Journal of Pharmacology and Toxicology

ISSN 1816-496X

Academic Journals Inc.

www.academicjournals.com

Journal of Pharmacology and Toxicology 6 (5): 505-515, 2011
ISSN 1816-496X / DOI: 10.3923/jpt.2011.505.515

Pattern of Food Poisoning in Egypt, a Retrospective Study

Z.A. Abd-Elhaleem and M.A. Abd-Elkarim
Department of Forensic Medicine and Clinical Toxicology, Faculty of Medicine, Ain Shams University, Cairo, Egypt

Corresponding Author: Z.A. Abd-Elhaleem, Department of Forensic Medicine and Clinical Toxicology, Faculty of Medicine, Ain Shams University, Cairo, Egypt

ABSTRACT

This retrospective study was conducted at the Poisoning Control Centre, Ain Shams University, Cairo, Egypt with the aim of evaluating the pattern of food poisoning. Hence, the medical profiles of 1748 food poisoning patients, admitted during the period from January 2010 to June 2010, were carefully reviewed. The greatest proportion of food poisoning occurred between the ages of 18 and 29 years, with preponderance of male gender. Most cases of poisoning were accidental. The study revealed that the most common cause involved in acute poisoning was unspecified food poisoning, followed by food contaminated with organophosphate insecticides. Ciguatera and botulism were the third and fourth agents that induced food poisoning. Botulism and organophosphate compounds were the most serious toxicities. All patients had favorable outcome. To achieve a reduction in food poisoning, measures need to be taken across the food chain-from farms to slaughterhouses, food businesses, caterers, consumers and imported foods. Implementation of good hygiene practices and enforcement of legislation are crucial. In addition, strict rules must be followed regarding the sale of insecticides. Establishing poison information centers in different parts of the country, preparing national treatment guidelines, training healthcare providers and ensuring easy availability of the antidotes are also recommended.

Key words: Organophosphate insecticides, ciguatera, botulism, hypotension, motor paresis

INTRODUCTION

Food poisoning is defined as an illness caused by the consumption of food or water contaminated with bacteria and/or their toxins, viruses or chemicals. Although the GI tract is the primary target, autonomic nervous system disturbances and CNS impairment are prominent manifestations in chemical-related, plant-related and seafood-related poisonings and in botulism (Goldfrank and Flomenbaum, 2007).

Food poisoning affects the public health and the economy in a negative way. In addition to its negative effects on human health, there are work hour losses related to inefficient working or not being able to work. The health costs can cause bigger economical losses and may result in deaths (Sahingoz and Sahin, 2009). It is widely agreed that there has been a genuine increase in food poisoning. It is likely that a combination of many factors is responsible, changing social patterns, the moves towards shopping less frequently and thus storing food for longer; the increasing use of pre-prepared dishes, which are not always stored or reheated appropriately; the trend towards eating out more often and the increase in international travel. Also, emergence of new diseases as *E. coli* O157 which was reported in England and Wales in 1982. Moreover, the increasingly

globalised food market with the variation in standards of food safety between countries could allow micro-organisms to spread quickly across the globe (Parliamentry Office of Science and Technology, 2003). This has a great influence on the society in terms of health, economy and culture (Afshari *et al*., 2004).

Food borne pathogens are virtually inescapable reaching every aspect of life. Furthermore, microbial contaminants are extremely difficult to pinpoint precision of their presence and role in food systems (Biswas *et al*., 2011).

Escherichia coli O157 is an uncommon but serious cause of gastroenteritis. This bacterium is noteworthy because a few, but significant, number of infected people develop the haemolytic uraemic syndrome, which is the most frequent cause of acute renal failure in children in the Americas and Europe (Pennington, 2010).

Botulism is a paralytic illness caused by neurotoxins synthesized by spore-forming anaerobic bacterium named *Clostridium botulinum*. It is most frequently caused by the ingestion of inappropriately processed or stored, home-canned foods, which create an anaerobic environment in which spores thrive, germinate, reproduce and synthesize the toxin. Human botulism is usually caused by toxins types A, B and E which are the most powerful poisons ever known (Arnon, 2007).

Non pathogenic food poisoning as fish borne toxins; scombrotoxin, ciguatoxin, paralytic shellfish; chemicals, heavy metals, monosodium glutamate and plants as poisonous mushrooms represent another cause of food poisoning (Tunik, 2007).

Scombroid poisoning originally was described with the Scombroidae fish (including the large dark-meat marine tuna, albacore, mackerel and kingfish). However, nonscombroid fish and marine mammals such as mahi mahi (dolphin) and blue fish also have been linked to outbreaks of poisoning. The appearance, taste and smell of the fish usually are unremarkable (Salerno and Arnoff, 2007).

Ciguatera is a food-borne illness in tropical regions related to the consumption of marine fish contaminated by natural toxins consumed through their diets. Globally, ciguatera affects between 25000 and 500 000 people per year (Dickey and Plakas, 2010). The potent neurotoxins responsible for ciguatera are predominantly ciguatoxins . These toxins enter the food chain when herbivorous fishes consume Gambierdiscus toxicus while grazing on larger reef-colonizing species of red, green and brown algae (Lehane and Lewis, 2000). The toxin accumulate in the tissues of the fishes and bioaccumulate in higher predator species of the food chain-with humans at the apex of the trophic pyramid, experiencing the full effects of bio-accumulation (Parsons *et al*., 2010). Unfortunately, these toxins are odorless, tasteless and colorless, heat stable and lipid soluble, remaining active after cooking, freezing or smoking and so, passed unnoticed (Madin *et al*., 2008).

Food contamination by chemical substances is also possible. Insecticides, rodenticides, arsenic, lead, or fluoride preparations can be mistaken for a food ingredient. Moreover, the possibility of unintentional acute heavy-metal ingestion must be considered. This type of poisoning most typically occurs when very acidic fruit punch is served in metal-lined containers. Antimony, zinc, copper, tin, or cadmium in a container may be dissolved by an acid food or juice medium (Kotsonis and Burdock, 2008).

The pattern of acute poisoning may be different even within a region or a country (Akhlaghi *et al*., 2009) Moreover, food poisoning depends on the nature of the environment, socio-cultural, socio-economic and dietary differences (Parliamentry Office of Science and Technology, 2003). It affects masses especially in places like schools, hospitals and offices where food is produced in large quantities. It is therefore essential to increase the number of studies

carried on the importance of food poisoning (Sahingoz and Sahin, 2009). Moreover, epidemiological surveillance is necessary to assess the magnitude of the problem and the major risk factors so that the preventive measures can be taken. In this context, this study was carried out to evaluate the pattern of food poisoning in both adults and children in Poison Control Centre, Ain Shams University, a referral center for poisoning, covering great area of Great Cairo, between January 2010 and June 2010.

MATERIALS AND METHODS

This retrospective study was conducted at the Poison Control Center, affiliated to Ain Shams University, Cairo, Egypt. The medical records of 1748 patients admitted due to food poisoning were retrospectively reviewed during the period from January 2010 to June 2010. Diagnosis was based on clinical picture and history of ingestion of common food or a common source of food.

The demographic data that included age, gender, residence, marital status, mode of poisoning, causative agent, clinical picture, duration of hospital stay and final outcome, were collected from the patient's files.

Patients were classified according to the causative agent into four groups' unspecified toxins (group 1), organophosphate insecticide (group 2), ciguatera (group 3) and botulism (group 4).

According to their age, the patients were categorized as preschool children (less than 6 years), school children (6-17 years), young adults (18-29 years), adults (30-39 years), middle age (40-59 years) and elderly (more than 60 years).

Continuous variables such as, age, gender, residence, marital status, mode of poisoning, severity of clinical picture, duration of hospital stay and final outcome were presented as percent. Comparing the gender, severity of clinical picture and duration of hospital stay, were carried out using chi square test. A p value of less than 0.05 was considered to be significant.

RESULTS

A number of 1687 out of 1748 patients (96.51%) presented with unspecified food poisoning (group1). Forty eight cases of food contaminated with organophosphate (OP) insecticides (2.74%) (group 2), 7 cases of ciguatera (0.4%) (group 3) and 6 cases of botulism (0.34%) (group 4) were recorded (Fig. 1).

Age and gender: In group 1 (unspecified food poisoning), young adults were the most vulnerable, involved in 36% of the cases, followed by school children and preschool children with 22 and 14%,

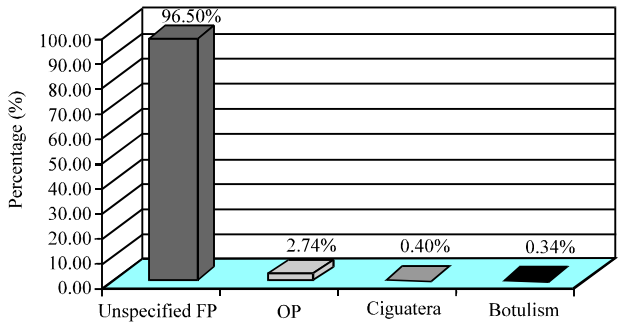

Fig. 1: Percentage of affected patients in different food poisoning groups

respectively. Adults and middle age groups showed equal percentage (13%) followed by elderly representing 2% (Fig. 2).

Regarding group 2 (OP group), preschool children were the most vulnerable age group, involved in 33.3% of the cases, followed by school children and young adults with 23 and 20.8%, respectively. Adults and middle age represented 12.5 and 8.3% followed by elderly representing 2% (Fig. 3).

In group 3 (ciguatera group), the middle age were the main affected age group (85.7%), with only one child of preschool age representing 14.3% (Fig. 4).

In group 4 (botulism), there was predominant affection of adult age group representing (66.6%) followed by equal percentage (16.7%) of young adult group and school children (Fig. 5).

As regard sex, there was higher male to female ratio (59 to 41%) in unspecified food poisoning group. In contrast, ciguatera group showed predominance of females (71.4%) to males (28.6%). Regarding OP and botulism groups non significant difference was observed between both sexes (Table 1).

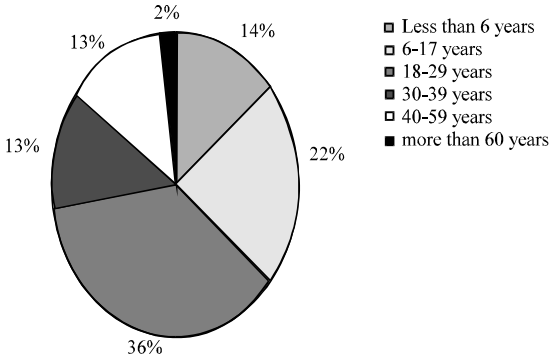

Fig. 2: Affected age groups in unspecified food poisoning

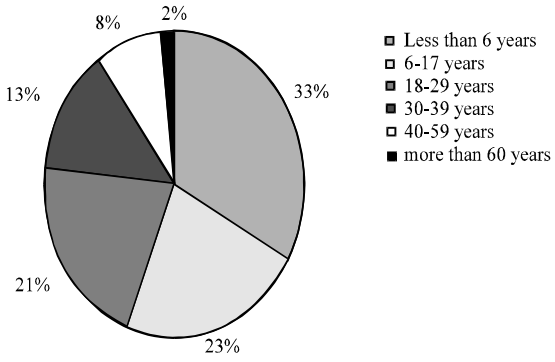

Fig. 3: Affected age groups in organophosphate food poisoning

Table 1: Chi square test of gender in different food poisoning groups

Groups of food poisoning	Male (%)	Female (%)	x	p
Unspecified food poisoning	59*	41	5.78	<0.05
OP	52	48	0.48	>0.05
Ciguatera	28.6	71.4**	33.6	<0.0001
Botulism	50	50	--	--

OP: Organophosphate, *p<0.05 significant, **<0.0001 highly significant

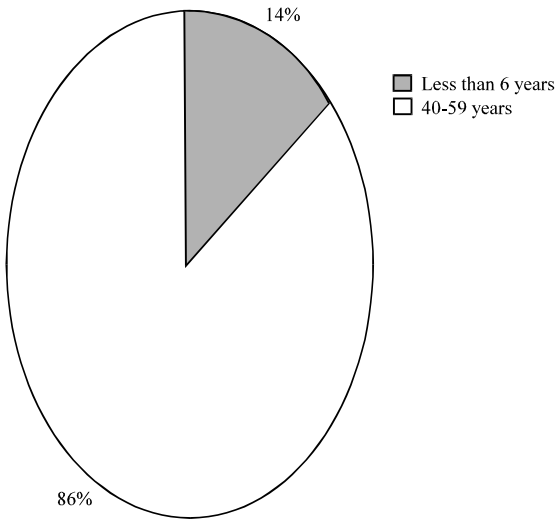

Fig. 4: Affected age groups in ciguatera poisoning

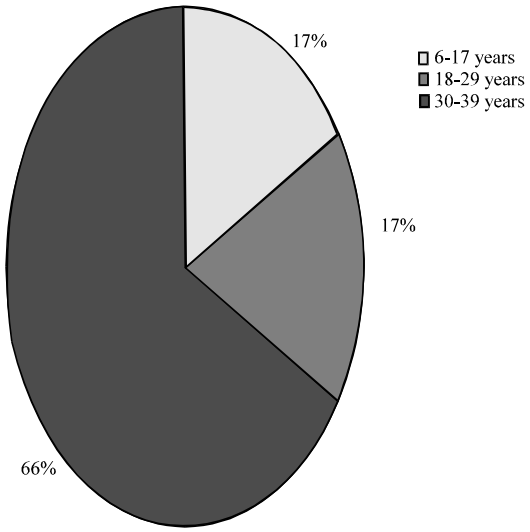

Fig. 5: Affected age groups in botulism poisoning

Residence: In unspecified food poisoning group, the ratio of urban to rural areas was 4:1, while in OP group, the ratio was 2:1. Regarding ciguatera, all cases were from urban area whereas in Botulism groups the ratio of urban to rural area was 5:1 (Table 2).

The percentage of cases refereed from urban areas in unspecified food poisoning, OP, ciguatera and botulism groups was 80, 66.7, 100 and 83.3%, respectively .On the other hand the percentage of patients refereed from rural areas was 20, 33.3 and 16.7 for unspecified food poisoning, OP and botulism groups, respectively.

Marital status: In groups 1 and 2 (unspecified food poisoning and OP groups), there was predominance of single population to married one, whereas in ciguatera and botulism groups all cases were single (Table 2).

Table 2: Variable categories of food poisoned patients (n= 1748)

Variable categories		Unspecified food poisoning (%)	OP (%)	Ciguatera (%)	Botulism (%)
Residence	Urban	80	66.7	100	83.3
	Rural	20	33.3	0	16.7
Marital state	Single	70	60.0	100	100
	Married	30	40.0	0	0
Mode of poisoning	Accidental	100	87.5	100	100
	Suicidal	0	08.33	0	0
	Homocidal	0	04.17	0	0
Final outcome	Full recovery	100	100.00	100	100
	Death	0	00.0	0	0

OP : Organophosphate

Mode of poisoning: All cases in groups 1, 3 and 4 (unspecified food poisoning, ciguatera and botulism) were accidental. As regards OP group, 87.5 % of cases were accidental whereas 8.33% of cases were suicidal and 4.17% suspicious of criminal purpose (Table 2).

Severity of clinical manifestations and duration of hospital stay: Mild clinical manifestations in the form of nausea, vomiting, diarrhea, abdominal colic and hypotension were the main presenting manifestations of unspecified food poisoning. Patients received intravenous fluids with electrolytes, H2 blockers and kept under observation with full recovery within 1 day. Abdominal colic, vomiting, diarrhea, constricted pupil were the main presenting picture of OP group (2). Other manifestations in the form of muscle fasciculation bronchorrhea, bronchospasm were also reported. Investigations revealed mild to moderate cholinesterase inhibition in all cases. Patients presented with mild symtoms (75%) recovered rapidly with atropine, IV fluids and spasmolytics, within 1-2 days, those with moderate symptoms (18.7%) required admission for longer period (3-4 days), while those who presented with severe clinical symptoms (6.25%) as pulmonary oedema and those who developed complications as intermediate syndrome were admitted for longer period (5-8 days) (Table 3,4).

In group (3) (ciguatera) tingling, numbness and motor paresis were the predominant manifestations with only one case of ataxia and vomiting. Arterial blood gases, Na and K levels were normal in sex out of seven patients (85.7%). They were admitted for 1 day, received activated charcoal, intravenous fluids with electrolytes and steroids. One patient (14.3%) developed hypernatremia and hypokalemia and was admitted to intensive care unit for 3 days where he received intravenous fluids, electrolytes and steroids.

Neurological manifestations in the form of diplopia, blurring of vision, dilated pupils, dysphoria and motor paresis were the main presenting features in patients with botulism (group 4). Gastrointestinal manifestations in the form of nausea, vomiting and constipation were also present. Arterial blood gases were normal in all cases except for one case of botulism that developed hypoxemia and respiratory alkalosis. Hypokalemia with normal Na levels were found in all cases of botulism. All cases of botulism (group 4) were admitted to intensive care unit for 4 days where they received anti-botulism antidote, intravenous fluids, electrolytes, H2 blockers and antibiotics (Table 3, 4).

Final outcome: All patients enrolled in this study were discharged from the poison control centre, with complete recovery with no recorded mortalities (Table 2).

Table 3: Chi square test of severity of clinical manifestations in different food poisoning groups

Groups of food poisoning	Mild (%)	Moderate (%)	Severe (%)	x	p
Unspecified food poisoning	95**	5.0	00.00	158.40	<0.0001
OP	75**	18.7	06.25	107.12	<0.0001
Ciguatera	0	85.7**	14.30	100.80	<0.0001
Botulism	0	16.7	83.30**	84.50	<0.0001

OP: Organophosphate, **<0.0001 highly significant

Table 4: Chi square test of duration of hospitalization in different food poisoning groups

Groups of food poisoning	<1 day	1-3 days	4 days	> 4 days	x	p
Unspecified food poisoning	97%**	3%	0	0	172.9	<0.0001
OP	70.8%**	20.8%	4.2%	4.2%	117.6	<0.0001
Ciguatera	85.7%**	0	14.3%	0	095.22	<0.0001
Botulism	0	0	100%	0	---	---

OP: Organophosphate, **<0.0001 highly significant

DISCUSSION

Acute food poisoning is a major public health problem. Most of the cases are mild and improve without any specific treatment. Some patients have severe disease and require hospitalization, aggressive hydration, and antibiotic treatment (Kotsonis and Burdock, 2008).

In the present study, 1748 cases of food poisoning were reported from January to June 2010. 96.51% of the cases were due to unspecified food poisoning with predominance affection of young adult age group (18-29 years) representing 36% followed by school children (6-17 years) representing 22%. It affected males (59%) more than females (41%). The patients were presented with nausea, vomiting and diarrhea that resolved within 1 day with intravenous fluids and electrolyte replacement.

This clinical presentation was most probably due to bacterial toxins which are the most common cause of food poisoning. This is consistent with Tunik (2007) who reported that episodes of acute gastroenteritis not associated with fever usually are caused by organisms producing toxins. The author added that bacteria toxins account for approximately 75% of cases of food poisoning and 80% of the cases of unknown causes of acute poisoning in the United States. The most common bacteria involved are *Clostridium perfringens*, *Staphylococcus aureus*, *Campylobacter Salmonella*, *Shigella*, *Bacillus cereus* and *Escherichia coli*.

These cases were unspecified as the resources to investigate and confirm a presumptive diagnosis in food poisoning are not available in Poison Control Center of Ain Shams hospitals. Sophisticated techniques such as toxin detection, matching the organism in the food by phage type with a food handler, matching an organism by phage type with other persons, isolating 10 or more organisms per gram of implicated food (Evans *et al*., 1995) or PCR identification of bacterial or plasmid DNA are potentially useful although it is generally not possible using the laboratory and personnel available in most hospitals (Brian *et al*., 1992; Goossens *et al*., 1995).

In this study, young adults (18-29 years) were the most vulnerable age group with males being more affected than females. In contrast, the previous study of Kasilo and Nhachi (1994) found preponderance of the unspecified food poisoning in the 2.1-10 years age group accounting for 79% of the total recorded cases with no significant difference between males and females indicating that both sexes were at risk of food poisoning.

Affection of young adults in the present study could be attributed to the tendency of this age group to eat outdoor especially male gender. It is thought that around half of food poisoning cases can be attributed to food consumed outside home (Parliamentry Office of Science and Technology, 2003). Many food borne outbreak investigation reports have identified the hands of food workers as the source of pathogens in the implicated food. Some activities increase the risk of finger contamination by pathogens more than others, such as the use of toilet paper to clean up following a diarrheal episode, blowing a nose, or touching raw food materials (Todd *et al*., 2010).

In the current study, hypotension was predominant in many cases. This could be due to vomiting, diarrhea with fluid depletion. The pathogenesis of diarrhea in food poisoning is classified broadly into either non inflammatory or inflammatory type. Non inflammatory diarrhea is caused by the action of enterotoxins on the secretory mechanisms of the mucosa of the small intestine, without invasion. This leads to large volume watery stools in the absence of blood, pus, or severe abdominal pain. Occasionally, profound dehydration may result. The enterotoxins may be either preformed before ingestion or produced in the gut after ingestion as in enterotoxic *E. coli*, *Clostridium perfringens*, *Bacillus cereus* and *Staphylococcus* organisms. On the other hand Inflammatory diarrhea is caused by the action of cytotoxin on the intestinal mucosa, leading to invasion and destruction. The colon or the distal small bowel is commonly involved. The diarrhea usually is bloody; mucoid and leukocytes are present. Dehydration is less likely than with non inflammatory diarrhea because of smaller stool volumes. Sometimes, the organisms penetrate the mucosa and proliferate in the local lymphatic tissue, followed by systemic dissemination as in *Campylobacter jejuni*, enterohemorrhagic and enteroinvasive *E. coli*, *Salmonella* and *Shigella* species. Moreover, in some types of food poisoning such as staphylococci, B cereus, the enterotoxins may have a dramatic effect on the emesis center in the brain and diverse other organ systems (Wei and Chiou, 2002).

The second group in this study was the organophosphates (OP) group which has a specific clinical picture. This group represented 2.74% of the total cases of food poisoning referred to our poison control centre. This small number of patients is relatively low in comparison to general cases of OP insecticides poisoning as we selected only the cases ingested food contaminated with OP insecticides.

The main age group affected was preschool children followed by school children. Young children might not discriminate the taste of food contaminated with OP. Also, the children generally respond more adversely to chemicals than adults on a dose response relationship. Furthermore, it is easy to obtain these toxic materials from the local market and so they are a common household making this age group more vulnerable to toxicity. This may also explain the increased percentage of these cases in comparison to cases of botulism and ciguatera. No significant difference was observed between males and females.

87.5% of cases were accidental whereas 8.33% of cases were suicidal and 4.17% suspicious of criminal purpose. Abdominal colic, vomiting, diarrhea, constricted pupil were the main presenting picture of this group. Other manifestations in the form of muscle fasciculation bronchorrhea, bronchospasm were also reported. Investigations revealed mild to moderate acetylcholinesterase inhibition in most of the cases.

The mechanism of toxic effect of organophosphates is based on acetylcholinesterase (AChE) inhibition in the nervous system. The OP after entering the body of an organism reaches the cholinergic sites of the nervous system and inhibits the activity of AChE by binding to its active sites. As a result of this enzyme inhibition, the substrate acetylcholine accumulates with continued stimulation of the acetylcholine receptor (Ram *et al*., 2011).

Patients presented with mild symptoms (75%) recovered within 1-2 days with atropine and symptomatic treatment. Those with moderate symptoms (18.75%) required admission for longer period (3-4 days), while (6.25%) presented with severe clinical symptoms as pulmonary oedema or developed complications as intermediate syndrome were admitted for longer periods (5-8 days).

Poisoning with OP compounds is responsible for great morbidity and mortality in developing countries. According to the World Health Organization, 1 million serious accidental and 2 million suicidal poisonings with insecticides occur worldwide every year, and of these, approximately 200,000 die, mostly in developing countries (Akdur *et al*., 2010). Mortality rates were reported to be 9.1% for adults in a study performed in Turkey by Yurumez *et al*. (2007).

Diminished mortality rate in the current study in relation to other studies may be attributed to unintentional ingestion of small, non concentrated form of OP that contaminated food. Early diagnosis and proper management might also contribute.

Cases of the third group of food poisoning, due to ciguatera, were of middle age group except one case of preschool age with no significant difference between males and females. They represented (0.4%) of total cases of food poisoning. All patients were from Port Said city (a coastal city) and presented mainly with neurological manifestations in the form of tingling, numbness, motor paresis and ataxia. Six cases out of seven recovered within 1 day of admission after receiving IV fluids electrolytes and steroids. The remaining case developed hypoxemia, hypokalemia and hypernatremia and was admitted to ICU, then recovered within 4 days of admission.

In Egypt, there were 280 reported cases of ciguatera poisoning with seven deaths in the year 2007.

The number of ciguatera cases in the present study (7 cases) is relatively few in relation to other reported cases in different areas in Egypt. This is due to the fact that great Cairo is far away from coastal cities and thus, most of patients with ciguatera poisoning might be referred to other nearby poisoning control centers. In addition, the treatment options of ciguatera poisoning are limited and supportive in nature and so, cases could be managed in any general hospitals following instructions from poisoning information centers.

The characteristic clinical picture of ciguatera reported in the studied cases is due to the binding of ciguatoxin to Na channels in diverse tissues thus, increasing Na permeability of the channels (Tunik, 2007). The variability of symptoms and difference of severity in cases under the study could be attributed to multiplicity of ciguatoxins in the same fish. Although deaths have been previously reported, there was no mortality in the cases of current study as only one case developed hypoxemia that was properly managed.

The fourth group (botulism), was diagnosed by its characteristic clinical findings matching the disease and previous exposure to suspicious food. The patients were referred from other general hospitals with nausea, vomiting, constipation and neurologic manifestations in the form of diplopia, blurred vision, dysphagia, hypotonia and motor paresis.

Botulism cases were the most severe form of food poisoning that represented only 0.34% of reported cases in the current study. This agrees with Rebagliati *et al*. (2009) who stated that although food borne botulism is uncommon, it is the most dreaded of all food poisoning that needs immediate public health attention and acute care resources.

Home-processed food accounted for 65.1% of botulism outbreaks. In Egypt, it is mostly due to uneviscerated salted fish "called faseikh" eaten by Egyptians in which spores thrive, germinate, reproduce and synthesize the toxin . This might explain that most of cases were from Port Said

(a coastal city) and of adult age group that prefer to consume fish in their diet. Also, no significant difference between males and females were also observed as families usually share the same diet.

The characteristic clinical picture of botulism cases is due to rapid and irreversible binding of botulinum toxin to the neuronal cell membrane with subsequent impairment of cholinergic transmission at all acetylcholine-dependent synapses in the peripheral nervous system (Lalli *et al*., 2003). The patients underwent complete recovery within 4 days with proper treatment. This is consistent with a recent analysis that recorded improvement of case fatality of botulism (Goldfrank and Flomenbaum, 2007). This probably represents increasing awareness of the problem associated with earlier diagnosis, appropriate and early use of antitoxin, and better and more easily accessible life support techniques

Finally, as in retrospective studies, there is some limitation as missing data of type of food ingested; time elapsed between food ingestion and clinical presentation and delay time in cases refereed from general hospitals.

CONCLUSION

Food poisoning is a common health problem in Egypt. Major age group affected was young-adult followed by school children. In particular unspecified food poisoning followed by OP insecticides contaminated food were the most common causes of food poisoning. Botulism and OP insecticides were the most serious toxicity. To achieve a reduction in food poisoning, measures need to be taken across the food chain-from farms to slaughterhouses, food businesses, caterers, consumers and imported foods. Implementation of good hygiene practices and enforcement of legislation are crucial. In addition, strict rules must be followed regarding the sale of insecticides. Establishing poison information centers in different parts of the country, preparing national treatment guidelines, training healthcare providers and ensuring easy availability of the antidotes are also recommended.

REFERENCES

Afshari, R., R. Majdzadeh and M. Balali-Mood, 2004. Pattern of acute poisonings in Mashhad, Iran 1993-2000. J. Toxicol. Clin. Toxicol., 42: 965-975.

Akdur, O., P. Durukan, S. Ozkan, L. Avsarogullari, A. Vardar, C. Kavalci and I. Ikizceli, 2010. Poisoning severity score, Glasgow coma scale, corrected QT interval in acute organophosphate. Hum. Exp. Toxicol., 29: 419-425.

Akhlaghi, M., Z. Arbabi and R. Khadivi, 2009. Pattern of acute poisoning in Shahrekord (Western Iran). Asian J. Epidemiol., 2: 9-12.

Arnon, S.S., 2007. Botulism (Clostridium Botulinum). In: Nelson Textbook of Pediatric, Kleigman, R.M., R.E. Behrman, H.B. Jenson and B.F. Santon (Eds.). 18th Ed., Saunders, Elsevier, USA., pp: 1224-1227.

Biswas, A.K., N. Kondaiah, A.S.R. Anjaneyulu and P.K. Mandal, 2011. Causes, concerns, consequences and control of microbial contaminants in meat-a review. Int. J. Meat Sci., 1: 27-35.

Brian, M.J., M. Frosolono, B.E. Murray, A. Miranda, E.L. Lopez, H.F. Gomez and T.G. Cleary, 1992. Polymerase chain reaction for diagnosis of enterohemorrhagic *Escherichia coli* infection and hemolytic-uremic syndrome. J. Clin. Microbiol., 30: 1801-1806.

Dickey, R.W. and S.M. Plakas, 2010. Ciguatera: A public health perspective. Toxicon, 56: 123-136.

Evans, M.R., S.M. Parry and C.D. Ribeiro, 1995. Salmonella outbreak from microwave cooked food. Epidemiol. Infect., 115: 227-230.

Goldfrank, L.R. and N.E. Flomenbaum, 2007. Botulism. In: Goldfrank's Toxicologic Emergencies. Nelson, L.S., R.S. Hoffman, N.A. Lewin, L.R. Goldfrank, M.A. Howland and N.E. Flomenbaum (Eds.). 9th Edn., McGraw Hill Companies, New York, pp: 394-400.

Goossens, H., B.A. Giesendorf, P. Vandamme, L. Vlaes and C. Van den Borre *et al.*, 1995. Investigation of an outbreak of *Campylobacter upsaliensis* in day care centers in Brussels: Analysis of relationships among isolates by phenotypic and genotypic typing methods. J. Infect. Dis., 172: 1298-1305.

Kasilo, O.M. and C.F. Nhachi, 1994. Food poisoning admissions in referral hospitals in Zimbabwe: A retrospective study. Hum. Exp. Toxicol., 13: 77-82.

Kotsonis, F.N. and G. A. Burdock, 2008. Food Toxicology. In: Casarett and Doull's Toxicology, The Basic Science of Poisons, Klaassen, C.D. (Ed.). 7th Ed., McGraw-Hill, New York, pp: 1191-1236.

Lalli, G., S. Bohnert, K. Deinhardt, C. Verastegui and G. Schiavo, 2003. The journey of tetanus and botulinum neurotoxins in neurons. Trends Microbiol., 11: 431-437.

Lehane, L. and R.J. Lewis, 2000. Ciguatera: Recent advances but the risk remains. Int. J. Food Microbiol., 61: 91-125.

Madin, J.S., M. O'Donnell and S.R. Connolly, 2008. Climate-mediated mechanical changes to post-disturbance coral assemblages. Biol. Lett., 4: 490-493.

Parliamentry Office of Science and Technology, 2003. Food poisoning. January 2003 No. 193, pp: 1-4. http://www.parliament.uk/documents/post/pn193.pdf.

Parsons, M.L., C.J. Settlemier and P.K. Bienfang, 2010. A simple model capable of simulating the population dynamics of *Gambierdiscus*, the benthic dinoflagellate responsible for ciguatera fish poisoning. Harmful Algae, 10: 71-80.

Pennington, H., 2010. *Escherichia coli* O157. Lancet, 376: 1428-1435.

Ram, S.B.H., C.U. Devi, C. Susma, V.R. Jasti, T.M.V. Kumar and G. Thirumurugan, 2011. Effect of polytrin C (combination pesticide) on the ach ease inhibition in plasma and brain of wistar rats. Am. J. Biochem. Mol. Biol., 1: 101-105.

Rebagliati, V., R. Philippi, M. Tornese, A. Paiva, L. Rossi and A. Troncoso, 2009. Food-borne botulism in Argentina. J. Infect. Dev. Ctries, 3: 250-254.

Sahingoz, S.A. and H. Sahin, 2009. Consumer awareness on food poisoning. Pak. J. Nutr., 8: 1218-1223.

Salerno, D.A. and S.C. Arnoff, 2007. Non Bacterial Food Poisoning. In: Nelson Textbook of Pediatric, Kleigman, R.M., R.E. Behrman, H.B. Jenson and B.F. Santon (Eds.). 18th Edn., Elsevier, New York, pp: 2918-2921.

Todd, E.C., B.S. Michaels, D. Smith, J.D. Greig and C.A. Baetleson, 2010. Outbreaks where food workers have been implicated in the spread of foodborne disease. Part 9. Washing and drying of hands to reduce microbial contamination. J. Food Prot., 73: 1937-1955.

Tunik, M.G., 2007. Food Poisoning. In: Goldfrank's Manual of Toxicologic Emergency. Nelson, L.S., R.S. Hoffman, N.A. Lewin, L.R. Goldfrank, M.A. Howland and N.E. Flomenbaum (Eds.). 9th Edn., McGraw Hill Companies, New York, pp: 386-394.

Wei, H.L. and C.S. Chiou, 2002. Molecular subtyping of *Staphylococcus aureus* from an outbreak associated with a food handler. Epidemiol. Infect., 128: 15-20.

Yurumez, Y., P. Durukan, Y. Yavuz, I. Ikizceli and L. Avsarogullari *et al.*, 2007. Acute organophosphate poisoning in university hospital emergency room patients. Intern. Med., 46: 965-969.