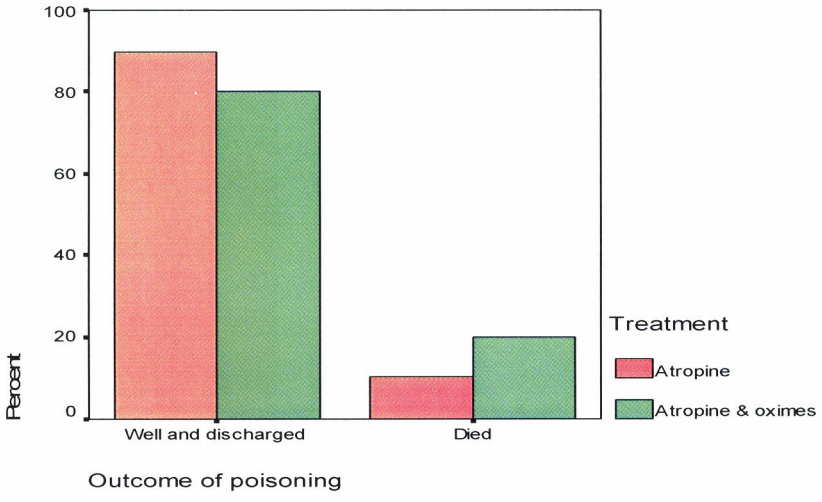
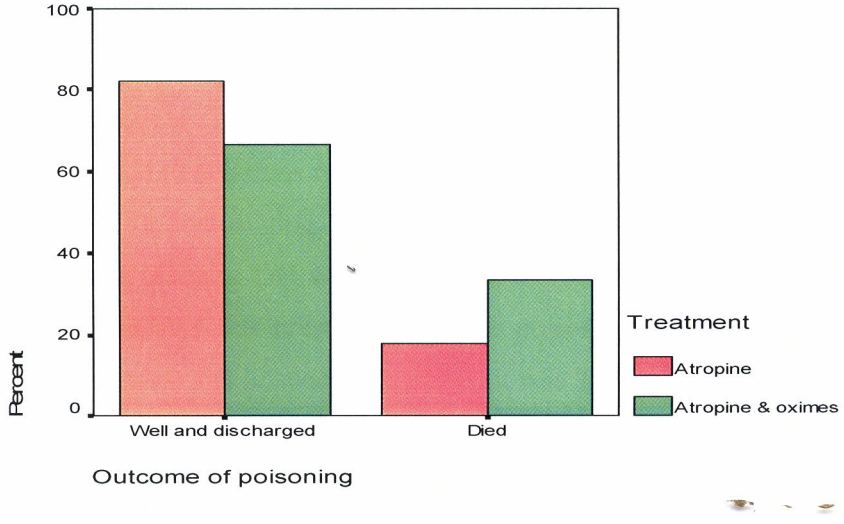


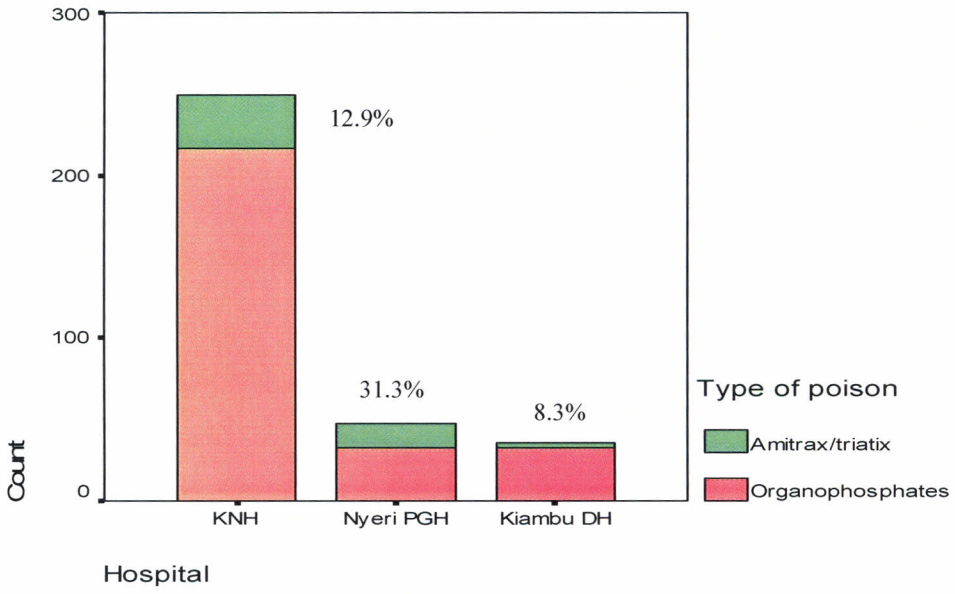
Figure 4.5: Outcome of poisoning at Kiambu after management with atropine and oximes



4.2.9 Misclassification of amitrax (triatix)

It was noticeable that amitrax (triatix) was misclassified as organophosphate in the hospitals making a proportion 15% of poisons categorized as caused by organophosphate poisoning. The misclassification was highest in Nyeri PGH (31.3%) followed by KNH (12.9%) and Kiambu (8.3%) as illustrated in the figure 4.6 below.

Figure 4.6: Misclassification of amitrax (triatix) as organophosphate poison



4.3 POISONINGS OTHER THAN DUE TO ORGANOPHOSPHATES

Other poisonings not categorized under the organophosphate group were also recorded and their descriptions are indicated in Table 4.6 below. The poisonings affected 57.8% and 42.1% male and female populations respectively. Males were the most affected group in both KNH and Nyeri PGH contributing 55.8% and 65.8% of the poisoning respectively. Similarly, contribution in Kiambu DH was high in males (60.3%) than females (39.7%). The overall median age for those that presented with this poisoning was estimated at 21 years (Range: 2 months – 84 years) and in specific hospitals it was 22 years (Range: 2 months – 78 years), 18 years (Range: 3 months – 84 years) and 20.5 years (Range: gh1.25 years – 52 years) for KNH, Nyeri PGH and Kiambu DH respectively.

4.3.1 Prevalence of poisonings other than organophosphates

Generally, estimated at 18.7%, zinc phosphide (rodenticide) contributed the highest proportion of other poisonings. The poison was 19% at KNH, 12.6% at Nyeri PGH and Kiambu DH recorded a high of 36.1%. Kerosene was also a significant poison that was treated in this category contributing 14.4% of overall poisoning. Kerosene overtook zinc phosphate in Nyeri PGH recording 19.4% compared to 12.6% of the later. Other poisons included food poisoning (10.1%), prescription drugs (8.4%), sedatives (8.3%) and amitrax/triatix (8.0%). The rest of the proportion was shared among other poisons as shown in Table 4.6. Though other pesticides/insecticides contributed to a small proportion overall (3.6%), it was considerably high in Nyeri PGH (10.4%) compared to KNH (2.0%) and Kiambu DH (4.9%).

Table 4.13: Poisonings other than organophosphates

Characteristics	Overall Count (%)	KNH Count (%)	Nyeri PGH Count (%)	Kiambu DH Count (%)
Patient's sex				
Male	741 (57.8)	557 (55.8)	146(65.8)	35 (60.3)
Female	539 (42.1)	440 (44.1)	76 (34.2)	23 (39.7)
Unidentified	1 (0.1)	1 (0.1)		
Poison name				
Amitrax (Triatix)	103 (8.0)	45 (4.5)	54(24.3)	4(6.6)
Zinc phosphide (Rodenticide)	240 (18.7)	190 (19.0)	28(12.6)	22(36.1)
Kerosene	185 (14.4)	137 (13.7)	43(19.4)	5(8.2)
Food poisoning	129 (10.1)	105 (10.5)	21(9.5)	3(4.9)
Herbicides	6 (0.5)	1 (0.1)	4(1.8)	1(1.6)
Snake venom	61 (4.8)	59 (5.9)	2(.9)	
Plants products	17 (1.3)	15 (1.5)	1(.5)	1(1.6)
Insect bites	7 (0.5)	5 (0.5)	2(.9)	
Analgesics	14 (1.1)	12 (1.2)	2(.9)	
Corrosive (Acids+Alkali)	35 (2.7)	31 (3.1)	2(.9)	2(3.3)
Carbon monoxide	18 (1.4)	17 (1.7)	1(.5)	
Methanol	9 (0.7)	9 (0.9)		
Antipsychotic drugs	36 (2.8)	27 (2.7)	8(3.6)	1(1.6)
Antiseptics/disinfectants	22 (1.7)	19 (1.9)	3(1.4)	
Antimalarial	37 (2.9)	34 (3.4)	3(1.4)	
Other pesticides/insecticides	46 (3.6)	20 (2.0)	23(10.4)	3(4.9)
Prescription drugs	108 (8.4)	97 (9.7)	6(2.7)	5(8.2)
Other acaricides	12 (0.9)	5 (0.5)	7(3.2)	
Others	23 (1.8)	19 (1.9)	2(.9)	2(3.3)
Unknown	67 (5.2)	45 (4.5)	10(4.5)	12 (19.7)
Sedatives	106 (8.3)	106 (10.6)		

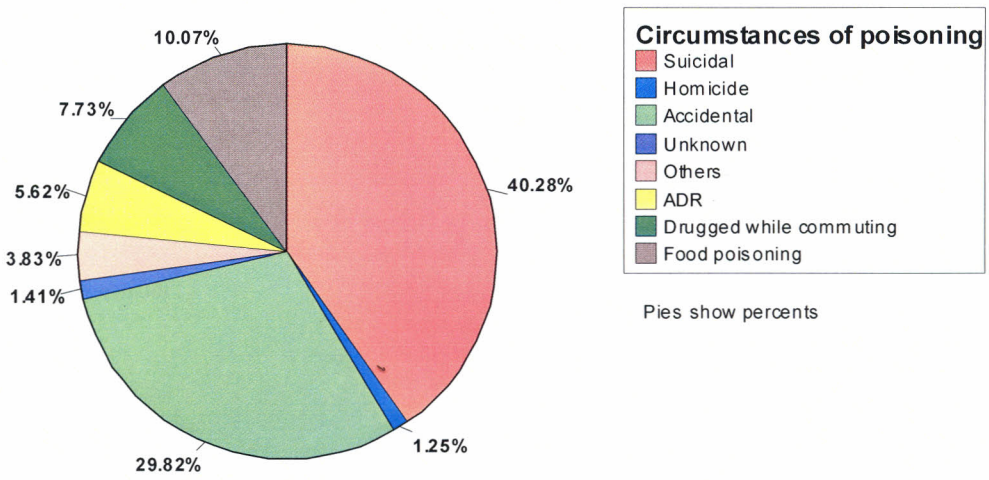
4.3.2 Circumstances of poisonings

As illustrated by figure 4.7 below, the circumstances that contributed to poisoning were mostly suicide estimated at 40.3%. Poisoning as a result of accidents was also relatively high giving a proportion of 29.8% of the circumstances that led to poisoning in this category. Similarly, food poisoning seemed to be important by contributing 10.1% of poisoning. The same trend is repeated in each of the studied hospitals with suicide contributing the highest while accidents and food poisoning were also significant. In addition, though cases of people drugged while commuting were absent in Nyeri PGH and Kiambu DH, it contributed to 9.9% of poisonings treated at KNH.

Table 4.14: Circumstances and outcome of other poisonings

Characteristics	Overall Count (%)	KNH Count (%)	Nyeri PGH Count (%)	Kiambu DH Count (%)
Circumstances of poisoning				
Suicidal	516 (40.3)	369 (37.0)	109 (49.1)	38(62.3)
Homicide	16 (1.2)	11 (1.1)	4(1.8)	1(1.6)
Accidental	382 (29.8)	282 (28.3)	88(39.6)	12(19.7)
Unknown	18 (1.4)	12 (1.2)		6(9.8)
Others	49 (3.8)	48 (4.8)		1(1.6)
ADR	72 (5.6)	72 (7.2)		
Drugged while commuting	99 (7.7)	99 (9.9)		
Food poisoning	129 (10.1)	105 (10.5)	21 (9.5)	3 (4.9)
Outcome of poisoning				
Well and discharged	1244 (97.1)	970 (97.2)	215(96.8)	59 (96.7)
Died	37 (2.9)	28 (2.8)	7 (3.2)	2 (3.3)

Figure 4.7: Circumstances of non-organophosphate poisoning



4.3.3 Association of age and the type of poisons

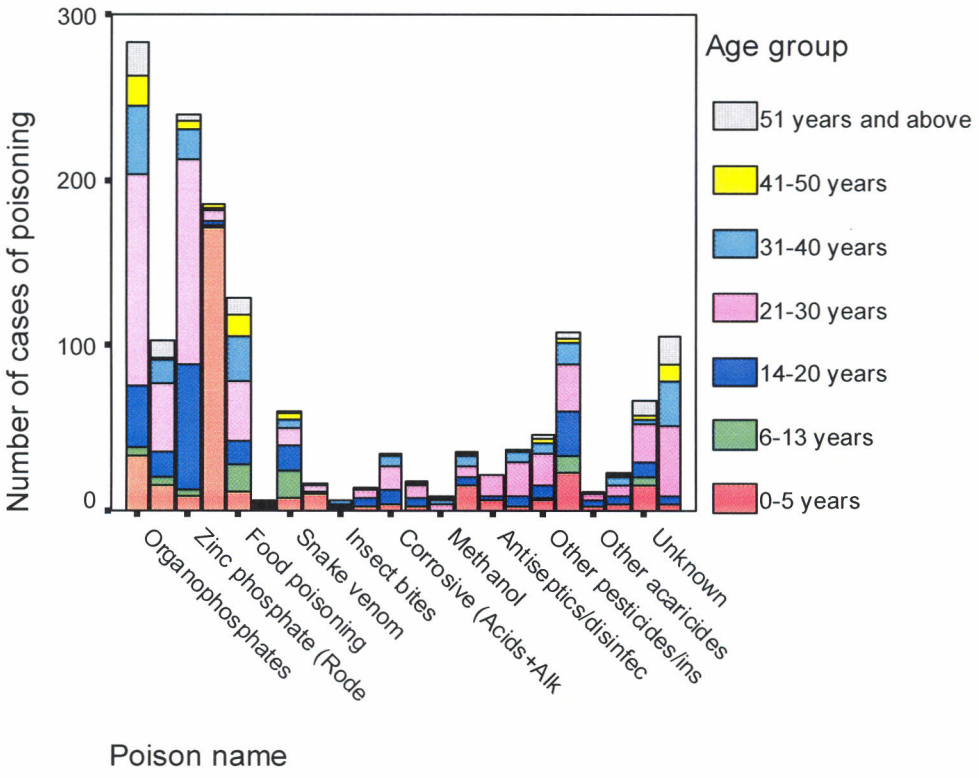
From table 4.8, the most affected age group by poisoning was 21 – 30 years at 34.9% followed by 0 – 5 years at 22.4% and 14 – 20 years at 16.2%. The age thirty years and below contributed 79.9% of all poisoning.

Among the seven age groups, the group that was highly affected by organophosphate poisoning was 21 – 30 years contributing 45.2% followed by 31 – 40 years (14.1%) then 14 – 20 years (13.4%) and 0 – 5 years (12.0%). The leading cause of poisoning among 0 - 5 years age group was kerosene with a contribution of 92.4% of the group affected.

Table 4.15: Association of age and poisons

Name of poison	Age group							Total
	0-5	6-13	14-20	21-30	31-40	41-50	=> 51	
Organophosphates	34	4	38	128	40	19	20	283
	12.0%	1.4%	13.4%	45.2%	14.1%	6.7%	7.1%	100.0%
Amitrax (Triatix)	16	5	15	41	14	2	10	103
	15.5%	4.9%	14.6%	39.8%	13.6%	1.9%	9.7%	100.0%
Zinc phosphate (Rodenticide)	9	4	76	123	18	5	5	240
	3.8%	1.7%	31.7%	51.3%	7.5%	2.1%	2.1%	100.0%
Kerosene	171	2	2	7	1	2	0	185
	92.4%	1.1%	1.1%	3.8%	.5%	1.1%	.0%	100.0%
Food poisoning	11	17	15	35	27	13	11	129
	8.5%	13.2%	11.6%	27.1%	20.9%	10.1%	8.5%	100.0%
Herbicides	1	0	1	2	1	1	0	6
	16.7%	.0%	16.7%	33.3%	16.7%	16.7%	.0%	100.0%
Snake venom	8	17	15	10	5	4	2	61
	13.1%	27.9%	24.6%	16.4%	8.2%	6.6%	3.3%	100.0%
Plants products	10	2	0	3	1	0	1	17
	58.8%	11.8%	.0%	17.6%	5.9%	.0%	5.9%	100.0%
Insect bites	1	1	0	2	2	0	1	7
	14.3%	14.3%	.0%	28.6%	28.6%	.0%	14.3%	100.0%
Analgesics	3	0	5	5	1	0	0	14
	21.4%	.0%	35.7%	35.7%	7.1%	.0%	.0%	100.0%
Corrosive (Acids+Alkali)	4	0	9	14	6	1	1	35
	11.4%	.0%	25.7%	40.0%	17.1%	2.9%	2.9%	100.0%
Carbon monoxide	2	0	6	8	0	1	1	18
	11.1%	.0%	33.3%	44.4%	.0%	5.6%	5.6%	100.0%
Methanol	0	0	0	4	2	2	1	9
	.0%	.0%	.0%	44.4%	22.2%	22.2%	11.1%	100.0%
Antipsychotic drugs	15	0	6	6	7	1	1	36
	41.7%	.0%	16.7%	16.7%	19.4%	2.8%	2.8%	100.0%
Antiseptics/disinfectants	7	0	2	13	0	0	0	22
	31.8%	.0%	9.1%	59.1%	.0%	.0%	.0%	100.0%
antimalarial	2	0	7	21	6	1	0	37
	5.4%	.0%	18.9%	56.8%	16.2%	2.7%	.0%	100.0%
Other pesticides/insecticides	7	1	8	19	6	3	2	46
	15.2%	2.2%	17.4%	41.3%	13.0%	6.5%	4.3%	100.0%
Prescription drugs	23	11	27	28	13	2	4	108
	21.3%	10.2%	25.0%	25.9%	12.0%	1.9%	3.7%	100.0%
Other acaricides	3	0	3	4	2	0	0	12
	25.0%	.0%	25.0%	33.3%	16.7%	.0%	.0%	100.0%
Others	4	0	5	7	4	2	1	23
	17.4%	.0%	21.7%	30.4%	17.4%	8.7%	4.3%	100.0%
Unknown	15	5	9	24	3	2	9	67
	22.4%	7.5%	13.4%	35.8%	4.5%	3.0%	13.4%	100.0%
Sedatives	4	0	5	42	27	11	17	106
	3.8%	.0%	4.7%	39.6%	25.5%	10.4%	16.0%	100.0%
Total	350	69	254	546	186	72	87	1564
	22.4%	4.4%	16.2%	34.9%	11.9%	4.6%	5.6%	100.0%

Figure 4.8: Proportions of poisons per age group



4.3.4 Association of age and the circumstances of poisoning

Suicidal poisoning was highest in the 21 – 30 years age group and formed a 52.3% of all suicide patients. Similarly, accidents occurred most in children of 5 years and below forming 74.3% of all accidental poisoning. Homicide was the least occurring incident of poisoning in general and when it occurred the likely victims were 21 – 30 years age group at 37.8% of all homicidal poisoning. As illustrated in figure 4.9 & 4.10, the most affected group in all the circumstances of poisoning except accidental poisoning were those aged between 21 and 30 years.

Table 4.16: Association of age and the circumstances of poisoning

Circumstances of poisoning	Age group							Total
	0-5 years	6-13 years	14-20 years	21-30 years	31-40 years	41-50 years	=> 51 years	
Suicidal	0	12	173	371	91	29	33	709
	.0%	1.7%	24.4%	52.3%	12.8%	4.1%	4.7%	100.0%
Homicide	9	5	3	14	2	4	0	37
	24.3%	13.5%	8.1%	37.8%	5.4%	10.8%	.0%	100.0%
Accidental	309	22	30	30	10	7	8	416
	74.3%	5.3%	7.2%	7.2%	2.4%	1.7%	1.9%	100.0%
Unknown	1	1	10	22	10	2	7	53
	1.9%	1.9%	18.9%	41.5%	18.9%	3.8%	13.2%	100.0%
Others	7	3	5	17	10	5	2	49
	14.3%	6.1%	10.2%	34.7%	20.4%	10.2%	4.1%	100.0%
Adverse Drug Reaction (ADR)	12	9	16	20	10	1	4	72
	16.7%	12.5%	22.2%	27.8%	13.9%	1.4%	5.6%	100.0%
Drugged while commuting	1	0	2	37	26	11	22	99
	1.0%	.0%	2.0%	37.4%	26.3%	11.1%	22.2%	100.0%
Food poisoning	11	17	15	35	27	13	11	129
	8.5%	13.2%	11.6%	27.1%	20.9%	10.1%	8.5%	100.0%
Total	350	69	254	546	186	72	87	1564
	22.4%	4.4%	16.2%	34.9%	11.9%	4.6%	5.6%	100.0%

Figure 4.9: Proportions of the circumstances of poisoning per age group

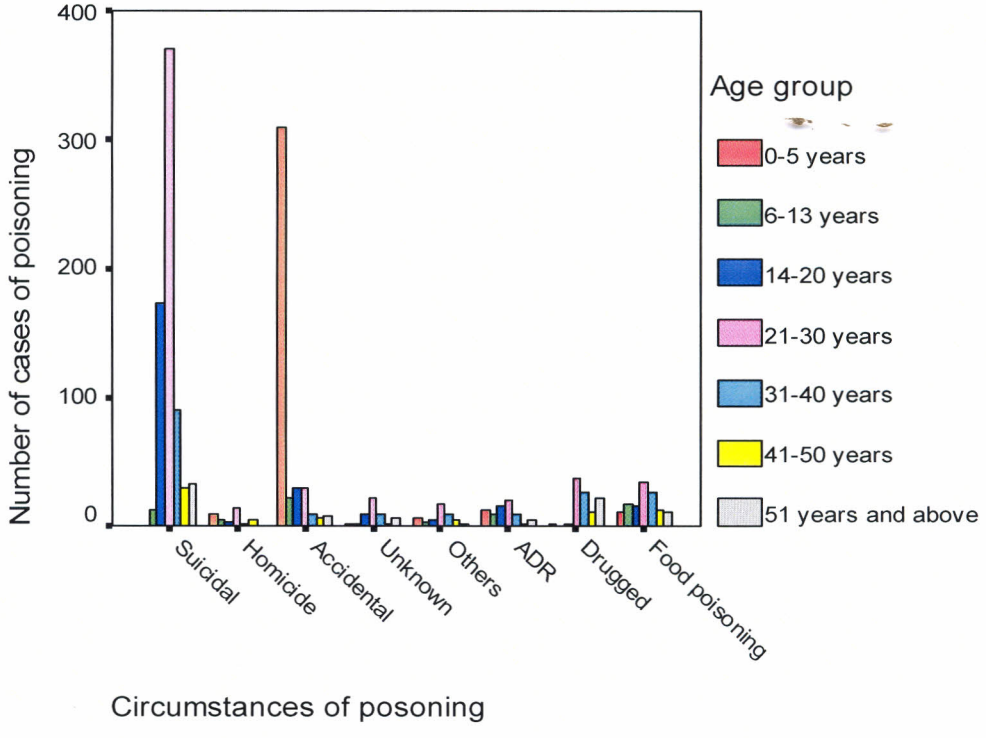


Figure 4.10: Trend of the circumstances of poisoning per age.

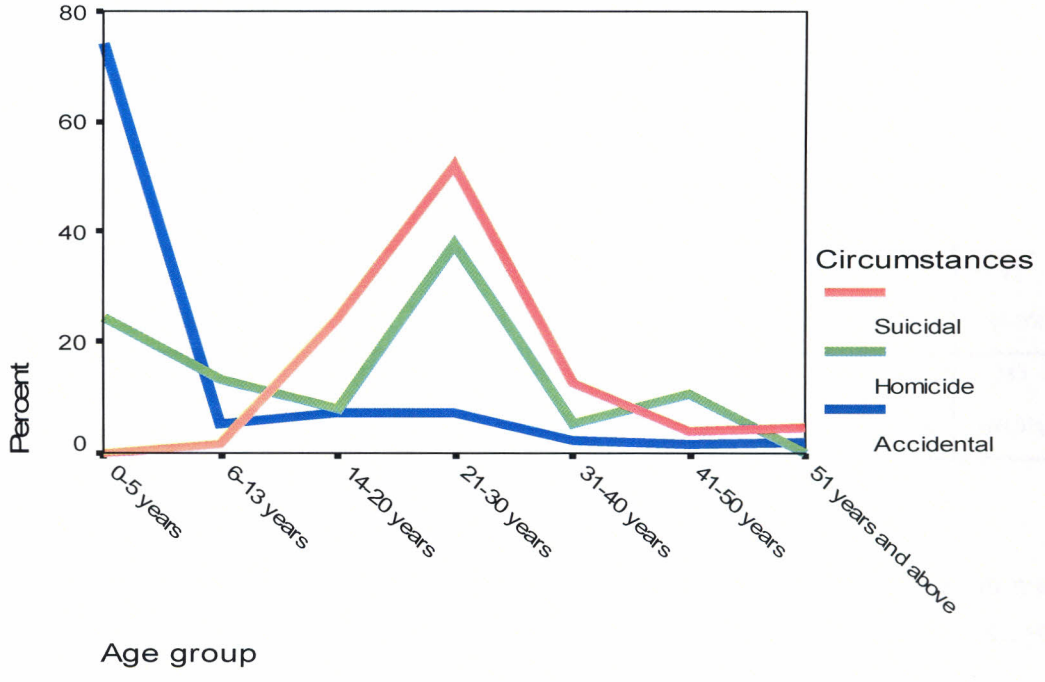
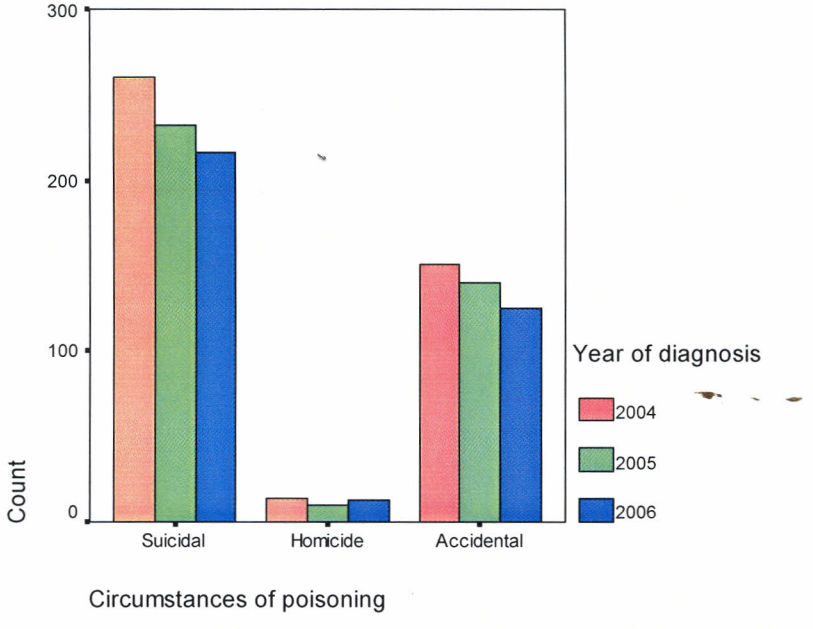


Figure 4.11: Trend of circumstances of poisoning per year



4.3.5 Association of age and outcome of organophosphate poisoning

As shown in the table 4.17, the outcome of poisoning seemed to be varying between age groups. The data showed an increasing trend from the young to the old populations. The death rate was 2.4% in 5 years and below age group and it increased to 46.3% in 21 – 30 years group then 14.6%, 12.2% and 17.1% in 31-40 years, 41-50 years and 51 years and above age groups respectively. The differences in the death rates were significantly associated with age groups of the patients (P=0.023).

Table 4.17: Association of age and outcome of organophosphate poisoning

Outcome of poisoning		Age group							Total
		0-5 years	6-13 years	14-20 years	21-30 years	31-40 years	41-50 years	51 years and above	
Well and discharged	Count	33	4	35	109	34	14	13	242
	%	13.6%	1.7%	14.5%	45.0%	14.0%	5.8%	5.4%	100.0%
Died	Count	1	0	3	19	6	5	7	41
	%	2.4%	.0%	7.3%	46.3%	14.6%	12.2%	17.1%	100.0%
Total	Count	34	4	38	128	40	19	20	283
	%	12.0%	1.4%	13.4%	45.2%	14.1%	6.7%	7.1%	100.0%

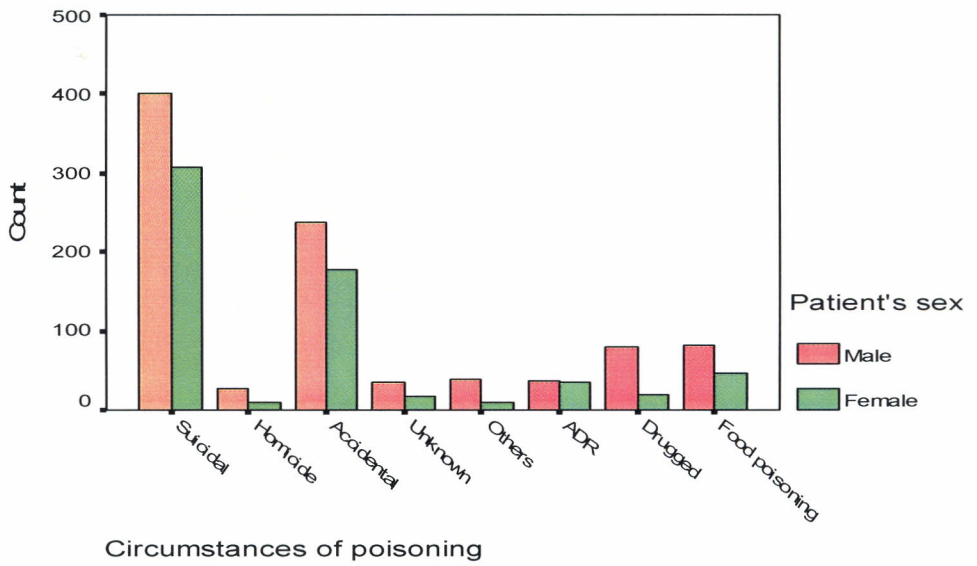
4.3.6 Association of sex and the circumstances of poisoning

As shown in Table 4.18 below, any circumstance of poisoning was more prevalent in male population than the females. Suicide, homicide and accidental poisoning affected 56.6%, 73% and 57.2% of the male population respectively. The figure 4.12 illustrates the difference between the males and females in terms of circumstances of poisoning.

Table 4.18: Association of sex and the circumstances of poisoning

Circumstances of poisoning	Patient's sex		Total
	Male	Female	
Suicidal	401	308	709
	56.6%	43.4%	100.0%
Homicide	27	10	37
	73.0%	27.0%	100.0%
Accidental	238	178	416
	57.2%	42.8%	100.0%
Unknown	35	18	53
	66.0%	34.0%	100.0%
Others	39	10	49
	79.6%	20.4%	100.0%
Adverse Drug Reaction (ADR)	37	35	72
	51.4%	48.6%	100.0%
Drugged while commuting	79	20	99
	79.8%	20.2%	100.0%
Food poisoning	82	47	129
	63.6%	36.4%	100.0%
Total	938	626	1564
	60.0%	40.0%	100.0%

Figure 4.12: Proportions of circumstances of poisoning per sex



4.4 OUTCOME OF POISONING

Out of the 1564 patients that were treated for poisoning, 78 (5%) died while 1486 (95%) were discharged well. The outcome of poisoning was significantly different in terms of whether the poison was an organophosphate or not. Organophosphate poisoning caused the highest proportion of deaths at 14.5% and the data from specific hospitals revealed a low proportion of deaths at KNH (13.4%) compared to a uniform 18.2% each for Nyeri PGH and Kiambu DH. Similarly, other poisoning caused 2.8% deaths and was distributed as 2.8%, 3.2% and 3.3% deaths in KNH, Nyeri PGH and Kiambu DH respectively.

Figure 4.13: Outcome of organophosphate poisoning

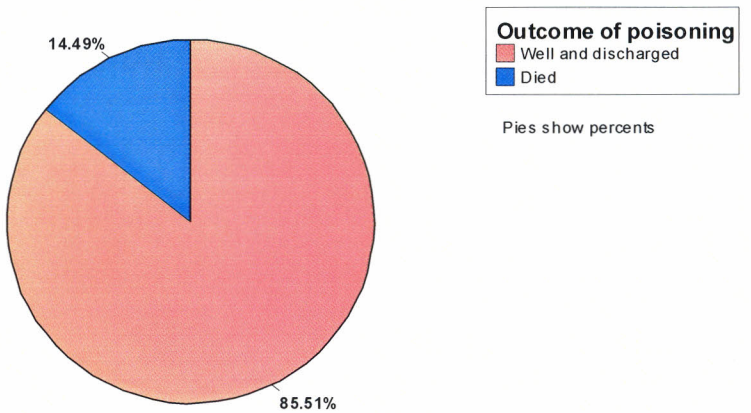
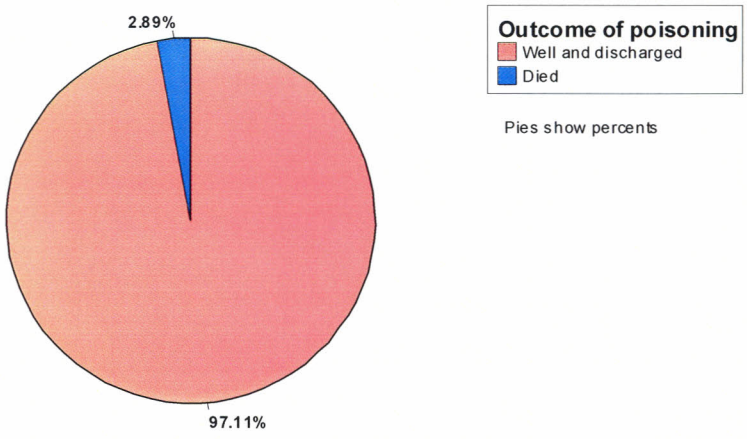


Figure 4.14: Outcome of other poisonings



4.5 KNOWLEDGE LEVELS AMONG HEALTHCARE PROVIDERS ON ORGANOPHOSPHATE POISONING

4.5.1 Characteristics of healthcare providers

Out of the 74 health providers interviewed to assess knowledge on management of organophosphate poisoning, 34 (45.9%) and 40 (54.1%) were from Nyeri PGH and Kiambu DH respectively. The study population comprised of 12 (16.2%) medical doctors, 9 (12.2%) pharmacists, 2 (2.7%) pharmaceutical technologists, 15 (20.3%) clinical officers and 36 (48.6%) nursing officers. The health providers were distributed almost evenly, as shown in table 4.19, between the two hospitals except that the two pharmaceutical technologists were found in Kiambu DH and none in Nyeri PGH. Furthermore, the sex distribution was 47 (63.5%) females and 27 (36.5%) males with an overall mean age of 35.0 years (36.2 and 33.9 years in Nyeri PGH and Kiambu DH respectively).

Table 4.19 Characteristics of healthcare providers

Characteristics	Overall Count (%)	Nyeri PGH Count (%)	Kiambu DH Count (%)
Sex			
Male	27 (36.5)	11 (32.4)	16 (40.0)
Female	47 (63.5)	23 (67.6)	24 (60.0)
Designation			
Medical doctor	12 (16.2)	7 (20.6)	5 (12.5)
Pharmacist	9 (12.2)	4 (11.8)	5 (12.5)
Pharmaceutical technologist	2 (2.7)		2 (5.0)
Clinical officer	15 (20.3)	7 (20.6)	8 (20.0)
Nursing officer	36 (48.6)	16 (47.1)	20 (50.0)

Table 4.20: Knowledge of health providers

Characteristics	Overall N (%)	Nyeri N (%)	Kiambu N (%)
Knowledge of circumstances of poisoning			
Yes	73 (98.6)	33 (97.1)	40 (100)
No	1 (1.4)	1 (2.9)	
General management knowledge			
Yes	72 (97.3)	32 (94.1)	40 (100)
No	2 (2.7)	2 (5.9)	
Knowledge of organophosphate poisoning mechanism			
Yes	45 (60.8)	20 (58.8)	25 (62.5)
No	29 (39.2)	14 (41.2)	15 (37.5)
Knowledge of organophosphate poisoning symptoms			
Yes	63 (85.1)	27 (79.4)	36 (90.0)
No	11 (14.9)	7 (20.6)	4 (10.0)
Knowledge of organophosphate poisoning management	67 (90.5)	30 (88.2)	37 (92.5)
Yes	7 (9.5)	4 (11.8)	3 (7.5)
No			
Specific therapeutic interventions			
Don't know	19 (25.7)	8 (23.5)	11 (27.5)
Atropine	41 (55.4)	20 (58.8)	21 (52.5)
Atropine & oximes	14 (18.9)	6 (17.6)	8 (20.0)
Attended training in poisoning management			
Never	10 (13.5)	3 (8.8)	7 (17.5)
More than 6 yrs	32 (43.2)	17 (50.0)	15 (37.5)
More than 3 years	23 (31.1)	11 (32.4)	12 (30.0)
Last 12 months	8 (10.8)	2 (5.9)	6 (15.0)
2 years ago	1 (1.4)	1 (2.9)	
Who organized the training?			
College	63 (98.4)	30 (96.8)	33 (100)
Hospital	1 (1.6)	1 (3.2)	
Availability of printed guidelines			
Yes	18 (24.3)	17 (50)	1 (2.5)
No	56 (75.7)	17 (50)	39 (97.5)
Location of keeping the guidelines			
Casualty	12 (66.7)	11 (64.7)	1 (100)
Wards	5 (27.8)	5 (29.4)	
Don't know	1 (5.6)	1 (5.9)	
New staff updated on guidelines			
Yes	7 (38.9)	7 (41.2)	
No	11 (61.1)	10 (58.8)	1 (100)
Review of guidelines by staff			
No	12 (66.7)	12 (70.6)	
Yes	3 (16.7)	2 (11.8)	1 (100)
Don't know	3 (16.7)	3 (17.6)	
Availability of management protocol			
No	74 (100)	34 (100)	40 (100)
Emergency kit			
Yes	63 (85.1)	30 (88.2)	33 (82.5)
No	8 (10.8)	1 (2.9)	7 (17.5)
Don't know	3 (4.1)	3 (8.8)	
Are antidotes in the kit?			
Yes	49 (66.2)	23 (67.6)	26 (65.0)
No	25 (33.8)	11 (32.4)	14 (35.0)
Antidotes in the kit			
Atropine & Plaridoxine	28 (57.1)	14 (60.9)	14 (53.8)
Others	14 (28.6)	5 (21.7)	9 (34.6)
Don't Know	7 (14.3)	4 (17.4)	3 (11.5)
Location of keeping the kit			
Yes	62 (98.4)	30 (100)	32 (97.0)
No	1 (1.6)		1 (3.0)

4.5.2 Level of knowledge among healthcare providers on organophosphate poisoning

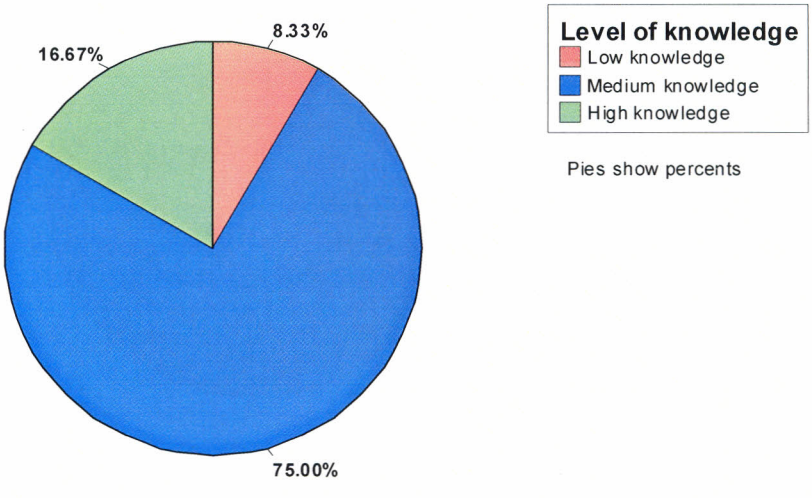
Generally, the level of knowledge in relation to poisoning and specifically organophosphate poisoning was low among the healthcare providers. 62.2% of the total healthcare providers interviewed exhibited low knowledge with only 5.4% reporting high level of knowledge in diagnosis and management of poisoning. A relatively higher proportion (70%) of health providers had low knowledge in Kiambu DH and none reported high knowledge.

There was a significant association (P=0.00) between level of knowledge and the designation of the healthcare provider. 75% of the medical doctors showed medium knowledge and 16.7% had high knowledge. Medium knowledge was also displayed by 55.6% of the pharmacists and another 33.3% had low knowledge. Clinical and nursing officers had the lowest knowledge that was exhibited by 60% and 86.1% respectively and interestingly, none of the nurses reported high knowledge. The two pharmaceutical technologists who were interviewed in the study had low knowledge of diagnosis and management of organophosphate poisoning. Figures 4.15 (a) to (e) below illustrate the proportions of health workers in terms of the level of knowledge.

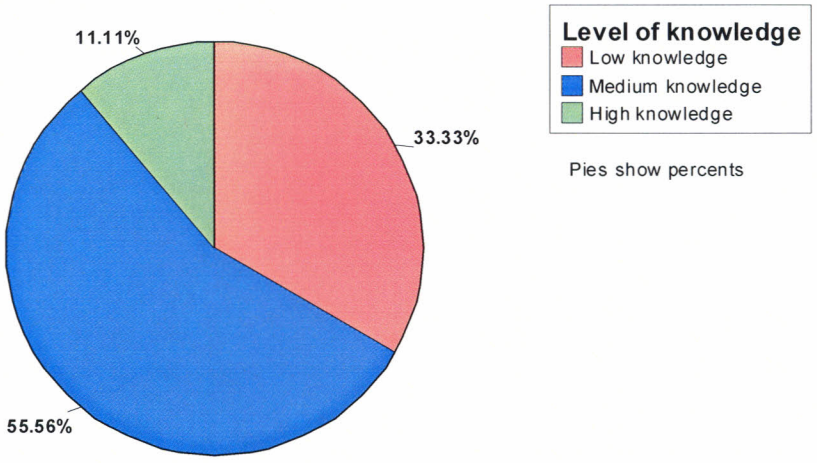
Table 4.21: Level of knowledge of health providers

Characteristics	Overall N (%)	Nyeri N (%)	Kiambu N (%)
Level of knowledge			
Low knowledge	46 (62.2)	18 (52.9)	28 (70)
Medium knowledge	24 (32.4)	12 (35.3)	12 (30)
High knowledge	4 (5.4)	4 (11.8)	

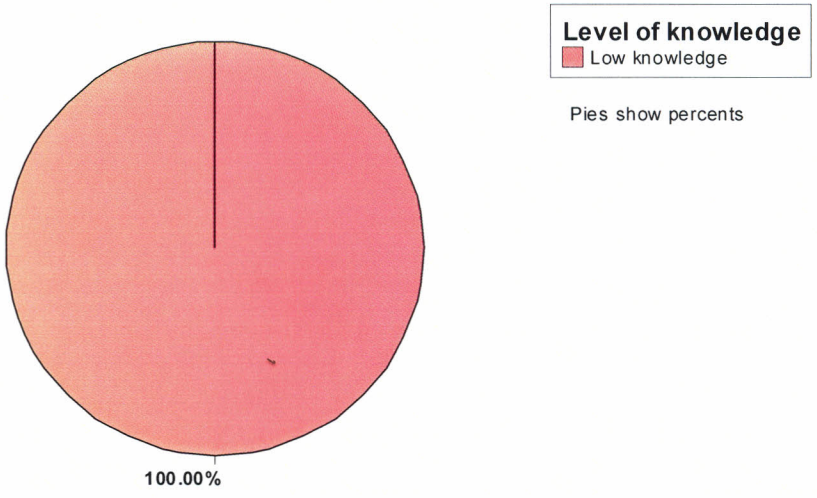
Figure 4.15
(a) Level of knowledge of medical doctors



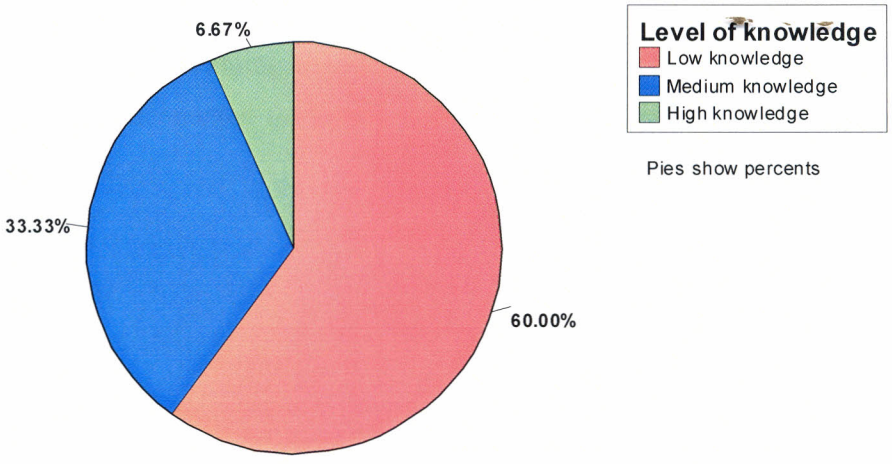
(b) Level of knowledge of pharmacists



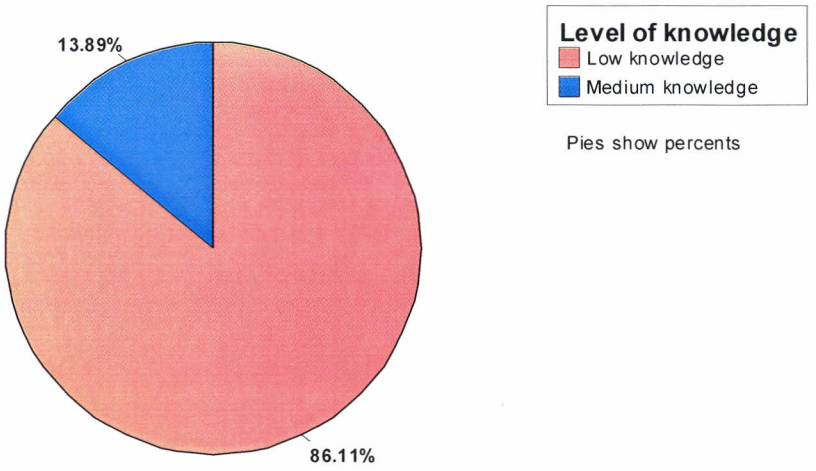
(c) Level of knowledge of pharmaceutical technologists



(d) Level of knowledge of clinical officers



(e) Level of knowledge of nursing officers



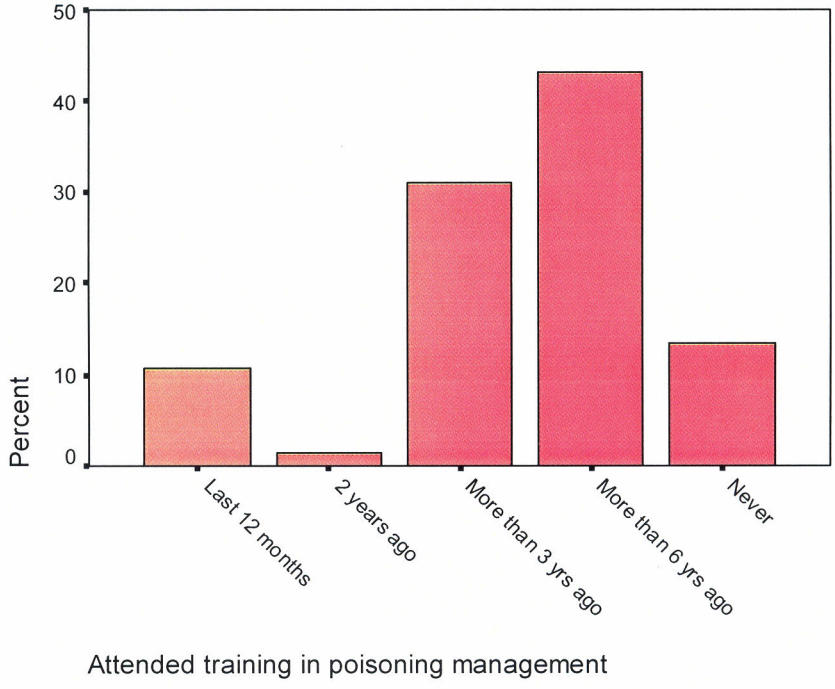
4.5.3 Knowledge on specific therapeutic management of organophosphate poisoning

Most of the healthcare providers (90.5%) interviewed said that they had knowledge on management of organophosphate poisoning and 88.2% and 92.5% knowledge was reported in Nyeri PGH and Kiambu DH respectively. However, 55.4% of the respondents knew the use of atropine for specific therapeutic intervention in organophosphate poisoning. 18.9% knew both atropine and oximes as specific therapeutic interventions and 25.7% did not know any specific interventions. Each hospital data reflected the same trend of knowledge.

4.5.4 Training on poisoning management

43.1% of the healthcare providers attended training for poisoning management more than 6 years ago while another 31.1% were trained more than 3 years ago. An overall 13.5% had never attended any training and the proportion was higher in Kiambu DH (17.5%) than Nyeri PGH (8.8%). The training reported by the healthcare providers was mainly received from college and it accounted for 98.4% with one (1) healthcare provider that reported obtaining from hospital.

Figure 4.16: Time the healthcare providers attended training in management of poisoning



4.5.5 Printed guidelines

Printed guidelines were not available according to 75.7% of the healthcare providers and it was 97.5% at Kiambu DH and 50 % at Nyeri PGH. Among those that reported the availability of printed guidelines, 66.7% were kept in the casualty and 27.8% in the wards. 61.1% of healthcare providers reported that new staff are not updated on the guidelines and it was high in both Nyeri PGH (58.8%) and Kiambu DH (1 health provider). additionally, guidelines were not reviewed by the staff as it was reported by 66.7% of the healthcare providers. Similarly, there was no management protocol reported to be available in both hospital.

4.5.6 Emergency Kit

85.1% of the healthcare providers reported that emergency kits were available in the hospitals with 88.2% and 82.5% reported in Nyeri PGH and Kiambu DH respectively. Also, 66.2% knew that the emergency kit had antidotes with little difference between Nyeri PGH (67.6%) and Kiambu DH (65%). 57.1% of those that knew antidotes as part of the kit reported atropine and plaridoxine to be in the kit. A high proportion (98.4%) knew the location of keeping the emergency kit.

CHAPTER FIVE

5.0 DISCUSSION, CONCLUSIONS AND RECOMMENDATIONS

5.1 DISCUSSION

Organophosphate poisoning was the most prevalent poisoning compared to all other poisonings. Other studies that have been conducted in other parts of the world suggest that poisoning by organophosphate poisons is higher than other poisons. Except in Nyeri PGH, organophosphate poisoning contributed the highest proportion of poisoning treated in the hospitals. Prevalence seemed to be high (45.2%) among the 21 – 30 years age group. Organophosphate poisoning was studied in urban Zimbabwe that revealed that the 21-30-year-old age group is significantly more prone to poisoning (42% of the total) than other age groups [20].

Diazinon poisoning was the most prevalent contributing 43.5% of the organophosphate poisoning. Over the 3 years studied, the proportion of diazinon poisoning was found to have increased in overall and the trend was similar for KNH while the rest of the hospitals did not show any specific pattern.

According to this study, poisoning in males is higher than in females and the same results have been reported in other studies that reported 53.1% males affected [22]. The three most prevalent circumstances of poisoning included suicides, accidents and homicides. Suicidal poisoning contributed the highest proportion and other studies have reported the same findings. In Zimbabwe, one study obtained 74% [21] and another reported 75% [20] of organophosphate poisoning was due to suicide. This study reported 68.2% overall circumstances of poisoning to be as a result of suicide. Accidental poisoning was considerably high, especially among the children of 5 years and below. In overall, accidents contributed 12% of all poisoning and accidental poisoning treated in the hospitals was 74.3% in children of age 5 years and below. A study over ten years in Zimbabwe on acute organophosphate poisoning in children reported that 93.2% of the incidences were due to accidents. Poisoning in children belonging to age group 0 – 5 years was mainly due to kerosene ingestion representing 92.4% of overall kerosene poisoning. Organophosphate poisoning in 0 – 5 years age group contributed 12% of overall organophosphate poisoning. Previous survey in 19 hospitals in Kenya reported similar findings on child poisoning with 41.09% caused by kerosene compared to 15.71% caused by organophosphates [23].

Continuous observation, re-evaluation and supportive management approaches are the cornerstone of management of a poisoned patient. This was very evident in management of

organophosphate poisoning in this study recording an overall proportion of 96.1% while KNH, Nyeri Provincial General Hospital and Kiambu District Hospital recorded 98.6%, 93.9% and 81.8% respectively.

The other principles in management are removal of unabsorbed poisons, inactivation of unabsorbed poison and removal of absorbed poison. Amongst this, removal of unabsorbed poison was the preferred one with overall usage of 75.3% with KNH at 74.7%, Nyeri Provincial General Hospital at 90.9% and Kiambu at 63.6%. Gastric lavage was the method of choice with overall usage of 88.3% with KNH at 85.2%, Nyeri Provincial General Hospital 100% and Kiambu at 95.2%.

Inactivation of unabsorbed poisons was rarely practised in all hospitals with an overall usage of 3.9% with KNH at 3.2%, Nyeri Provincial General Hospital 6.1 % and Kiambu D. Hospital at 6.1%. Usage of activated charcoal was the preferred method for inactivation of poison.

Removal of absorbed poison was rarely practised with an overall usage of 11.7% with KNH at 8.8%, Nyeri Provincial General Hospital 18.2% and Kiambu District Hospital at 24.2%. Alkalinization of urine was only practised at KNH; other method used was induction of diuresis which was done in all the three hospitals.

Use of atropine in management of organophosphate was quite high with an overall usage of 92.6% with KNH at 95.4%, Nyeri PGH at 81.8% and Kiambu at 84.8%. The route methods of administration of atropine were IV, IM SC and infusion. Among these IV was the preferred method at overall 45% with KNH at 53.1%, Nyeri PGH at 14.8% and Kiambu 14.3%. The other three methods had low preference. It was noted that overall 48.9% cases the route of drug administration was not indicated. This is where the drug was prescribed but the route of administration was not indicated. It is more often than not assumed if the route is not indicated by the prescriber then it could more probably have been given through IV. There is wide variation in the dosage of atropine administered and frequency of atropine administration. This was contributed by prescribers prescribing different dosages probably depending on the prescriber perception of patient condition and their knowledge. However, it is important to have a standardized dosage regimen which is evidence-based.

Use of oximes was low with an overall usage of 23.7% of all cases and at 29.5% at KNH, none at Nyeri Provincial General Hospital and 9.1% at Kiambu District Hospital. The reason

for non use of oximes was because it was not prescriped, which overall accounted for 93.1% with KNH at 92.8%, Nyeri Provincial General Hospital 100% and Kiambu District Hospital at 86.7%.

The outcome of poisoning showed a difference depending on whether atropine or both atropine and oximes were used to manage the poisoning. Therefore, a higher proportion of deaths were reported when oximes were used together with atropine than when atropine was used alone. Similar findings were reported overall and in KNH and Kiambu District Hospital that used oximes in managing some of the poisoning cases. The poor outcome after oximes use has been observed in other studies with mortality increased to as high as 29.1% from rate ranging between 8% and 13% when oximes are not used [18]. The observed different outcomes when oximes and atropine were used together might be difficult to explain in this study because the outcome of poisoning is dependent on several factors that include:

1. Amount of poison ingested
2. The time taken before the patient is taken to hospital
3. The toxicity of the chemical ingested
4. The supportive management available to the patient in the hospital
5. Application of key general management principles to poisoned patients
6. the correct usage and dosage of atropine until atropinisation
7. The dosage of oximes used
8. The time of initiation of oximes administration after poisoning

In the study, it is apparent that the dosages of oximes used were very low and the frequency of dosage administration was very irregular to make significant contribution to the patient condition. The recommended dosage for oximes is 1gram in 4 to 6 hourly period and the literature also quotes 12gms per day as the effective dose [19]. In all the doses given, none was near the effective dose. This might mean then the effectiveness of oximes could not be ascertained in the study cases. Further, the amount of poison ingested, time taken before the patient admission and the time of poisoning and initiation of oximes treatment could not be ascertained. Oximes have been documented to be not effective after the combination of pesticides and AChE has aged and therefore is best given in the first 36- 48 hrs [19]. Generally, organophosphate poisoning caused more deaths (14.5%) compared to the other poisons that contributed a significantly lower combined proportion of 2.8%.

The knowledge of the healthcare providers in terms of management of organophosphate poisoning was generally low. The designation of the healthcare providers seemed to

determine the level of knowledge. Medical doctors and pharmacists displayed a better knowledge on management of organophosphate poisoning as compared to the other group that included the clinical officers, nurses and the pharmaceutical technologists. This may be attributed to the level of their training because, except one (1) provider that reported receiving training from the hospital, the rest (98.4%) reported that they got their training on management of organophosphate poisoning from college education. Evidently over 80% of the healthcare providers have never or obtained refresher training on management of organophosphate poisoning more than 3 years ago.

5.2 CONCLUSIONS

In the three hospitals, the prevalence of organophosphate was generally high compared to the other poisonings. The deaths resulting from organophosphate poisoning are higher compared to death from all other poisonings. This underpins the importance with which organophosphate poisoning should be regarded. The circumstances under which organophosphate poisoning occur are generally similar to other poisoning with suicide being the most common circumstance and the age group affected most is 21 – 30 years. Accidental poisoning occurs mostly in children of ages 0 – 5 years.

The knowledge levels of organophosphate poisoning among healthcare providers differ among different professional categories with medical officers having higher knowledge followed by pharmacists, clinical officers, nursing officers and the least knowledgeable were pharmaceutical technologists. Most of the healthcare providers reported having been trained on poisoning in school/college and most of them received training on poisoning management over 3 years ago. There is no training on poisoning in hospitals and also among professional bodies as part of continuous professional development (CPD) programmes.

In management of organophosphate poisoning supportive treatment was generally in use in all the hospitals studied. The other key aspects of general management of poisoning; removal of unabsorbed poison, inactivation of unabsorbed poison were used but in varying degrees. The widely used method was removal of unabsorbed poison and gastric lavage was the preferred method. Other methods were rarely used.

In the specific management of organophosphate poisoning, atropine was widely used and the preferred route of administration was intravenous. The use of oximes was very low, with the highest use registered at Kenyatta National Hospital and no reported use in Nyeri Provincial General Hospital. The reason for non-usage was because it was not prescribed in most cases.

The use of oxime in management of organophosphate poisoning showed poor outcome compared to when atropine was used alone. This needs to be investigated further.

5.3 RECOMMENDATIONS

1. Development and establishment of Poison Management Centre or Poison Information Centre.

The Mission of the Poison Control Centre will be " To prevent poisoning, save lives and limit injury from poisoning and help decrease healthcare cost of poisoning cases". The Mandate of the Poison Centre will include:

- Provision of immediate and expert treatment advice and assistance over the telephone.
- Development of uniform poisoning patient management guidelines and their distribution to all health facilities.
- Development of data collection systems for enhanced capability to capture national poisoning data.
- Provision of answers to questions about potential poisons
- Provide poison prevention education through

  (i) Distribution of teaching materials to schools, public and private institutions and other relevant organisations.

  (ii) Media-based programs (press release, interviews, Public Service announcements, radio and TV appearances).
- Carrying out research and dissemination of research findings.

2. Minimizing the risk of poisoning through

- Reducing and eliminating possible sources of pesticide exposure to children and at work.
- Keeping pesticides out of children's reach and storing them securely in containers that are properly labeled and using child properly proof tops.
- Training healthcare providers on the recognition and management of pesticide poisoning.
- Provision of training for people on how to use pesticides judiciously and how to prevent exposure.
- Running information and education campaigns via TV and radio programmes

- Having poisoning prevention week as means for the national and local poisons centres, pharmacies, public health departments and local communities to raise awareness of the dangers of unintentional poisoning, especially among children under age five years and other poisoning and the major causes.

3. The Pharmacy and Poisons Board should liase with the Pest Control Product Board to enforce the following regarding the packaging of the organophosphate pesticides.
    - Proper labeling and use of child proof tops
    - Colour coding for packaging of organophosphate poison for ease of identification by the healthcare providers.
    - Requirement that a literature insert be provided for each package detailing the procedure for management in case of poisoning.

4. Provision of the training to all healthcare providers on:
    - Poisoning and its management
    - Importance of proper record keeping for poisoning and other medical conditions
    - Incoporation of poisoning management training as a key component of continuous profession development programs.

5. The Ministry of Public Health, Ministry of Medical Services, Ministry of Agriculture, Ministry of Livestock and Fisheries and other relevant ministries should encourage more research on poisoning through funding for research, working with research institutions, institutions of higher learning. Research finding should be dissemination via print media and electronic media.

6. Establishment of clinical toxicology department at the respective Schools of Health Sciences to spearhead the postgraduate training programs on clinical toxicology for various cadres of healthcare providers.
7. Further research need to be carried out to establish the role of oximes in management of organophosphate poisoning. A prospective study is recommended where some of the confounding factors can be controlled for example:
    a) The drugs necessary for management of acute organophosphate poisoning are provided i.e. atropine, oximes and other necessary drugs
    b) The evidence based dosages of these drugs should be used
    c) Supportive facilities for patients are available

d) All the key principles in management of poisoned patients are applied
e) The health workers specifically follow the patient from admission to the time of discharge taking the necessary patient history and the patient progress

It is recommended that this study be carried out at Kenyatta national hospital.

8. The Ministry of Health through the Office of Director of Medical Services should endevour to inform all healthcare providers that amitrax is not an organophosphate and therefore the management strategies employed should be distinguished. This can be done through a ministry circular to all heads of departments, PMO, MOH, Mission hospitals and other relevant organisations

9. Patients with exposure due to suspected self-harm, accidental ingestion, accidental contamination or potentially malicious administration should be referred to an emergency department immediately regardless of the reported doses.

10. Establishment of Intensive Care Units in District Hospitals should be considered. Currently the situation is that Intensive Care Units are up to level of Provincial Hospitals. If established, the critical patients can be managed at District Hospitals and this will avoid many referrals and also loss of patient as time is of essence in management of poisoned patients.

11. That all patients with poisoning as a result of suicidal intentions should be discharged through psychiatrist clinic which should also establish a follow up program for these patients.

12. The hospital should strive to ensure that the supplies for management of organophosphate poisoning are always in stock through establishment of emergency kits.

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ANNEX I

DATA COLLECTION FORM FOR ORGANOPHOSPHATE POISONING

HOSPITAL NAME----------

BIO DATA

Patient no.....

Age.....

Sex.....

Occupation.....

Date of Admission .....

Date of discharge.....

POISON DETAILS

Name of poison brand name.....

Approximate time when poison ingested.....

Circumstances of poisoning.....

MANAGEMENT DETAILS

1. General management of poisoning (ABCD)*

Yes ☐ no ☐

2. Prevention of absorption of un absorbed poison

Yes ☐ no ☐

If yes what was used?.....

.....

*A - Airway support

B - Breathing support

C - Cardiovascular support

D - Dextrose

3. Inactivation of un absorbed poison

Yes ☐ no ☐

If yes what was used?

....

....

4. Removal of absorbed poison

Yes ☐ no ☐

If yes what was used ?

---

5. Use of atropine

Yes ☐ no ☐

If yes what doses ?

---

6. Use of Oximes

Yes ☐ no ☐

If no why ?.....

.....

If yes, approximate time of use after ingestion of poison

.....

.....

Doses given

.....

OUTCOME

1. Well and discharged ☐
2. Discharged with complications ☐
3. Died as a result of poisoning. ☐

ANNEX II

DATA COLLECTION TOOL FOR POISONING OTHER THAN ORGANOPHOSPHATE

BIO DATA

Patient no.....

Age.....

Sex.....

Occupation.....

Date of admission.....

Date of discharge.....

POISON DETAILS

Name of poison (brand name) .....

Circumstances of poisoning.....

Outcome.....

- Well and discharged ☐
- Died ☐

ANNEX III

QUESTIONNAIRE FOR ASSESSMENT OF KNOWLEDGE LEVEL AMONG HEALTH PROVIDERS AT RURAL AGRICULTURAL HOSPITALS

Name of hospital.....

Age.....

Sex.....

Designation.....

Tel.....

1. Are you familiar with common circumstances under which poisoning occurs ?

Yes ☐ no ☐

If yes explain.....

.....

.....

2. Are you familiar with general management of poisoning ?

Yes ☐ no ☐

If yes, name the key aspects in general management of poisoning

.....

.....

3. Do you know how organophosphate causes poisoning?

Yes ☐ no ☐

If yes explain

.....

.....

4. Are you familiar with symptoms of acute organophosphate poisoning ?

YES ☐ NO ☐

If yes, which are they?

.....

.....

5. Are you familiar with management of acute organophosphate poisoning?

Yes ☐ no ☐

If yes:

a. Give details of specific therapeutic interventions

.....

b. How would you assess atropinisation ?

.....

6. When did you have training in poisoning management?

a. Never had
b. More than 6 years
c. More than 3 years
d. Last 12 months

7. Who organized your last training on poisoning management?

a. College as part of study subject
b. Hospital
c. Professional body
d. Never had training
e. Don't know

8. Does your hospital have printed guidelines for management of acute organophosphate poisoning?

Yes ☐ no ☐

If yes

a. Where are they kept?

.....

b. Are new members of staff updated on the guidelines?

Yes ☐ no ☐

c. Do members of staff regularly review the guidelines ?

i. No
ii. Yes
iii. Don't know
iv. Never been reviewed

9. Do your hospital have management protocol for acute organophosphate poisoning?

Yes ☐ No ☐

If yes explain the procedure for receiving and handling the patients

.....

.....

10. Does your hospital have an emergency drug kit?

    a. Yes
    b. No
    c. Do not know

11. Are antidotes part of the emergency kit ?

Yes ☐ No ☐

If yes:

Which antidotes are in the emergency kits?

.....

12. Where is the emergency kit kept?

    a. Pharmacy
    b. At the casualty
    c. In theatre
    d. In the wards
    e. Don't know
    f. Other places

If others, explain.....

ANNEX IV

AUTHORITY TO PARTICIPATE IN A RESEARCH INTERVIEW

CONSENT INFORMATION

RESEARCHER

I am Dr. Stephen Kimathi Kaugu a Pharmacist by Profession. I am currently undertaking a program in Clinical Pharmacy at the Faculty of Pharmacy, University of Nairobi.
The master program includes course work, practical work(wards) and clinical research in one area of interest.

RESEARCH

My area of interest is poisoning and I am carrying out a retrospective study of acute organophosphate poisoning at Kenyatta National Hospital and selected hospitals in Central Province(Nyeri Provincial Hospital, Kiambu District Hospital and Thika District Hospital)

PURPOSE OF THE STUDY

The purpose of the study is;

1) To determine the prevalence of acute organophosphate in selected hospitals in Central Province. This will elucidate the magnitude of the problem.
2) To assess the levels of preparedness of hospitals in selected rural agricultural areas of Central Province to manage cases of acute organophosphate poisoning and make necessary recommendations.
3) To assess the knowledge of organophosphate poisoning among health care providers working in selected hospitals in rural areas of Central Kenya Province. The purpose of which is identification of any knowledge gaps and formulating solutions to address them with resultant improvement in patient managements.

STUDY OBJECTIVES

- To carry out market survey of organophosphate pesticides in Kenyan market
- To determine how organophosphate poisoning is managed at Kenyatta National Hospital and selected hospitals in Central Province

- To determine organophosphate poisoning prevalence at Kenyatta National Hospital and selected hospitals in Central Province
- To assess preparedness and knowledge of organophosphate poisoning among health care providers working in selected hospitals in Central Province.

METHODOLOGY

i. It will involve review of files for poisoning for a period of three years (January 2004 to December 2006) in respective medical records department

ii. Visiting selected Agrochemical shops and determining the range of organophosphate pesticides in the Kenyan market

iii. Carrying out interviews with selected health care providers in selected hospitals in Central Province (Nyeri Provincial Hospitals, Thika District Hospital and Kiambu District Hospital). The categories of health care providers to be interviewed include;

   - Medical Officer
   - Pharmacist
   - Clinical officers
   - Nursing officers
   - Pharmaceutical technologist

SELECTION OF HOSPITALS

Hospitals in central Kenya have been selected because they are in rural agricultural potential areas where use of organophosphate pesticides is high with subsequent expectation of high incidences of organophosphate poisoning.

Kenyatta National Hospital is a National Referral Hospital and it is also adjacent to Central Province and hence acute organophosphate poisoning patients can readily be referred to Kenyatta National Hospital.

PEOPLE TO BE INTERVIEWED

The following categories of health care providers will be interviewed:

- Medical officers
- Pharmacists
- Clinical officers
- Nursing officers

- Pharmaceutical technologists

These health care professionals have been selected for interview because of their contribution in management of acute organophosphate poisoning. Their collective contribution more often than not determines the patient out come if the patient is brought to hospital in time.

BENEFITS OF YOUR PARTICIPATION

There won't be any monetary reward for participation in the research study. The information gathered in the study will guide policy development for management of acute organophosphate poisoning. This will be aimed at improving the patient care.

INTERVIEW

I will be asking you some predetermined questions to enable me know:

i. The level of preparedness in your hospitals to handle cases of acute organophosphate poisoning.

ii. The knowledge of organophosphate poisoning among health care providers.

CONFIDENTIALITY

The information which you are going to give me will be kept in confidence and will only be used for purpose of this research.

The information given to me will not be traced back to you as your name will not be written down in the questionnaire. Only your profession will be entered in the questionnaire. The raw data will only be handled by the researcher and research analyst.

PARTICIPATION IN THE STUDY

Your participation in the study is voluntary and you can refuse to participate without giving reason for your decision and there is no consequence for your refusal. You are advised to ask questions regarding the research study to help you adequately understand and make informed consent.

CONSENT FORM

1,.....

Have been adequately informed about the research study and understand the need for my participation.

I hereby agree to voluntary participation in the research study.

Signed.....

Date. .....

1, ...

Being the study researcher have adequately explained the purpose of the study to the above named person and has agreed to voluntary participate in the study.

Signed.....

Date.....

ANNEX V

KENYATTA NATIONAL HOSPITAL
Hospital Rd. along, Ngong Rd.
P.O. Box 20723, Nairobi.
Tel: 726300-9
Fax. 725272
Telegrams: MEDSUP", Nairobi.
Email: KNHplan@Ken.Healthnet.org

Ref: KNH-ERC/ 01/ 4395

7th June 2007

Dr. S.K. Kaugu
Dept. of Pharmaceutics & Pharmacy Practice
School of Pharmacy
University of Nairobi

Dear Dr. Kaugu

RESEARCH PROPOSAL: "A RETROSPECTIVE STUDY OF ACUTE ORGANOPHOSPHATE POISONING AT K.N.H AND SELECTED RURAL HOSPITALS IN CENTRAL PROVINCE, KENYA JANUARY 2004 TO DECEMBER 2004" (P101/5/2007)

This is to inform you that the Kenyatta National Hospital Ethics and Research Committee has reviewed and approved your revised research proposal for the period 7th June 2007 – 8th June 2008.

You will be required to request for a renewal of the approval if you intend to continue with the study beyond the deadline given. Clearance for export of biological specimen must also be obtained from KNH-ERC for each batch.

On behalf of the Committee, I wish you fruitful research and look forward to receiving a summary of the research findings upon completion of the study.

This information will form part of database that will be consulted in future when processing related research study so as to minimize chances of study duplication.

Yours sincerely

Dr. L. Muchiri
For: SECRETARY, KNH-ERC

c.c. The Deputy Director CS, KNH
Prof. K.M. Bhatt, Chairperson, KNH-ERC
The Dean, School of Pharmacy, UON
The Chairman, Dept. of Pharmaceutics & Pharmacy Practice, UON
Supervisors: Prof. A. N. Guantai, Dept. of Pharmacology, UON
Prof. C.K. Maitai, Dept. of Pharmacology, UON

ANNEX VI

UNIVERSITY OF NAIROBI
COLLEGE OF HEALTH SCIENCES
SCHOOL OF PHARMACY

DEPARTMENT OF PHARMACEUTICS & PHARMACY PRACTICE
P.O. BOX 19676-00202, Tel: 054 020 2721215
NAIROBI, KENYA.

8.2.08
Approved. Can commence the research

MEDICAL SUPERINTENDENT
KIAMBU DISTRICT HOSPITAL

28TH August, 2007

The Hospital Superintendent,
Kiambu District Hospital,
P.O Box 39,
Kiambu.

Dear Sir/Madam,

REF: DR. STEPHEN KIMATHI KAUGU (REG.NO. U59/8341/2005) RESEARCH FOR MASTERS IN CLINICAL PHARMACY ON ORGANOPHOSPHATE POISONING:

Dr. Stephen Kimathi Kaugu is a bona fide Master's Degree student in the School of Pharmacy, University of Nairobi. He is carrying out a research on organophosphate poisoning as part of his masters degree in clinical pharmacy programme. Your institution is among the institutions he will be carrying out his research.

Please accord him the necessary assistance.

CHAIRMAN
DEPT. OF PHARMACEUTICS
AND PHARMACY PRACTICE

Dr. K. A. M. Kuria
Chairman Dept. of Pharmaceutics and Pharmacy Practice
School of Pharmacy

c.c. Dean,
School of Pharmacy