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Case Series

Ten Real Critical Iatrogenic Errors in Clinical Toxicology Practice

Ahmed Refat Ragab Ali*

Department of Forensic Medicine and Clinical Toxicology, Mansoura University, Egypt

***Corresponding author**

Ahmed Refat Ragab Ali, Department of Forensic Medicine and Clinical Toxicology, Faculty of Medicine, Mansoura University, Mansoura, Egypt, Email: ahmedrefat1973@yahoo.com

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Abstract

The emphasis of the extant investigative research was on tracking patients with the most dangerous iatrogenic error in severe toxicological emergencies. The very first reported instance was that of a female patient with a dangerous intoxication that was caused due to caustic ingestion with a prescribed mega dose of methyl-predinisolone 30 mg/kg; she who visited the ER post seven days with a severe haematemisis attack. The next case was that of a severe atropine toxicity reported in a boy 4 years old reportedly suffering from a bout of food poisoning which was incorrectly diagnosed to be that a severe organophosphate toxicity and who was medically injected with five ampoules of atropine in a span of 20minutes. The third case comprised of seven instances of severe organophosphate poisoning who were rushed to the ER due to mixed atropine toxicity that was due to the acceleration of the procedure of complete atropinization without controlling the dosage/time schedule. The fourth case was a typical referral instance of mass food poisoning accident in five members of the family that was simultaneously identified to be a pure case of accidental toxicity due to leakage of carbon monoxide gas from their domestic bio gas system. The fifth case was an unsure choice to perform haemodialysis in five different cases of individuals reporting dangerous methanol consumption resulting in extreme diagnosis of irreversible blindness and grave worsening condition while sick. The sixth iatrogenic instance was an erroneous diagnosis in the strength of ethanol (10% or 100%) administered orally which resulted in a serious diagnosis in a 25 year old pharmacist who deliberately consumed 100 ml of pure methanol. The seventh case was that of a 51 year old female farmer who was not carefully observed and discharged untimely in a case of suspected snake bite after 4 hours; this led to her being re-admitted in the ER after 18 hours due to extreme progressive descending neuroparalytic symptoms.The eight case was a strict one that followed the intended dosage mentioned on the antivenin vial “One ampoule given intramuscularly” which resulted in a serious diagnosis of Disseminated Intravascular Coagulation by collaburdiae snake bite. The ninth case was that of a suicidal 37 year old female patient who had iatrogenic induction of vomiting due to consumption of the Zinc Phophite tablets which resulted in acceleration of the release of phophine gas and a quickly deteriorating deadly worsening of the inebriated condition. The last case was of cases of neuroleptic malignant syndrome prescription of medication to alter the neurotransmitter disturbance that was the due to the antipsychotic medicines that may result in more disturbance and dangerous result from anticholinergic medications that was due to paralytic ileus in a lady aged 46 years.

INTRODUCTION

Clinical toxicology is one of the rapidly advancing new fields in the emergency room. One of the most characteristic points in this field is the marked variation in the theory of management.

Regarding corticosteroid therapy in management of cases of caustic ingestion, the most serious complication of corrosive damage to the oesophagus besides perforation is stricture formation. The role of corticosteroids in preventing corrosive-induced strictures is controversial. The recent research suggests that systemic corticosteroids are not beneficial for second- and third-degree corrosive oesophageal burns [1].

As regards organophosphorus pesticide poisoning, this is an important clinical problem in rural regions of the developing world. Diagnosis is made on the basis of clinical suspicion, the characteristic clinical signs, smell of pesticides or solvents, and reduced acetylcholinesterase activity in the blood [2]. Patients

with severe organophosphorus poisoning typically present with pinpoint pupils, excessive sweating, reduced consciousness, and poor respiration. The major differential diagnosis is carbamate poisoning, which is clinically indistinguishable [3].

Regarding carbon monoxide (CO), this is a gas that has no odour or colour, but it is very dangerous. It can cause sudden illness and death. CO fumes can build up in places that do not have a good flow of fresh air. You can be poisoned by breathing them in. The most common symptoms of CO poisoning are headache, dizziness, weakness, nausea, vomiting, chest pain and confusion. It is often hard to tell if someone has CO poisoning because the symptoms may be like those of other illnesses. People who are sleeping or intoxicated can die from CO poisoning before they develop symptoms [4,5].

Methanol poisoning can produce significant toxicity in humans, including acidosis, blindness, and death. The current mainstay of therapy is haemodialysis to correct acidosis and remove both the parent compound and toxic metabolite, and alcohol dehydrogenase (ADH) inhibition to prevent formation of formic acid [6].

Snake bite is one of the most neglected public health issues in poor rural communities living in the tropics. Despite the increasing knowledge of snake venom composition and mode of action, good understanding of clinical features of envenoming and sufficient production of antivenom by specialized manufacturers, snake bite management remains unsatisfactory in many regions [7].

Aluminium and zinc phosphides are highly effective insecticides and rodenticides and are used widely to protect grain in stores and during transportation. Acute poisoning from these compounds may be directly due to ingestion of the salts or indirectly from accidental inhalation of phosphine generated during their approved use. Both forms of poisoning are mediated by phosphine, which has been thought to be toxic because it inhibits cytochrome oxidase.

There is usually only a short interval between ingestion of phosphides and the appearance of systemic toxicity. Phosphine-induced impairment of myocardial contractility and fluid loss leads to circulatory failure, and critically, pulmonary oedema supervenes, though whether this is a cardiogenic or non-cardiogenic is not always clear

Finally, the neuroleptic malignant syndrome is one of toxicological dilma in the emergency toxicological case practice. Neuroleptic malignant syndrome refers to the combination of hyperthermia, rigidity, and autonomic dysregulation that can occur as a serious complication of the use of antipsychotic drugs. The trend of some scientific research is to recommend overwhelming the spastic condition of the muscle by multiple chemical medications despite the huge magnitude of their adverse effects.

THE AIM OF THE WORK

The aim of this study is to present 10 cases of acute poisoning with various toxic substances with a high risk of iatrogenic error, diagnosed in the Toxicology Unit – Emergency Hospital - Mansoura University. I describe the clinical presentation, manifestations, management and treatment with the critical iatrogenic error of each type of poisoning.

RESULTS

The description of clinical case series:

In the current research we describe 10 real practical cases of toxicological emergency situations with various iatrogenic errors in the history taken, order of investigation and finally the line of management.

In the first condition, a female adolescent, 17 years old, attended the ER department after a suicide attempt and ingestion of about 150 ml of diluted NaOH solution. The patient appeared ill with caustic eschars in the mouth and liquafactive necrosis. She was put under observation for 12 hours for any developing laryngeal oedema or respiratory or gastrointestinal complaints. The laryngoscopic specialist recommended the patient to be on a mega dose of corticosteroid “Predinisolone 30mg/kg” without endoscopic examination. Also he recommended a brief course of antibiotics to prevent secondary infection. After a lapse of six days the patient represented with an acute attack of haematemisis and was immediately hospitalized and after endoscopic visualization the intervention therapeutic manoeuvre was conducted to stop the progression of the haemorrhagic condition. After 10 days the patient was discharged with long follow-up term therapy for oesophageal stricture pathology.

The second reported condition was a 4-year-old boy who presented with a typical characteristic picture of food poisoning, two attacks of vomiting and mild abdominal colic. The general practitioner, in a panicked state, believed the cause to be organophosphate poisoning despite the absence of any typical muscrurinic, nicotinic, neurological, or even neuromuscular manifestations. He immediately decided to start the atropinization of the patient without any calculation of atropine dosage. Four mg was immediately given to the patient as repeated bolus doses within 5 minutes. Immediately the child start to develop the toxic manifestations of acute atropinization in the form of acute rapidly progressive disturbed consciousness level with dilated sluggish reactive pupils(5 mm), dryness of all body secretions, and hyperthermia (39.7 °C) with a plethoric body.

In this condition we seven cases of patients represented with manifestations of acute severe organophosphate toxicities referred from a variable primary care unit and secondary hospitals after aggressive management with atropine therapy. For example, one of them received about 46 ampoules of atropine within 1.30 hours. These patients showed, beside their cholinergic manifestations that varied from “vomiting, incontinence of urine and stool, pulmonary secretion and bronchospasm and fasciculation”, manifestations of anticholinergic toxicity in the form of hyperthermia, tachycardia, irritability, visual and auditory hallucinations”.

The fourth condition a family consists of four members: a mother, two daughters and one son. They were found collapsed inside their home. The accident audience noticed recurrent vomiting of all family members, and the first diagnosis of the ER physician was that all of these family members were suffering from food poisoning. Fluid replacement therapy was started

Table 1: Clinical presentations, laboratory findings, management lines, prognostic criteria of the studied cases.

No	v	Sex	Mode of Poisoning	Clinical Presentations	Laboratory Findings	Management lines	Prognosis
1	17	Female	Intentional ingestion	Before Iatrogenic Error			Recovery after 17 days from time of exposure with pathological esophageal stricture injuries.
				Caustic erosions in buccal mucosa & tongue. Pain in epigastric region.	Normal CBC, Electrolytes level and Creatinine.	Antibiotics, Corticosteroids,	
				After Iatrogenic Error			
				Haematemisis	Endoscopic results revealed 3rd degree caustic erosions.	Endoscopic Sclerotherapy.	
2	4	Male	Accidental ingestion of contaminated food.	Before Iatrogenic Error			Recovery after 80 hours from time of onset of iatrogenic atropine therapy.
				Vomiting and abdominal colic.	———	Atropine	
				After Iatrogenic Error			
				Dilated pupil, hyperthermia 39.7 °C, Comatose, Dryness of body secretion and redness.	Choline-esterase enzyme 2426 IU/L	Cold fomentation, Fluids and Prostigmine.	
3	24	Female	Intentional ingestion of organophosphates pesticides	Before Iatrogenic Error			Progressive pulmonary distress ending by ventilatory support and grave respiratory failure.
				Vomiting, incontinence of urine and stool, pulmonary secretion and bronchospasm and fasciculation	Choline-esterase level 54 IN/L (1800-3600 IU/L)	Atropine and fluids	
				After Iatrogenic Error			
				Cholinergic signs fever, tachycardia, irritability, visual and auditory hallucinations.	Increase bronchial marking in the pulmonary X ray.	Oximes and Atropine	
4	31 14 9	Female Female Male	Unintentional exposure to butagase leakage.	Before Iatrogenic Error			Recovery and discharge after 36 hours
				Drowsy and repeated vomiting.	———	Spasmolytic, antiemetic and fluid therapy.	
				After Iatrogenic Error			
				Disturbed consciousness level and hypotonia and hyporeflexia.	Metabolic Acidosis 7.31, 7.29 and 7.28. Carboxyhaemoglobin level 17.5, 23.7 & 32.1 gm%.	Normo-baric Oxygen in well tight mask.	
5	23 37 31 40 18	Males	Intentional ingestion of cheap alcoholic beverages. "Methanol Contaminated"	Before Iatrogenic Error			Recovery with mild visual impartment (case 1,2), and recovery with complete loss of vision "case 3, 4, 5".
				Vomiting, abdominal colic.	Metabolic acidosis (7.15-7.26). Oedema of optic disc.	Fluid administration, Ethanol ingestion and leucovorine.	
				After Iatrogenic Error			
				Blurring of vision, severe metabolic acidosis and disturbed consciousness level.	Severe Metabolic Acidosis – Retinal Haemorrhage.	Haemodialysis	
6	25	Male	Intentional ingestion of cheap alcoholic beverages. "Methanol Contaminated"	Before Iatrogenic Error			Grave prognosis cardiorepiratory arrest after 5 days.
				Vomiting, abdominal colic. Blurring of vision and disturbed consciousness level. GCS: 13.	Metabolic acidosis 7.28. Methanol blood level. 60 mg/dl.	Ethanol by ingestion 1ml/kg of 70% conc.	
				After Iatrogenic Error			
				Comatose, Respiratory failure, GCS:5.	Metabolic acidosis 7.1, Methanol blood level. 55 mg/dl.	Five dialysis sets with duration "2-5" hours and leucovorine.	
7	51	Female	Unintentional exposure to snake bite.	Before Iatrogenic Error			Complete recovery and discharge after 6 days
				History of snake bite in the inner aspect of the sole of the right foot	———	Put under observation.	
				After Iatrogenic Error			
				Progressive descending neuroparalytic manifestations with ptosis, dysarthia, dysphagia and difficulty in breathing.	Positive myoglobinuria	IV infusion of antivenin antidotes "10 vials, 5 vials in every set with 6 hours interval.	

8	46	Male	Unintentional exposure to snake bite.	Before Iatrogenic Error			Fata outcome with rapid progressive DIC and death within 18 hours from the time of admission.
				Mild swelling and redness, oedema, haemorrhage in the index finger of right hand	———	Single IM vial of antivenin in the gluteal region.	
				After Iatrogenic Error			
				Progressive swelling, oedema, haemorrhage and gangrenous changes and disseminated intravascular coagulation.	Prolonged prothrombin time. Increase SGPT and SGOT enzymes.	Four set of antivenin were given five vial in each time vs IV infusion with 5 hours apart.	
No	Age	Sex	Mode of Poisoning	Clinical Presentations	Laboratory Findings	Management lines	Prognosis
9	37	Female	Intentional ingestion of one tablet of Zinc Phosphaide	Before Iatrogenic Error			Recovery after 72 hours from the time of admission.
				Chest tightness	———	Induction of vomiting by ingestion of large amount of bottle of water.	
				After Iatrogenic Error			
				Difficulty in breathing, palipation, dizziness, hypotension.	Mild pulmonary oedema.	Repeated gastric suction and saline 0.9% IV infusion.	
10	46	Female	Unintentional exposure to snake bite.	Before Iatrogenic Error			Fatal outcome and death from 96 hours from the time of the admission.
				Hyperthermia 39.4 ° C, rigidity,	Leucocytes count = 18.000, Creatinine phophokinase = 487 IU/L	Benzodiazepine, dantrolene, bromocritine, amantadine, apomorphine, dopamniergic agonist.	
				After Iatrogenic Error			
				Paralytic ileus, electrolytes disturbances, severe acute abdomen.	Erect plain X-Ray multiple air fluid levels.	dantrolene, bromocritine & paralytic ileus measurements.	

along with adjunctive management. By toxicological consultation for these case, the clinical toxicologist noticed markedly disturbed consciousness level with mild degree of metabolic acidosis for all family members "7.31, 7.29 and 7.28" mother, daughter and son respectively. The clinical toxicologist immediately ordered blood carboxyhaemoglobin levels to be checked and the results were 17.5 gm%, 23.7 gm% and 32.1 gm% for mother, daughter and son respectively. The line of management was immediately transferred to acute carboxyhaemoglobin toxicity management. After returning home one the patient's attendant found a biogas smell inside the home and there was a leakage from a heating stove.

The fifth clinical situation of the current case series related to five different patients with acute methanol toxicity and with severe gastrointestinal manifestations in the form of protracted vomiting, severe abdominal colic, neurological manifestations such as a variable degree of disturbed consciousness level and blurring of vision with marked metabolic acidosis. Facilities for laboratory toxicological detection of methanol blood level were not available. The diagnosis of acute methanol toxicity was dependent only on history taken and clinical evaluation. Immediate dialysis was ordered to remove the accumulated methanol inside the body. The nephrologists completely resisted putting the patients on the dialysis machine without a report of methanol level despite the criticality of the clinical situation. After deeply scientific discussion that took a long time with more deterioration of the patients' condition the patients were put on the dialysis machine, but had a grave prognosis - from irreversible blindness to death.

The sixth fatal iatrogenic error was related to a 25-year-old pharmacist student with acute methanol poisoning from deliberate ingestion of 100 ml of methanol and delayed presentation with recurrent attacks of vomiting, abdominal coli, metabolic acidosis 7.21 and blurring of vision. Once the diagnosed was established as acute methanol intoxication, the dosage of ethanol was calculated, 10 cc/kg at10% concentration. The responsible treating physician calculated the dosage of ethanol in a concentration of 70% "available concentration as an antiseptic solution in hospital" instead of 10% which led to an immediate deterioration of the consciousness level of the patient with grave deterioration of the clinical condition, ending in death.

The seventh case was a51-year-old female with a history of a symptomatic snake bite, presenting with only generalized fatigue. The responsible treating physician observed the local and systemic signs of toxicity for four hours then asked the patient to return home and relax to relieve her fatigue. After a lapse of 22 hours from the first admission she was readmitted with severe descending neuroparalytic manifestations, "ptosis, dysarthia and difficulty in breathing". Five antivenin vials were administered in the intensive care unit and she completely recovered after five days from the second admission.

The eighth real clinical toxicology case scenario was a toxic snake bite in the tip of the index finger of the right hand. The

patient visited the hospital eight hours after the time of the bite, and the responsible treating physician meticulously followed the instructions on the antivenin vial "give one ampoule IM" without any further addition to the antivenin dosage. After a lapse of 18 hours with rapid deterioration of the clinical condition, the patient was transferred to the tertiary hospital with gangrenous changes in the index finger and oedema, redness, tenderness, and swelling in the right hand. The patient received 15 vials of antivenin in three separate infusion settings with a four-hour interval between each of them. However, the condition did not respond to antidote therapy, and there was more deterioration of the clinical condition with progressive disseminated intravascular coagulation and compartmental syndrome. The patient died 15 hours after admission.

In the ninth clinical toxicology situation, a 37-year-old female made a suicide attempt by ingestion of one tablet of Zinc Phosphide. She was immediately transferred to the primary care unit. The treating physician attempted to induce vomiting as a decontamination procedure, but he actually induced more intoxication in the patient by providing a high humidity environment in the gastric lumen that helped the release of phosphine gas and more deterioration of the clinical scenario, so the patient was immediately referred to the tertiary hospital with immediate repeated gastric suction and adjunctive treatment in the ICU until she completely recovered after three days.

Finally, a 46-year-old female patient with neuroleptic malignant syndrome from overdose of prescribed antipsychotic medication. As well as benzodiazepine, dantrolene and bromocritine, the treating physician prescribed amantadine, apomorphine, and dopamniergic agonist. The previous prescribed medication augmented the anticholinergic side effects that resulted finally in severe paralytic manifestations with marked electrolyte disturbances, and finally ended in a grave prognosis and death within two days.

DISCUSSION

The clinical toxicology field is a highly developed field of emergency medicine. From the variable clinical data we can identify the various methods of treatment of toxic patients and find the most effective form of management. In cases of alkaline caustic ingestion, Peclova and Naravatil (2005) conclude that the use of corticosteroids in the management of caustic corrosive ingestions should be abandoned as they do not prevent the development of strictures and may lead to the development of serious adverse effects. Same result were be completely clarified in case one studied in the current case series [1].

As regards acute organophosphate poisoning diagnosis, the main signs and symptoms are similar to the various clinical manifestations of cholinergic syndrome (SLUDGEMM; Salivation, Lacrimation, Urination, Defecation, Gastrointestinal upset, Emesis, Miosis and Muscle spasm) beside mandatory lowering on cholinesterase enzyme level [8]. So from the previously mentioned criteria for organophosphate poisoning clinical picture we cannot diagnosis toxicity from one clinical sign or symptom.

In cases of acute organophosphate toxicity, the main causes of deterioration in the clinical course of the studied cases were inappropriate atropine dosage and lack of oxime therapy, as atropine medication should be given in a careful protocol regimen to prevent patient stress, and acceleration of atropine dosage in a non-appropriate very short dose interval should be avoided. In accordance with this conclusion, Vucinić et al [9] conclude that continuous infusion with atropine did not accelerate the improvement of the clinical condition of organophosphate toxicity nor did it prevent the development of intermediate syndrome. Prolonged intensive care monitoring and respiratory care with steady supplementation of atropine are the key management approaches to dealing with organophosphate toxicity with its complications.

Regarding clinical presentation of carbon monoxide poisoning, the early presentation simulates flu-like manifestations or food poisoning but by taking a meticulous history, examination and basic routine investigation, the examiner can clarify strange, alarming symptoms and signs of normal food poisoning corresponding to the clinical pathway such as recurrent vomiting, marked disturbed consciousness level, significant metabolic acidosis and absence of intractable abdominal colic [10]. Nikkanen and Skolnik document the manifestations of acute carbon monoxide toxicity in vague circumstantial evidence as remarkable disturbed conscious level that gradually returns to normal on arrival at the emergency department.

There are various methods of methanol toxicity management via ethanol haemodialysis and fomizole. From deep clinical evaluation of the efficiency of the various methods of management, haemodialysis was found to be the most effective procedure to remove poisonous material from the intoxicated body. The dangers of ethanol usage especially in wrongly calculated dosages in the studied cases were particularly serious in regard to inhibition to the central nervous system that leads to more disturbed conscious level and respiratory failure. On the same side of the fined conclusion, Brent and Martin (1985) [11] documented seven patients with methanol poisoning who were treated with ethanol, haemodialysis and supportive measures. Their patients survived, but one had permanent visual impairment. A 10% ethanol solution administered intravenously is a safe and effective antidote for severe methanol poisoning. Ethanol therapy is recommended when plasma methanol concentrations are higher than 20 mg per dl, when ingested doses are greater than 30 ml, and when there is evidence of acidosis or visual abnormalities in cases of suspected methanol poisoning.

In cases of snake bite it is a critical to put a patient with suspected poisonous snake bite under observation for at least 12-24 hours for any developing symptoms or signs of toxic manifestations. Once any signs of toxicity appear locally or systemically, the antivenin therapy should be immediately started with a dosage of five vials via the intravenous infusion route. The same conclusion was supported by Monzavi et al. 2014. [12].

The most dangerous step in zinc phosphide pathogenesis is contact of toxic material with humidity, which leads to the release of phosphine gas and various toxic effects, so all decontamination procedures should be prohibited that bring water into contact with the toxic zinc phosphide substance. This is supported by Proudfoot (2009), who documented that there is no antidote

to phosphine or metal phosphide poisoning and many patients die despite intensive care. Supportive measures are all that can be offered and should be implemented as required with strict prohibition of increasing the release of phosphine gas by increasing contact with a humid environment [13].

Tonkonogy [14] states that the line of management for neuroleptic malignant syndrome were summarized in the care of airway and breathing, as follows: give intravenous benzodiazepines for agitation; avoid physical restraint as it can worsen the hyperthermia; discontinue the offending drug; give IV fluids for dehydration. If there has been a recent overdose of the agent then activated charcoal is indicated. Dopaminergic drugs, such as bromocriptine and amantadine, and muscle relaxants, such as dantrolene sodium, are frequently used in severe cases. Any other medications added to the previous management list may worsen the clinical condition by creating more disturbances in the neurotransmitter balance at the brain. The current case series reached the same conclusion as adding non-supportive medication to the treatment process of neuroleptic malignant syndrome is critical and may lead to a fatal outcome resulting from disturbances in the brain neurotransmitter.

CONCLUSION

The current study demonstrates that it is essential to strictly follow the line of management when treating toxic substance exposure, and any missing steps in diagnosis, treatment or follow-up, even if minor, may lead to a grave prognosis or to death.

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