Food Poisoning Investigation in an Under-resourced Environment: A Case Report

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Abstract

Food borne diseases are of public health importance. A food poisoning incident occurred on 24th October 2010 amongst 22 mourners who had consumed porridge one day after the funeral rites of a child in Mubachi Sub-Location of Suba West Division in Migori District, south western Kenya. An investigation of the incident was conducted with only two objectives in mind, i.e. (1) to document the poisoning incident for learning and for future reference and (2) to determine the causative factors so as to recommend appropriate public health interventions. Both patient and clinician interviews were conducted. A total of seven patients were interviewed. Patients denied any knowledge of environmental contamination within or without the homestead. They also denied careless handling of tobacco farming pesticides. The investigation established that the water used in preparing the porridge had been left uncovered on the verandah overnight and it had developed a brownish discoloration. The porridge was prepared from a mixture of cassava and sorghum flour in the ordinary way and mourners had consumed between 250ml-500ml in clean plastic cups. The victims developed signs and symptoms about 30–60 minutes after consumption. Children were affected first. They collapsed, vomited, complained of abdominal pains and had diarrhea. Other signs and symptoms occurring in both adults and children were fever, generalized body weakness, and profuse sweating. The frequency distribution of signs and symptoms showed 63% generalized body weakness, 50% vomiting, 27% nausea, 23% abdominal pain, 14% diarrhea, 9% cough and 5% restlessness. The attack rate was 100%. The suspected cause of poisoning was either hydrocyanic acid in cassava flour or pesticide contamination of the water used in preparing the porridge. In order to arrive at a differential diagnosis, an internet-based literature search was conducted to provide an overview of the signs and symptoms associated with the suspected toxicological agents. On this basis, hydrocyanic acid, organochlorine and pyrethroid pesticides were ruled out. There was a near-perfect match between the signs and symptoms of the poisoning to classical signs and symptoms of organophosphate poisoning in the literature. We conclude that the poisoning agent was an organophosphate. Though the victims denied any knowledge of environmental contamination, we strongly believe that the water used in preparing the porridge had been chemically contaminated either by careless handling of pesticides or foul play. We recommend that since the affected area and indeed the rest of Migori district are tobacco growing areas, there is need for mass health education on the safe handling and disposal of pesticide products as these are widely used. Pesticide dispensing outlets should provide also pesticide safety education to customers on each pesticide sold to encourage safe use and handling.

Keywords    Food Poisoning, Investigation, Kenya, Hydrocyanic Acid, Pesticide Handling

1. Introduction

Unsafe food causes many acute and life-long diseases, ranging from diarrhoeal diseases to various forms of cancer[1]. WHO estimates that foodborne and waterborne diarrhoeal diseases jointly cause about 2.2 million deaths annually, 1.9 million of them children[1]. This study set out to investigate an incidence of food poisoning in a Kenyan village with a view to document the poisoning incident for learning and for future reference; and to determine the causative factors of the poisoning incident so as to recommend appropriate public health interventions.

1.1. Setting

Mubachi Sub-Location has a population of about 5,723 inhabitants, composed mainly of three ethnic groups i.e. Luo, Abasuba and the Maragoli sub-tribe of the Luhya. It is the second lowest administrative area in Suba West Division of Migori District, in southern Nyanza, western Kenya. The main economic activity is tobacco farming practiced by households on small-scale farms. The literacy levels are...
fairly low, with almost half of the population hardly able to read and write.

There was a funeral of a young child in Mubachi Sub-Location on 24th October 2010. As per the local ethnic custom, friends, relatives and villagers attended the funeral on the day of the burial, but close relatives stayed on. The next day, two women were assigned responsibility to prepare porridge for the guests. The porridge was served at about 9am and about 30-60 minutes later, everyone who had taken the porridge developed signs and symptoms of food poisoning. An ambulance was called and they were rushed to hospital and arrived after one-and-a-half hours. The incident was investigated on 25th October 2010.

2. Methodology

The affected patients and the clinicians who attended to the victims were interviewed.

A total of seven (7) patients (4 females and 3 males) were interviewed using an interview guide by the investigating team. Also interviewed were the two women who cooked and served the porridge.

The laboratory technician and patient attendant who received and managed the victims in hospital were also interviewed. They were asked to describe the condition of victims on arrival to hospital and to plot a frequency distribution of the major signs and symptoms seen.

3. Results

3.1. Environmental Hygiene

Respondents denied any knowledge of pesticide containers (potential contaminants and a possible indicator for unsafe pesticide handling) strewn in the homestead. They also denied ever having used the water storage containers for mixing pesticides for spraying tobacco or any other careless handling of pesticides.

The water separately used by the two women in preparing the porridge was in 2 containers (locally known as super-drum) of 100 litres each. The water had been fetched the previous day (on the day of burial) from a local spring located 15 minutes’ walk away from the homestead. Part of the water in each container had been used during the day. During the night, it rained and each container got re-filled from the roof catchment. That night, the two containers were left outside uncovered as the people slept. The next day, water in one container (let’s call it Container A) had turned brownish but had no unusual odour.

Two sets of flour obtained and kept separately were used by the two women to prepare the porridge. In the first instance, consistent with local custom, villagers consoling the bereaved family contributed small quantities of dried cassava and sorghum that all amounted to one sack which was then milled into flour (let’s call it Flour Y) a day before the preparation of the porridge.

The other set of flour also from cassava and sorghum (let’s call it Flour Y) had been present in a household in the homestead and had been milled long before the family was bereaved.

3.2. Porridge Preparation

The 2 women separately cooked each porridge in the normal way. Water was heated to boiling point after which flour was added and systematically stirred for about 10 minutes. The mixture was then allowed to boil for a further 20-30 minutes. Utensils used in the preparation were cleaned in the morning just before the cooking began.

Porridge cooked from Flour X and water from Container A was the first to be ready and was served to both adults and children. All utensils used had been washed the same morning.

People complained that the porridge had an unusual smell though it had the usual taste.

Porridge from Flour Y and water from Container B was served about 15 minutes later and everything about it was normal – usual taste and no smell.

On average, between 250ml (half a big plastic cup) to 500ml (2 big plastic cups) were consumed per person. Everyone present within the homestead drank either one or both porridges served.

3.3. Development of Signs and Symptoms as Reported by Patients

About 30 minutes after consumption of the porridge, three children aged two years, three years and two-and-a-half years collapsed on the ground and began to vomit. They also complained of abdominal pains and had diarrhoea.

Within one hour of consumption of the porridge from Flour X and water from Container A, adults complained of abdominal pain, fever, generalized body weakness and also vomited repeatedly. Adults also complained of fever and some had diarrhoea. One adult man also collapsed and was sweating profusely. Neither difficulty in breathing nor blurred vision was reported.

People who only consumed porridge from Flour Y and water from Container B developed no signs and symptoms at all.

3.4. Vomitus

The vomitus had an offensive smell and was foamy in character. Similarly, patients also reported diarrhea with a very offensive smell. The laboratory technician observed a gaseous reaction upon pouring Jik (sodium hypochlorite) solution to disinfect vomitus on the hospital floor. The colour of vomitus and stool could not be established as patients could not recall.

3.5. Attack Rate

Everyone who consumed the porridge complained of some signs and symptoms. The attack rate was therefore 100%.
Table 1. Characteristics of water and flour (unga) used for cooking porridge

| Water source | Water Characteristics | Flour (Unga) | Flour Characteristics | Product | Outcomes |
|--------------|----------------------|--------------|----------------------|---------|----------|
| Container A  | Left uncovered on verandah overnight | Flour X | Cassava and sorghum Milled a day before burial | Uji A.X | Development of signs and symptoms 30-60 |
| Container B  | Left uncovered on verandah overnight, No recognizable change | Flour Y | Cassava and sorghum Milled long before the family was bereaved | Uji B.Y | No development of signs and symptoms |

Table 2. Frequency distribution of signs and symptoms from patient case notes (N=22)

| Signs and symptoms      | Frequency (Number) | Percentage (%) |
|-------------------------|-------------------|----------------|
| 1 Generalized body weakness | 14               | 63             |
| 2 Vomiting              | 11                | 50             |
| 3 Nausea                | 6                 | 27             |
| 4 Abdominal pain        | 5                 | 23             |
| 5 Diarrhea              | 3                 | 14             |
| 6 Cough                 | 2                 | 9              |
| 7 Restlessness          | 1                 | 5              |

Table 3. Age and sex distribution of patients (N=22)

| Age          | Frequency (Number) | Percentage (%) |
|--------------|--------------------|----------------|
| 6-12 months | 1                  | 4.5            |
| 1-4 years   | 7                  | 31.8           |
| 5-10 years  | 2                  | 9.1            |
| 10-20 years | 5                  | 22.7           |
| 20-60 years | 7                  | 31.8           |
| Total       | 22                 | 100            |

4. Discussion

Overview of toxicity of suspected toxicological agents for differential diagnosis.

4.1. Hydrocyanic Acid Poisoning

Cassava contains Hydrocyanic Acid which persists in cassava without adequate or proper processing[2]. The symptoms of hydrocyanic acid are dilated pupils; spasmodic breathing; convulsions; insensitivity; fixed eyes; spasmodic closure of jaws; very feeble pulse and speedy death[2]. None of these symptoms was observed in the patients. In addition, hydrocyanic acid tastes bitter[2] but this was not reported by any of the patients. This poison, potentially present in cassava meals is therefore ruled out.

4.2. Organophosphate Poisoning

Many organophosphates are potent nerve agents, functioning by inhibiting the action of acetylcholinesterase (AChE) in nerve cells. Organophosphorus pesticides can be absorbed by all routes, including inhalation, ingestion, and dermal absorption[3].

Patients often present with evidence of a cholinergic toxic syndrome, or toxidrome. The toxidrome elicits 3 clinical effects on nerve endings, namely: nicotinic effects at neuromuscular junctions and autonomic ganglia, CNS effects, and muscarinic effects. Nicotinic signs and symptoms include weakness, fasciculations, and paralysis, whereas CNS effects may lead to seizures and CNS depression[2].

Other symptoms include: At first a person feels sick; complains of headache; later they begin to sweat and salivate and may vomit and have diarrhea. They may also complain of stomach cramps; pupils (of the eyes) may become very small, their vision may be blurred; muscles may twitch and hands shake; breathing may become bubbly and they may have a fit and become unconscious.

In the layman’s language[4], the signs and symptoms of the onset of organophosphate poisoning are usually described as follows: At first a person feels sick; complains of headache; later they begin to sweat and salivate and may vomit and have diarrhea. They may also complain of stomach cramps; pupils (of the eyes) may become very small, their vision may be blurred; muscles may twitch and hands shake; breathing may become bubbly and they may have a fit and become unconscious.

Usually, two common mnemonics are used to remember the muscarinic signs and symptoms of the cholinergic toxidrome. These are SLUDGE/BBB and DUMBELS, as follows[3],[6],[7]. SLUDGE/BBB mnemonic means Salivation; Lacrimation; Urination (urinary incontinence); Defecation; GI upset (diarrhea); Emesis; Bronchorrhoea; Bronchospasm and Bradycardia. DUMBELS mnemonic stands Diarrhoea and diaphoresis (excessive sweating, commonly associated with shock); Urination; Miosis – constriction of the pupil of the eye; Bronchorrhoea (excessive mucal secretion from bronchial mucous membranes) bronchospasm (sudden constriction in the walls of the bronchioles), and bradycardia (slow heart rate, usually less than 60 beats per minute); Emesis i.e. vomiting; Lacrimation and Salivation.

The symptoms of organophosphate poisoning and its prognosis have also been documented by Chaudhry and his colleagues who investigated a case of organophosphate poisoning among 60 Indian men who had eaten food from a communal kitchen[8]. On the morning of the outbreak the kitchen had been sprayed with pesticide containing malathion, an organophosphate. The raw materials for cooking were stored in open jute bags. All the men developed nausea, vomiting, and abdominal pain within 3 hours of eating the food. The men were taken to a local primary healthcare centre where they received treatment for their symptoms. Fifty six (93%) responded to the treatment and were discharged home the same day. However, the condition
of the remaining four patients deteriorated. Their level of consciousness fell, and they developed respiratory distress and generalized muscular weakness. Severity of illness was greatest in one particular case who had eaten at least eight chapatti compared to the others who had eaten three or four. He developed type II respiratory failure with paralysis of thoracic, neck, and diaphragmatic muscles and on day 10 he suffered a cardiac arrest and could not be revived. The other three patients developed mild generalized muscle weakness and respiratory distress, and their level of consciousness was reduced. They responded to treatment for their symptoms, and were discharged from hospital a week later.

4.3. Organochlorine Poisoning

CNS depression and excitation are the primary effects observed from organochlorine toxicity; therefore, the patient may appear agitated, lethargic, intoxicated, or even unconscious. Organochlorines lower the seizure threshold, which may precipitate seizure activity. Initial euphoria with auditory or visual hallucinations and perceptual disturbances are common in the setting of acute toxicity. Patients may have pulmonary complaints or may be in severe respiratory distress. Cardiac dysrhythmias may complicate the initial clinical presentation[5]. Other symptoms, by system affected, include the following[5]: Pulmonary - Cough, shortness of breath; gastrointestinal - Nausea, vomiting, diarrhea, or abdominal pain; dermatological - Skin rash; and Nervous system - Headache, dizziness, or paresthesias (sensation of tingling, pricking, or numbness of one's skin with no apparent long-term physical effect. It is more generally known as the feeling of "pins and needles") of the face, tongue, and extremities. If the exposure route is ingestion, the physical examinations findings may be nausea and vomiting; confusion, tremor, myoclonus (involuntary twitching of a muscle or a group of muscles. It describes a medical sign and, generally, is not a diagnosis of a disease. The myoclonic twitches are usually caused by sudden muscle contractions), coma, and seizures. There may also be respiratory depression or failure and unusual odour - toxaphene may have a turpentine-like odour. Endosulfan may have a sulfur odour.

Toxic reactions to these compounds usually follow their accidental or suicidal ingestion. The initial symptoms therefore, are gastrointestinal (nausea and vomiting), followed by headache, dizziness, paraesthesiae of the tongue, lips and face, irritability, delirium (acute confusional state), tremors and tonic and clonic convulsions. Later, there is progressive depression of the CNS resulting in paralysis, coma and death. Rarely, severe hepatotoxicity and ventricular fibrillation may occur.5

4.4. Pyrethroid Poisoning

The symptoms of pyrethroid poisoning include[5]; Contact dermatitis; Asthma-like symptoms; Eye irritation; Skin irritation; Burning facial sensation; Itchy face sensation; Tingling face sensation; Paresthesia – sensation of numbness or tingling of the skin; Mucosal irritation; Respiratory tract irritation; Diarrhea; Headache; Dizziness; Nausea; Epigastric pain; Vomiting; Anorexia; Fatigue; Twitching muscles; Salivation; Fluid in lungs; Runny nose and Convulsions.

5. Conclusions

We conclude that the poisoning agent was most likely an organophosphate due to similarity of the signs and symptoms observed in victims with classical signs and symptoms of organophosphate poisons recorded in literature. A major limitation of the study is that no laboratory investigations on samples contaminated with the suspected toxicant were conducted, mainly due to logistical and resource constraints. Though the victims denied any knowledge of pesticide cans strewn within their compound or vicinity or careless handling of pesticides, we conclude that the water used in preparing the porridge may have been contaminated either by careless handling of pesticides or by foul play.

Since Mubachi Location and indeed the rest of Migori district are tobacco growing areas and pesticides are widely used, there is need for mass health education on the safe handling and disposal of pesticide products. Collaborative efforts are required from the relevant stakeholders to implement this recommendation, i.e. the departments of health (medical services and public health), agriculture, education, the Office of the President (Internal Security and Provincial Administration), the tobacco companies, farmers’ groups, and the media, especially ethnic language FM radio which are now common in Kenya.

Pesticide dispensing shops/outlets should provide pesticide safety education to customers on each pesticide sold to encourage safe use and handling. Public health officers and agricultural extension workers should help implement this recommendation.

REFERENCES

[1] World Health Organization. Food Safety Section[Online]. Available: http://www.who.int/foodsafety/en/
[2] Chiwona-Karlhtun, L., Brimer, L., Kalenga Saka, J. D., Mhone, A. R., Mkumbira, J., Johansson, L., Bokanga, M., Mahungu, N. M. and Rosling, H. 2004, Bitter taste in cassava roots correlates with cyanogenic glucoside levels. Journal of the Science of Food and Agriculture, 84: 581–590.
[3] Sungur M, Guven M, 2001. Intensive care management of organophosphate insecticide poisoning. Crit Care.; 5:211–215
[4] Agriculture online distance learning net[Online]. Available: http://www.agLearn.net
[5] J. Routt Reigart and James R. Roberts, 1999. Recognition and management of pesticide poisonings. 5th Ed. Washington
[6] Roberts, D. M., Aaron, C. K. 2007 Managing acute organo-phosphorus pesticide poisoning. BMJ; 334:629-34.

[7] Liang, H. K., 1996. Clinical evaluation of the poisoned patient and toxic syndromes. Clinical Chemistry 42:8(B) 1350-1355.

[8] Chaudhry, R., Bala, S., L., Mishra B., Dhawan, B. 1998. A foodborne outbreak of organophosphate poisoning. BMJ; 317:268.