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CASE REPORT

Teenage organophosphate insecticide poisoning: An ugly trend in Enugu, Nigeria

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ABSTRACT

Background: Organophosphate poisoning is still a major problem in developing countries owing to indiscriminate use of these compounds in many households. The risk of poisoning is worsened by uncontrolled sale of organophosphorus insecticides on the streets and in open markets. We report three cases of organophosphate compound poisoning among adolescents with suicidal intent.

Methods: We reviewed the hospital admission case records of three cases of organophosphate poisoning among adolescents managed at the children emergency room of University of Nigeria Teaching Hospital, Ituku Ozalla, Enugu, South-east Nigeria. Relevant information on the clinical characteristics of the patients, investigations and treatment, and outcome of treatment were obtained.

Results: The events of poisoning were preceded by strained family relationship in two of the cases while failure in a promotional examination preceded the incident in one of them. Atropine monotherapy in addition to airway management and oxygen support successfully reversed the symptoms and signs in 2 of the 3 cases. One died within 18 hours of admission from cardio-respiratory depression. Mean duration of admission in patients that survived was 48 hours.

Conclusion: This report highlights the ugly trend of suicidal ideation among adolescents and the challenges of management of organophosphate poisoning in our setting. It serves as a wake-up call to Nigerian parents and healthcare providers on the increased risk of indiscriminate use of organophosphorus compounds as insecticides in the homes.

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INTRODUCTION

Organophosphate compounds are a diverse group of chemicals used in both domestic and industrial settings, examples of which include insecticides, nerve gases and antihelminthics.[1] These chemicals kill insects and cause poisoning in animals by inhibiting the enzyme, acetylcholinesterase (AChE) which degrades acetylcholine in nerve synapses. Inhibition of AChE in the nerves results in a

buildup of acetylcholine (ACh) and overstimulation of ACh receptors.[2] Death from acute poisonings is frequently due to respiratory failure resulting from inhibition of central (medullary) respiratory drive, excessive bronchial secretions, and bronchospasms coupled with depolarizing blockade at neuromuscular junctions (diaphragm and inter-costals).[2] Over a hundred organophosphate compounds representing a variety of chemical, physical and biological properties are in commercial use.[3] Organophosphate insecticides are common household products in Nigeria, where they are used mainly to control mosquitoes and other household pests such as cockroaches and bed bugs. More than 80% of the reported toxic exposures to insecticides are attributable to organophosphates.[4]

The most popular source of organophosphate insecticide/pesticide in Nigeria is the locally made variety called '*Otapiapia*'.[5] The main active ingredient in '*otapiapia*' is dichlorvos or 2, 2-dichlorovinyl dimethyl phosphate (DDVP), with most containing between 5-10% w/v[6] and are readily available for purchase on the streets. '*Sniper*', which is another common variety of DDVP has a more refined packaging and is available in stores nationwide at a higher cost. Organophosphate poisoning may occur through ingestion, inhalation or penetration through the intact skin; and may be suicidal, accidental or homicidal. Most of the cases are accidental.[7, 8] The World Health Organization (WHO) noted that pesticide poisoning is now the most common method of suicide worldwide.[9, 10] About two million people attempt suicide and one million accidental poisoning cases occur each year worldwide.[4] Suicide accounts for an estimated 849,000 deaths worldwide,[9] and ~ 300,000 deaths in the developing world every year.[11]

In Nigeria, suicide is traditionally a taboo. However, the recent report of cases of intentional poisoning is probably as a result of media influence or breakdown of our traditional social fabric. A ten-year retrospective study of childhood poisoning in Warri, South-south Nigeria, noted that intentional poisoning occurred in the adolescent age group and mainly involved female teenagers.[8] Shwe et al[12] observed that organophosphates were the sole agent for intentional poisoning in the retrospective review of cases seen in North-central Nigeria and these were adolescent females. We report three cases of adolescent organophosphates poisoning with suicidal intent in our setting and also the challenges of managing such cases in resource poor settings.

CASE REPORT

Case 1: UA was a 15 years old female who first presented at Enugu State University Teaching Hospital, (ESUTH) with history of abdominal pain, salivation, loss of consciousness and urinary incontinence about 30 minutes after ingestion of an organophosphate insecticide/pesticide, "*sniper*". Ingestion followed failure in her college promotional examination. Candidates that fail this promotional examination are usually demoted to a lower class. Her academic performance was said to be average in her previous levels. She was the first child in a monogamous family with 4 children and there was no history suggestive of a strained relationship with her family or peers. At presentation, she was unconscious with Glasgow Coma Score (GCS) of 3/15, drooling saliva, in respiratory distress, tachypneoic and febrile (37.8^{0}C). Both pupils were pin point and there were widespread crepitations on all lung zones. Pulse was 120 beats/minute and blood pressure was 100/60 mmHg. Oxygen saturation (SpO_2) by pulse oximetry was 64% on room air and Random blood Glucose (RBG) was 6.3mmol/litre.

The airway was cleared and intranasal oxygen commenced. Intravenous atropine 2.5mg was administered every 15 minutes and pupils became normal sized after the third dose. The level of consciousness improved and she was transferred to the Intensive Care Unit (ICU) of the University of Nigeria Teaching Hospital (UNTH), Ituku/Ozalla, Enugu, which was a distance of about 25km, for respiratory support. On arrival at the ICU, GCS was 10/15 (E-3, M-6, V-1), pupils were constricted (pin point) and she was in severe respiratory distress; SpO_2 was 71% on room air and respiratory rate was 20 breaths/minute. There were widespread coarse crepitations, prolonged capillary refill time, cold extremities, small volume pulse with a rate of 80 beats/minute, and a blood pressure of 90/40 mmHg; RBG was 3.3mmol/litre. She was restarted on hourly doses of atropine. Pralidoxime was not administered due to unavailability of the drug. Intravenous boluses of normal saline were given. Subsequently, intravenous dopamine and epinephrine were commenced to maintain systemic circulation. She also received 5% dextrose saline to maintain euglycaemia. She regained full consciousness about 12 hours later. Blood pressure normalized and SaO_2 remained between 96% and 99% on 100% oxygen; she was then extubated after 12 hours on mechanical ventilator. Following extubation, her condition deteriorated rapidly with loss of consciousness and rapid drop in oxygen saturation. Attempt at resuscitation failed.

Case 2: KA was a 15 years old adolescent female who presented to UNTH with history of excessive vomiting and increased sweating following ingestion of '*otapiapia*'- a locally made organophosphate14 hours prior to presentation. Patient's mother was said to have unjustly accused her of having unhealthy relationships with some adolescent males within their neighbourhood. Following the rancour, she locked herself in a room and attempted suicide by ingesting the '*otapiapia*'. Estimated volume of the ingested substance was about 10mls. A few minutes later, she started frothing from the mouth, necessitating her calling for help. An attempt to reverse the effects of the poison with some local remedies such as palm oil and coconut water believed to be antidote to poisons for a period of 2 hours yielded no results. She was subsequently taken to a nearby hospital (ESUTH). She vomited twice on the way to the health facility. While on admission at the hospital she also developed fever, cough, fast and difficult breathing, and chest pain. She received intravenous fluids and intramuscular injections and was subsequently referred to UNTH for further evaluation and treatment.

Patient is the first child of her family and a senior secondary 2 student in a government school, whose academic performance has been good; she belonged to the low socioeconomic class. There is no antecedent history suggestive of psychosocial instability prior to this incident.

Salient findings on presentation were a conscious adolescent female with pyrexia, mild pallor, respiratory distress, and bi-basal coarse crepitations. Initial vital signs were as follows: temperature of 38^{o}C, respiratory rate of 44 breaths per minute, oxygen saturation on room air (SpO_2) of 95%, capillary refill time was < 2 seconds, pulse rate was 120 beats/minute and blood pressure of 120/70 mmHg. Random blood glucose was 5.8mmol/L and Haematcrit 29%. Dipstick urinalysis showed: specific gravity 1.030, blood: 3+, leukocyte: 2+, protein: +, ketone: +. Serum electrolyte, urea, creatinine was essentially normal. She was commenced on intranasal oxygen, intravenous infusion, stat dose of intravenous atropine 2.5mg and intravenous ceftriaxone 2g daily. Urine output

and vital signs remained normal with close monitoring. Subsequently, oxygen saturation fluctuated between 82% and 92% on intranasal oxygen but improved to between 95% and 99% with periodic airway suction. She stabilized within 72 hours with complete resolution of symptoms and signs. She was counselled and discharged home to continue follow up at the adolescent and psychological medicine clinics.

Case 3: EK was a 13 year old male who presented to our facility with a history of vomiting, and excessive sweating following ingestion of about 3mls of organophosphate insecticide/pesticide (*otapiapia)* an hour prior to presentation. There was no lacrimation or wheezing.
He was accused of stealing his mother's cell phone on instruction by his peers. On account of this act, he had a dispute with his parents and he then ingested the organophosphate compound with suicidal intent. Prior to presentation, he had received about 20ml of palm oil as home-made remedy. Patient is the last child in a monogamous family with 5 children and a junior secondary 2 student with good academic performance; he belonged to the upper socioeconomic class. There was no antecedent behavioural abnormality prior to this incidence.

Findings on presentation were an anxious adolescent male, diaphoretic, with strong stench of organophosphate perceived from his body. Pupillary examination showed bilateral miosis. Cardiopulmonary examination and vital signs showed tachypnoea but no dyspnoea. Respiratory rate was 40 breaths/minute; pulse rate - 82 beats/min., blood pressure - 110/ 60 mmHg and temperature - 36^{o}C. There was no adventitia on the lung fields. The general and neurological examinations were normal. No other significant findings. Laboratory investigation including a complete blood count, urea and electrolytes were normal. Lactic acid levels and arterial blood gasses were not checked. Plasma pseudocholinesterase and red cell cholinesterase levels could not be assayed.

Emergency resuscitative measures included gastric lavage with normal saline; maintenance intravenous 5% dextrose saline infusion. Urine output remained essentially normal. He also received intravenous atropine at 0.05mg/kg/dose, ~ 2mg stat, repeated after 15 minutes. On subsequent review an hour later, symptoms had reversed. Vomiting had subsided, no diaphoresis; pupils were normal size reacting normally to light. Twenty four hours into admission, patient was alert; vital signs had normalized (temperature 36^{o}C, respiratory rate 20 breaths/minute, pulse rate 80 beats/ minute). No abnormal findings on systemic examination. He was discharged after 24 hours of admission. He was counselled and subsequently referred to adolescent and psychological medicine clinics for review.

DISCUSSION

Organophosphate (OP) poisoning is not uncommon, and may result in death because of its high toxicity.[13] Adolescents usually attempt suicide following emotional trauma from family stress or failed relationships with the opposite sex. Organophosphate insecticide/pesticide seems to be the commonest agent because it is readily available and easily accessible to users despite its marked toxicity. The reported cases buttress this fact. Some of the documented factors contributing to fatal poisonings are availability, low price, inappropriate use and storage of pesticides; unsafe disposal or reuse of pesticide containers as well as lack of adequate regulations controlling their sale.[14, 15] The ease of access to these hazardous products resulted in serious health threats as observed in these patients. Nigeria has no strict

regulations/guidelines on importation and sales of these hazardous products to the general public. Similarly, many African countries do not have well established poison control centres.[16] *'Sniper'*, one of the poisoning agents in this series, belonging to the dichlorvos or 2,2-dichlorovinyl dimethyl phosphate compound (DDVP) chemical family is very popular in Nigeria and readily available in Nigerian markets and shops. It is meant for agricultural and industrial use and never for indoor use. However, many Nigerian families use it as insecticide/ rodenticide in their homes.

Organophosphates is the sole agent for intentional poisoning in adolescents[8, 12] especially in females. Intentional poisoning in adolescents are precipitated by psychosocial factors such as family stress, trauma from a failed relationship[8] and sometimes failure in examinations as was obtainable in one of the cases highlighted. The events of poisoning were preceded by strained family relationship in two of the cases while failure in a promotional examination preceded the incident in one of them. All organophosphate preparations are absorbed through the bronchi, intact skin, mucous membrane and conjunctiva as well as through the gastrointestinal tract.[17-20] These patients were exposed to the toxic agents through the gastrointestinal route. Toxic effects of the different compounds vary considerably depending on age; route and duration of exposure; and inherent characteristics (lipophilicity) of the specified chemical justifying the identification of the agent involved as soon as possible.

Organophosphates inhibit the cholinesterase activity thereby prolonging and intensifying the effects of acetylcholine.[17-20] Acetylcholine (ACH), a neurotransmitter binds with nicotinic and muscarinic receptors to trigger off action potential. It is inactivated by Cholinesterase via hydrolysis. Organophosphates bind to and inactivate Cholinesterases (acetyl cholinesterase), and allows ACH to accumulate at the synapse leading to overstimulation. Cholinesterases are found in the red blood cell (true cholinesterase) and plasma (pseudo cholinesterase). Symptoms of exposure which typically appear within 30 to 90 minutes after exposures[18] are due to the continuous stimulation of the muscarinic and nicotinic receptors. Muscarinic symptoms include excessive secretion from mucus membranes, increased pulmonary and oropharnygeal secretions, pupillary constriction manifesting as diarrhoea, excessive urination, miosis, bronchorrhea, bradycardia, emesis, lacrimation and salivation,[13, 17, 18] commonly tagged with the mnemonic 'DUMBBELS'. Adrenal medulla activity manifest as increased sweating and garlic smell because of vicarious excretion, bradycardia, abdominal cramping and intestinal hypermotility. Nicotinic activity results in autonomic nervous system stimulation manifesting as tachycardia, hypertension, sweating and rarely dilated pupil. Nicotinic overstimulation at the neuromuscular junction causes muscle weakness, fasciculation, fatigue and paralysis.[13, 17, 18] With irreversible binding of organophosphate compound and cholinesterase (aging), symptoms are continuously manifested. These manifestations are graded into mild, moderate and severe based on the World Health Organization classification of severity of organophosphate poisoning.[21] Based on this classification, two of the cases manifested features of moderate OP poisoning while the third had severe OP.

Diagnosis of OP poisoning is made both clinically and with biochemical indicators. History of exposure to a known OP is important. Consistent with other reports[19, 21], the most common clinical findings include

increased sweating, vomiting, respiratory distress, tachycardia, and miosis. Miosis is considered to be a very strong indicator of organophosphate poisoning.[22] Red cell cholinesterase level is thought to be a good marker of synaptic function and atropine needs; and by extrapolation a good marker for severity.[23] Clinical features of OP poisoning appear when RBC cholinesterase activity is <75% of normal and in clinically overt poisoning it is <10%.[22] Chest X-ray may show evidence of pulmonary edema; and electrocardiography may show evidence of cardiac arrhythmia, QT prolongation, ST and T wave abnormalities in patients with abnormal heart rate and persistent hypotension. Leucocytosis with or without neutrophilia may be present. Brain computerized tomography scan may be required in cases with altered mental status. Diagnoses in these patients were promptly made based on clinical findings and a few laboratory findings, but confirmation by estimation of red cell or plasma pseudocholinesterase activity was not done due to lack of laboratory facilities.

Following the key principles of management of OP poisoning[18, 24] airway/ventilation support were provided for these patients. Intermittent suctioning of oral secretions and oxygen therapy formed part of the initial management in our patients. Mechanical ventilator support was provided in the intensive care for the patient with cardiopulmonary compromise. The duration for which organophosphates remain in the stomach after ingestion is still unknown. However, it is known that organophosphates are easily absorbed through mucus membranes within minutes of ingestion and gastric lavage is likely to play a role at the early stage of poisoning.[24, 25] Guidelines for treatment of drug self-poisoning suggests that lavage should be considered only if the patient arrives within 1 hour of poison ingestion but there is no evidence that any form of gastric decontamination is of benefit to patients poisoned with organophosphorus.[26] Although gastric decontamination (irrigation with normal saline through a naso-gastric tube) was done in our patients. Activated charcoal has not demonstrated sufficient evidence that it reduces morbidity and mortality in poisoned patients.[27] Furthermore, late presentation makes any benefit doubtful. In our environment, victims of poisoning usually present late owing to the delays encountered while parents and sympathizers applying various 'first aid treatment'[28] (which are of no benefit) ranging from induction of emesis to use of palm oil, coconut water and so on, aimed at diluting the poisoning agent. Some of these were observed in the index cases.

Atropine seems to be the mainstay of management of OP as it reverses the muscarinic effects.[11, 22, 24, 29] Atropine is used for 24 hours while OP is being degraded. Using the intermittent bolus regimen[30], it is recommended that large doses of atropine be given at a dose of 0.03-0.05mg/kg. This should be given intravenously every 10-20 minutes or 'pro re nata', then every 1-4 hourly for at least 24 hours till signs of atropinisation (warm dry flushed skin, dilated pupil, increased HR>80b/min, systolic BP >80mmHg, and rapid reversal of bronchospasm and bronchorrhoea.[24, 31]) appear. Patient UA, with severe symptoms had delay in time to atropinisation and deteriorated rapidly while the other two had remarkable response and were subsequently discharged following full recovery. It has been documented that delay in atropinisation can result in death from central respiratory depression, hypoxia (due to bronchospasm and bronchorrhoea) and hypotension (due to bradycardia and myocardial depression).[32] Persisting

circulatory insufficiency may call for treatment with catecholamines to increase and maintain blood pressure as well as heart rate.[33] Atropine has no effect on nicotinic symptoms. In some cases, oximes like pralidoxime (2-PAM), obidoxime, or asoximeis started as soon as the diagnosis is confirmed may help optimize outcome.[23] Oximes are cholinesterase re-activators used as adjuncts to atropine in the treatment of organophosphate poisoning. They act primarily by reactivating the cholinesterase enzyme to restore the enzymatic destruction of acetylcholine at the neuromuscular junction and relieve muscle paralysis including respiratory muscles as well as reverse the nicotinic effect of the poison.[24, 34] The benefits of oximes can be limited by high concentration of organophosphate in the blood, usually after ingestion of a large dose. Pralidoxime, unlike atropine does not cross the blood brain barrier and becomes useless when aging has already occurred.[34] They are also ineffective if the patient presents after an hour of ingestion or develops severe complications like aspiration pneumonia or hypoxic brain injury before treatment.[22, 24] Unfortunately, dimethyl organophosphates such as 2,2-dichlorovinyl dimethyl phosphate compound, which is the active content in '*sniper*' do not readily respond to the oximes unlike diethyl formulations-parathion and diazinon.[35-38] This is due to the fact that diethyl compounds reactivate and 'age' at a significantly slower rate than dimethyl compounds.[36] The harmful effect of pralidoxime is another source of concern[34, 36, 37, 38]

Early enteral feeding is recommended in stable patients as it is said to reduce entero-hepatic circulation and further absorption of the poison. Two of the patients were commenced on oral feeds as soon as they were stabilized. The third remained unstable on mechanical ventilation thus calories were maintained by intravenous infusion. Overall mortality in organophosphate poisoning is about 50% for those with severe toxicity.[20] Mortality is mainly from respiratory failure.[20, 39] The cause of death in our patient was attributable to severe poisoning, and early withdrawal of ventilator support resulting in respiratory failure and death. Half of those with intentional poisoning in the study by Ugwu and colleagues[8] died largely as a result of late presentation while 8 out of 117 patients seen by Syed et al[38] with OP poisoning were dead on arrival. Mechanical ventilation is required especially in patients with severe poisoning at risk of death from respiratory failure. Syed et al[38] noted that mortality among those that were on mechanical ventilation for 2-7 days was 7.2%, mechanical ventilation for more than seven days resulted in 33.3% mortality while mechanical ventilation for < 2 days resulted in 100% mortality.

Conclusion

These cases highlight the tendency for poisoning with suicidal intent among adolescents in the study area environment and the need for anticipation of potential life threatening complications of organophosphate poisoning during treatment. Parents should be aware of the high risk of psychological imbalance among adolescents and endeavour to develop and maintain intimate relationship with their children in order to detect the early warning signs of emotional distrust in them. Increased awareness of this ugly trend will lead to early recognition of warning signs and thus prevent unnecessary loss of life. Parents and caregivers should learn to store these poisonous agents in locked, hard to access cabinets. There is an urgent need to establish poison control and information centres in each of the six geopolitical regions of Nigeria, and a national centre in Abuja, the Federal Capital Territory. Each centre should be

tagged with easy-to-recall telephone numbers for prompt referral.

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