An Unusual Case of Triazophos Poisoning Presenting with New-Onset Refractory Status Epilepticus

Sir,

A 45-year-old male presented to us with an alleged history of 500 ml of triazophos consumption since 2 h. In Emergency Department when the patient was attended he was having generalized tonic clonic convulsions.

The patient was immediately given thiamine 100 mg intravenous (IV) stat, 100 ml 25% dextrose, and 10 mg diazepam IV slowly. For a brief period of time convulsions stopped but the patient was not responding to verbal commands nor deep painful stimulus (GCS-E1, M1, V1). Vital signs revealed pulse = 120 beats/min regular, blood pressure = 160/90 mmHg, breathing was laborious = 35/min, and pupils were 1.5 mm reactive to light. Arterial blood gas revealed arterial hypoxemia. Airway was protected with endotracheal intubation and suctioning was done, gastric lavage was given was done which was smelling of organophosphorus (OP) compound and patient was immediately shifted to Intensive Care Unit and was put on mechanical ventilation and was ventilated with 100% oxygen. Injection atropine 3 mg was given IV stat and repeated every 10 min until the patient was fully atropinised. An atropine infusion was started at a rate of 2 ml/h. Injection pralidoxime chloride 2 g IV over 20–30 min was given and an infusion of pralidoxime 0·5 g/h in 0·9% normal saline was initiated and continued for 48 h. Serum cholinesterase was 2749 IU (normal range 4000–11,000 IU).

After 20 min patient again developed GCTS. Injection lorazepm 5 mg IV stat was given over 1–2 min; there was no response, so a repeat dose after 5 min was given but seizures continued, then injection phenytoin 50 mg/kg at a rate of 50 mg/min was given but seizures continued. Then a standard protocol of status epilepticus (SE) with loading doses of sodium valproate 25 mg/kg was given with no response. Then phenobarbitone 20 mg/kg IV was given, but seizures continued and lastly after 3 h seizures were controlled with propofol infusion 300 mg/h and gradually propofol infusion was tapered off in next 24 h and patient remained seizure free with IV phenytoin 100 mg 8 hourly. A computed tomography (CT) scan of the brain was normal. Until that time patients pulse rate was above 100 and pupils were normal sized and reactive to light. On the second day, that is, 24 h after admission patient developed bradycardia of 60 beats/min and bronchorrhoea. Examination at that moment revealed pinpoint pupils, endotracheal secretions had increased. Injection atropine bolus 3 mg every 20 min was given until pupils were dilated and tachycardia was achieved and atropine infusion was increased to 5 ml/h. Over next 2 days the patient was stable and gradually atropine was tapered off.

All other investigations, that is, complete blood count, kidney and liver function tests, metabolic profile, chest X-ray, and CT brain was normal. On fourth day, patient was extubated; by seventh day, patient recovered fully and a repeat serum cholinesterase was 6400 IU (4000–11,000 IU normal range); and by tenth day, patient was discharged.

Hattrick[1] (triazophos-O, O-diethyl, O 1 phenyl1h 1, 2, 4-triazol 3yl – phosphorothio) is a broad spectrum systemic insecticide and acaricide which is classified under OP compounds . Symptoms and signs of poisoning in humans are similar to OP compounds poisoning.[2-6] This pesticide is extensively used in agricultural practice throughout the world.

The basic effects of OP compounds poisoning are given in Table 1.

New-onset refractory SE (NORSE) is defined as refractory SE which usually lasts for 24–48 h without an obvious cause after initial investigations ruling out strokes, brain masses, drug overdoses, and encephalitis. Refractory SE is a condition in which patients suddenly experience continuous seizures or repeated attacks of seizures that do not respond to standard anticonvulsant medications. NORSE carries a high rate of complications and mortality, but a significant proportion of patients do eventually recover.

Table 1: Clinical signs and symptoms of OP compound poisoning

Muscarinic effects	Nicotinic effects	Central nervous system effects
Cardiovascular - bradycardia, hypotension Respiratory - rhinorrhea, bronchorrhea, bronchospasm, cough, ARDS Gastrointestinal - hypersalivation, vomiting, abdominal pain, diarrhea Genitourinary - urinary incontinence Ocular - blurred vision, miosis Glands - increased lacrimation, diaphoresis	Muscle fasciculations, cramping, weakness, diaphragmatic failure Autonomic nicotinic effects: hypertension, tachycardia, mydriasis, and pallor	Anxiety Emotional lability Restlessness Confusion Ataxia Tremors Seizures Coma

OP = Organophosphorus, ARDS = Acute respiratory distress syndrome

Our case was interesting because the patient first presented with predominant central nervous system effect in form of NORSE first time in life, which ultimately responded to general anesthesia and later on the management of OP was carried out after muscarinic effects of OP poisoning manifested a little later after 24 h. So it is imperative to keep this in mind while treating such cases. The average duration of NORSE as reported by various reports is believed to be 36 days (6–68 days).[7] In this case it lasted for 5 h.

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Conflicts of interest

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