CPD article

Tremorgenic mycotoxicosis in dogs

Dogs commonly ingest tremorgenic mycotoxins because of their indiscriminate eating habits. Common sources are mouldy food, compost and rotten, fallen fruits and nuts. The mycotoxins penitrem A and roquefortine are produced by species of the mould *Penicillium*. Tremorgenic mycotoxicosis is characterised by rapid onset whole-body muscle tremors, vomiting, pyrexia, convulsions, ataxia, twitching and hyperaesthesia. Dogs are also at risk of aspiration. Treatment is aimed at decontamination and control of increased muscle activity and seizures to prevent complications from prolonged seizure activity. This will involve gut decontamination (depending on the condition of the dog), repeat doses of activated charcoal (as the mycotoxins undergo enterohepatic recirculation) and administration of sedatives, anticonvulsants and/or anaesthetics. Lipid emulsion should also be considered in severe cases. Prognosis is typically good in dogs with mild signs or controlled seizures, but more guarded where there is uncontrolled seizure activity or aspiration pneumonitis.

https://doi.org/10.12968/coan.2021.0049

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Key words: | mouldy food | mycotoxins | penitrem A | roquefortine | toxicology

Mycotoxins are fungal metabolites that cause toxicity when ingested by humans and animals. Tremorgenic mycotoxins are commonly produced by the fungus *Penicillium*, which is found in mouldy (decomposing) foods, silage and compost. Most cases of tremorgenic mycotoxicosis involve dogs, as cats have more discriminating eating habits (Warantuke, 2017).

Sources

Mouldy foods, particularly mouldy dairy products (*Figure 1*), are common sources of tremorgenic mycotoxins (*Box 1*), but any food waste, including composted waste (*Figure 2*), rotting, fallen fruits and nuts and silage are potential sources.

The tremorgenic mycotoxins

There are a number of tremorgenic mycotoxins, but only a few are of clinical significance. Penitrem A and roquefortine are the most common mycotoxins associated with poisoning in small animals. Penitrem *A* (previously known as tremortin A) is primarily produced by the mould *Penicillium crustosum* (Frisvad and Filtenborg, 1989). It has been isolated from mouldy cheese, bread and walnuts. Roquefortine is mainly produced by the mould *Penicillium roqueforti* but also by other *Penicillium* species (including *P. crustosum*; Frisvad and Filtenborg, 1989). Roquefortine is found in blue cheese and decaying organic matter such as silage, rubbish and compost (and is produced by different strains of *P. roqueforti*) (Frisvad and Filtenborg, 1989).

Mechanism of action

The mechanism of action of these compounds is unclear and may vary with the mycotoxin, and they may also act synergistically. Tremorgenic mycotoxins interfere with inhibitory neuroreceptors and enhance excitatory amino acid neurotransmitter release mechanisms (Norris et al, 1980; Bradford et al, 1990). Both penitrem A and roquefortine are lipophilic, which allows their movement across the blood–brain barrier. Penitrem A is the most studied and has been shown to act on the central nervous system to induce seizures in experimental animals (Sobotka et al, 1978; Arp and Richard, 1981). The main neurotoxic effects of penitrem A appear to be caused by blocking of the high-conductance calcium-activated potassium (BK) channels (Knaus et al, 1994) and impairing GABAergic neurotransmission (Moldes-Anaya et al, 2011). It has also been shown to induce tremors in mice by acting as an antagonist to production of the neurotransmitter glycine (Catovic et al, 1975).

Effects on the peripheral nervous system are also thought to be a result of penitrem A inhibiting BK channels in muscle cells (Eriksen et al, 2013). This may cause an increase in action potentials at nerve terminals by preventing repolarisation and producing an increase in neurotransmitter release. Penitrem A also affects neuromuscular junctions, possibly by increasing pre-synaptic neurotransmitter (acetylcholine) release and/or by increasing the sensitivity of post-synaptic acetylcholine receptors (Eriksen et al, 2013). Penitrem A has also been shown to cause neuronal death in experimental animals, particularly of Purkinje

Figure 1. Mouldy food such as bread and particularly dairy products, such as cream cheese, are common and readily accessible sources of tremorgenic mycotoxins in dogs.

Figure 2. Composted and rotting food waste is also a source of tremorgenic mycotoxins.

Box 1. Potential sources of tremorgenic mycotoxins

- Food waste and rubbish (Lowes et al, 1992; Walter, 2002)
- Mouldy bread (Hocking et al, 1988)
- Mouldy rice (Naude et al, 2002)
- Mouldy dog food (Chapman, 2018)
- Dairy products
  - Mouldy cream cheese (Arp and Richard, 1979; Young et al, 2003)
  - Mouldy macaroni cheese (Young et al, 2003)
  - Mouldy blue cheese (Puls and Ladyman, 1988) and a 'huge amount' of blue cheese (Anich, 1990)
- Mouldy fallen fruit and nuts
  - Walnuts (Richard et al, 1981; Munday et al, 2008)
  - Apples (Eriksen et al, 2010)
  - Compost (Boysen et al, 2002)

cells located in the cerebellum and involved in co-ordination and movement (Cavanagh et al, 1998).

Toxicokinetics

There is limited information on the toxicokinetics of these tremorgenic compounds. Penitrem A is rapidly absorbed, as demonstrated by the rapid onset of signs in observed clinical cases and experimental studies (Hayes et al, 1976; Moldes-Anaya et al, 2012). In one study, a dog that ingested an estimated 0.4 mg of penitrem A per kg/body weight developed signs within 30 minutes of ingestion (Eriksen et al, 2010).

In a study in mice, penitrem A was distributed to the liver, kidneys and brain, with peak concentration occurring 1 hour after oral dosing and the highest concentrations being observed in the liver (Moldes-Anaya et al, 2012). Penitrem A is extensively metabolised in the liver and in a study in mice, no metabolites were detected in the brain, showing that the hydrophilic metabolites do not cross the blood–brain barrier (Moldes-Anaya et al, 2009). An in-vitro study of penitrem A in canine liver microsomes identified 11 phase I metabolites, including several isomers of mono- and di-oxygenated and hydrated products. These compounds were confirmed in the plasma of dogs with tremorgenic mycotoxicosis (Uhlig et al, 2020). In-vitro to in-vivo extrapolation predicted a high oral bioavailability and a short elimination half-life of penitrem A (Uhlig et al, 2020).

There is limited information on elimination of these compounds, but it is thought the main elimination route is via the bile and feces. Studies in rats and sheep have demonstrated that roquefortine (Laws and Mantle, 1987a) and penitrem A (Laws and Mantle, 1987b) are eliminated via the bile and undergo enterohepatic recirculation (Puschner, 2004).

Clinical effects

Onset

Signs of tremorgenic mycotoxicosis can occur rapidly. They may start within 30 minutes, but can be delayed up to 3 hours (Puschner, 2004; Eriksen et al, 2010). In 128 cases reported to the UK Veterinary Poisons Information Service (VPIS), the mean onset was 2 hours (VPIS case data).

Clinical signs

The most common signs in dogs with tremorgenic mycotoxicosis are whole-body muscle tremors, vomiting, pyrexia, convulsions, ataxia, twitching and hyperaesthesia (VPIS case data). There may also be hypersalivation, rigidity with hyperextension of extremities, hyperactivity, tachycardia, panting, tachypnoea, diarrhoea and nystagmus. Dilated pupils have been reported in some cases.

In severe cases, severe tremors and opisthotonos (*Case report 1*), status epilepticus and coma with paddling can occur. Aspiration is also a risk. Increased muscle activity can lead to exhaustion and dehydration, and complications of prolonged seizure activity include severe pyrexia and rhabdomyolysis, which can lead to acute kidney injury and disseminated intravascular coagulation. These complications can lead to multi-organ failure.

Laboratory findings

There may be hypoglycaemia, raised lactate dehydrogenase, creatine kinase and liver enzymes as a result of mycotoxin-induced increased muscle activity.

Duration

Most dogs with tremorgenic mycotoxicosis recover within 24–48 hours with aggressive treatment (Puschner, 2002), particularly if treatment is started soon after ingestion. In 187 cases reported to the VPIS, the mean recovery time was 40 hours (VPIS case data). However, in some cases, recovery may take 3–4 days (Walter, 2002; Eriksen et al, 2010). Most dogs recover fully (Naude et al, 2002; Young et al, 2003), but neurological sequelae have been reported in a small number of canine cases. In one dog, neurological signs (ataxia and tremor) took up to 6 months to resolve, and in two cases, ataxia was still present more than 3 years after the poisoning (Eriksen et al, 2010).

Diagnosis

Diagnosis of tremorgenic mycotoxicosis in dogs is usually based on the history of sudden onset tremors and convulsions in animals that have eaten mouldy food (often from the dustbin) or compost. Poisoning is often associated with mouldy dairy products, such as cream cheese. If a dog presents with a history of sudden onset tremors or seizures, particularly if they have a history of scavenging or bin-raiding, the owners should be questioned about possible access to mouldy material such as food, compost and fallen fruits and nuts.

The presence of roquefortine and penitrem A in suspect material or stomach contents can be confirmed by laboratory analysis. These compounds have also been detected in the organs of poisoned dogs (Eriksen et al, 2010) and roquefortine and penitrem A have been confirmed in samples from dogs with suspected strychnine poisoning where analysis for strychnine was negative (Lowes et al, 1992; Braselton and Rumler, 1996). Laboratory confirmation is not obtained in most cases. Other potential causes of tremors in dogs are listed in *Box 2*.

Prognosis

The prognosis for tremorgenic mycotoxicosis in dogs that have mild signs or controlled seizures is good. However, prognosis is more guarded in dogs with uncontrolled seizure activity or aspiration pneumonitis.

Death may occur from ingestion of mouldy material (Puls and Ladyman, 1988; Lowes et al, 1992). The fatality rate in 317 cases reported to the VPIS was 6.6% (5% euthanised, 1.6% died) (VPIS case data).

Box 2. Differential diagnosis for tremors in dogs

Tremors from tremorgenic mycotoxicosis are continutous and not intermittent, as with some other disorders (Barker et al, 2013).

- Poisoning:
  - Alphachloralose
  - Metaldehyde
  - Macadamia nuts
  - Xylitol (hypoglycaemia-induced tremors)
  - Organophosphorus and carbamate insecticides
  - Strychnine
  - Ivermectin
  - Lead
  - Methylated xanthines (caffeine, chocolate, theophylline)
  - Pyrethrins and pyrethroids
  - Bromethalin (not approved in the UK or Europe)
  - Cerebellar disorders
- Eclampsia (in pregnancy or lactation)
- Idiopathic tremor syndrome
- Hypomyelination
- Dysmyelination
- Metabolic disorders (such as hypoglycaemia or hypocalcaemia)
- Infection

Treatment

The mainstay of treatment in dogs with tremorgenic mycotoxicosis is prompt and safe decontamination, control of increased muscle activity and seizures to prevent complications from prolonged seizure activity and prevention of aspiration.

A treatment dose has not been established because the presence or concentration of tremorgenic mycotoxins in mouldy food cannot be determined without laboratory analysis and a toxic dose in dogs and cats has not been determined (Barker et al, 2013).

Gut decontamination

An emetic could be considered if ingestion was recent (within 1 hour) but only if the dog is asymptomatic, as there is a risk of aspiration if the animal has seizures while vomiting. Signs are usually rapid in onset, therefore, a gastric lavage under general anaesthesia could be considered, particularly if the quantity of mouldy material ingested is large or the animal is symptomatic. Repeat doses of activated charcoal (1–3 g/kg every 4 hours) should be given, if practical, as the mycotoxins undergo enterohepatic recirculation. Following gastric lavage, the first dose of activated charcoal can be left in the stomach. An anti-emetic should be given to reduce the risk of vomiting and aspiration pneumonia.

Case report 1. Mouldy cream cheese

A 3-month-old Australian Shepherd dog was admitted with severe muscle tremors, polypnoea, hyperkinesia, ataxia, intermittent opisthotonos and generalised seizures. The dog was sedated with phenobarbital and given intravenous fluids. On the same day, a 1-year-old Irish Setter from the same neighbourhood was admitted with muscle tremors and clonic seizures. It also received phenobarbital and intravenous fluids. Both dogs had improved by 12 hours with slight inco-ordination and moist pulmonary rales. The owner of the first dog reported finding a partly eaten package of mouldy cream cheese that he had thrown out the previous day (Arp and Richard, 1979). In an experimental study, three mice were given a saline suspension of toxic cream cheese; all developed tremors in 15 minutes and two died within 2.5 hours. They had received 0.6 ml of an emulsion of 2 ml of saline and 0.5 g of the mouldy cream cheese. The third mouse survived acute neurological signs and had a mild tremor by 22 hours. A 10 kg Beagle was given canned dog food mixed with 30 g of the mouldy cream cheese and by 2 hours had panting, generalised muscle tremor and limb rigidity. The severity of signs had reduced after 3 hours and the dog had only mild ataxia after 6 hours. Penitrem A and P. crustosum were isolated from the cream cheese (Arp and Richard, 1979; Richard and Arp, 1979).

Case report 2. Compost

Four dogs from the same household became unwell after ingestion of compost. The first dog (11 kg Shih Tzu) vomited 45 minutes later and within 2 hours, all the dogs developed ataxia and tremors, which progressed to seizure-like activity. The first dog was initially unresponsive to diazepam and required pentobarbital. It was also hyperpyrexic and required cooling. Once the temperature had been reduced and the tremors controlled, the animal was maintained on diazepam and had begun to improve by 12 hours after admission. It was discharged at 48 hours. The second dog (9 kg Shih Tzu) had similar features and was also treated with diazepam, pentobarbital and methocarbamol. It developed hypoventilation and aspirated, so ventilation was continued until 15 hours after admission. He recovered and was discharged at 48 hours with antibiotics for pneumonia. The third dog (8.5 kg Shih Tzu) had similar effects and was treated with diazepam, pentobarbital and methocarbamol. It also developed hypoventilation and was given flumazenil to reverse the diazepam. The dog was ventilated for 12 hours and discharged after 96 hours. The fourth dog, the largest (a 45 kg Great Pyrenees dog) was the last one to develop effects. By 80 minutes, it had a stiff gait and arching of the back. On admission, it had severe generalised tremors and pyrexia. It was given diazepam, pentobarbital and methocarbamol. It was well by 48 hours after admission and discharged the next day. Analysis of the gastric contents of the second dog was positive for penitrem A and roquefortine C (Boysen et al, 2002).

Supportive care

Respiratory rate, neurological status and temperature should be monitored. Dogs may initially be pyrexic, but cooling measures in combination with high dose sedation may precipitate hypothermia. Comatose animals should be turned regularly and positioned to reduce the risk of aspiration. Ventilatory support may be required in dogs with severe respiratory depression.

Intravenous fluids will correct electrolytes imbalance from vomiting and diarrhoea, help reduce body temperature in animals with pyrexia and reduce the risk of kidney injury from myoglobinuria secondary to rhabdomyolysis (Warantuke, 2017).

Management of tremor or convulsions

In many cases of tremorgenic mycotoxicosis in dogs, diazepam is ineffective (*Case report 2*) (Boysen et al, 2002; Young et al, 2003; Kormpou et al, 2018) and other sedatives and anticonvulsants will be required. Propofol, ketamine, levetiracetam or barbiturates can be used. Methocarbamol, a centrally acting skeletal muscle relaxant, can also be used for muscle tremors. An intravenous product is not available in many countries so the tablets will need to be crushed and given via a gastric tube in sedated animals. If tremors or convulsions are not controlled, general anaesthesia may be required. Inhalant anaesthetics can also be used, particularly where lipid emulsion is used (as barbiturates and propofol are themselves lipid soluble).

Lipid emulsion has been shown to be beneficial in cases of suspected tremorgenic mycotoxicosis in dogs, and can reduce hospitalisation time. In a study of 53 dogs with suspected tremorgenic mycotoxicosis treated with lipid emulsion, the mean time to improvement was 4 hours (Kormpou et al, 2018), compared to previous reports where recovery time was 24–96 hours (Boysen et al, 2002; Barker et al, 2013). Early use of lipid emulsion is recommended if the exposure is thought to be significant, there are significant neurological signs or there is failure to respond to other therapies.

Conclusions

Tremorgenic mycotoxicosis is relatively common in dogs because of their indiscriminate eating habits. It should be suspected in any dog with sudden onset tremors or seizures, particularly if there is a history of bin-raiding or eating mouldy material. Treatment is supportive and aimed at controlling increased muscle activity and preventing complications of prolonged seizure activity. Diazepam is often ineffective and other sedatives, anticonvulsants and anaesthetics will be required. Lipid emulsion is also recommended in tremorgenic mycotoxicosis in dogs.

Conflicts of interest

The author has no conflicts of interest to delcare.

References

Anich S. Cheese overload. Control Ther Ser. 1990;157:243
Arp LH, Richard JL. Intoxication of dogs with the mycotoxin penitrem A. J Am Vet Med Assoc. 1979;175(6):565–566

Arp LH, Richard JL. Experimental intoxication of guinea pigs with multiple doses of the mycotoxin, penitrem A. Mycopathologia. 1981;73(2):109–113
Barker AK, Stahl C, Ensley SM, Jeffery ND. Tremorgenic mycotoxicosis in dogs. Compend Contin Educ Vet. 2013;35(2):E2
Boysen SR, Rozanski EA, Chan DL et al. Tremorgenic mycotoxicosis in four dogs from a single household. J Am Vet Med Assoc. 2002;221(10):1441–1444. https://doi.org/10.2460/javma.2002.221.1441
Bradford HF, Norris PJ, Smith CC. Changes in transmitter release patterns *in vitro* induced by tremorgenic mycotoxins. J Environ Pathol Toxicol Oncol. 1990;10(1–2):17–30
Braselton WE, Rumler PC. MS/MS screen for the tremorgenic mycotoxins roquefortine and penitrem A. J Vet Diagn Invest. 1996;8(4):515–518. https://doi.org/10.1177/104063879600800427
Catovic S, Filipovic N, Stern P. The effect of penitrem A upon the level of glycine in the CNS. Bull Scientifique. 1975;20:284–285
Cavanagh JB, Holton JL, Nolan CC et al. The effects of the tremorgenic mycotoxin penitrem A on the rat cerebellum. Vet Pathol. 1998;35(1):53–63. https://doi.org/10.1177/030098589803500105
Chapman E. Nursing a canine with presumptive tremorgenic mycotoxicosis following ingestion of mouldy dog food – a case report. Vet Nurs J. 2018;33(6):166–169. https://doi.org/10.1080/17415349.2018.1457460
Eriksen GS, Hultin Jäderlund K, Moldes-Anaya A et al. Poisoning of dogs with tremorgenic Penicillium toxins. Med Mycol. 2010;48(1):188–196. https://doi.org/10.3109/13693780903225821
Eriksen GS, Moldes-Anaya A, Fæste CK. Penitrem A and analogues: toxicokinetics, toxicodynamics including mechanism of action and clinical significance. World Mycotoxin J. 2013;6(3):263–272. https://doi.org/10.3920/WMJ2013.1574
Frisvad JC, Filtenborg O. Terverticillate penicillia: chemotaxonomy and mycotoxin production. Mycologia. 1989;81(6):837–861. https://doi.org/10.2307/3760103
Hayes AW, Presley DB, Neville JA. Acute toxicity of penitrem A in dogs. Toxicol Appl Pharmacol. 1976;35(2):311–320. https://doi.org/10.1016/0041-008X(76)90290-8
Hocking AD, Holds K, Tobin NF. Intoxication by tremorgenic mycotoxin (penitrem A) in a dog. Australian Vet J. 1988;65(3):82–85. https://doi.org/10.1111/j.1751-0813.1988.tb07366.x
Knaus HG, McManus OB, Lee SH et al. Tremorgenic indole alkaloids potently inhibit smooth muscle high-conductance calcium-activated potassium channels. Biochemistry. 1994;33(19):5819–5828. https://doi.org/10.1021/bi00185a021
Kormpou F, Adamantos S, O'Sullivan A, Troth L. Use of lipid emulsion in dogs with suspected tremorgenic mycotoxicosis: 53 cases. VE. 2018;3(2):1–8. https://doi.org/10.18849/ve.v3i2.166
Laws I, Mantle PG. Elimination of roquefortine in the rat. Mycotox Res. 1987a;3(1):3–6. https://doi.org/10.1007/BF03191978
Laws I, Mantle PG, Smith BL. Excretion and transformation of 14C penitrem A mycotoxin in sheep bile. Res Vet Sci. 1987b;43(2):199–202. https://doi.org/10.1016/S0034-5288(18)30773-2
Lowes NR, Smith RA, Beck BE. Roquefortine in the stomach contents of dogs suspected of strychnine poisoning in Alberta. Can Vet J. 1992;33:335
Moldes-Anaya A, Wilkins AL, Rundberget T, Faeste CK. *In vitro* and *in vivo* hepatic metabolism of the fungal neurotoxin penitrem A. Drug Chem Toxicol. 2009;32(1):26–37. https://doi.org/10.1080/01480540802416232
Moldes-Anaya AS, Fonnum F, Eriksen GS et al. In vitro neuropharmacological evaluation of penitrem-induced tremorgenic syndromes: importance of the GABAergic system. Neurochem Int. 2011;59(7):1074–1081. https://doi.org/10.1016/j.neuint.2011.08.014
Moldes-Anaya A, Rundberget T, Fæste CK, Eriksen GS, Bernhoft A. Neurotoxicity of *Penicillium crustosum* secondary metabolites: tremorgenic activity of orally administered penitrem A and thomitrem A and E in mice. Toxicon. 2012;60(8):1428–1435. https://doi.org/10.1016/j.toxicon.2012.10.007
Munday JS, Thompson D, Finch SC et al. Presumptive tremorgenic mycotoxicosis in a dog in New Zealand, after eating mouldy walnuts. NZ Vet J. 2008;56(3):45–147
Naude TW, O'Brien OM, Rundberget T et al. Tremorgenic neuromycotoxicosis in 2 dogs ascribed to the ingestion of penitrem A and possibly roquefortine in rice contaminated with *Penicillium crustosum*. J S Afr Vet Assoc. 2002;73(4):211–215. https://doi.org/10.4102/jsava.v73i4.589
Norris PJ, Smith CCT, Belleroche JD et al. Actions of tremorgenic fungal toxins on neurotransmitter release. J Neurochem. 1980;34(1):33–44. https://doi.org/10.1111/j.1471-4159.1980.tb04618.x
Puls R, Ladyman E. Roquefortine toxicity in a dog. Can Vet J. 1988;29:569
Puschner B. Mycotoxins. Vet Clin North Am Small Anim Pract. 2002;32(2):409–419. https://doi.org/10.1016/S0195-5616(01)00011-0
Puschner B. Penitrem A and roquefortine. In: KH Plumlee (ed). Clinical Veterinary Toxicology. St Louis: Mosby, 2004, 258-259
Richard JL, Arp LH. Natural occurrence of the mycotoxin penitrem A in moldy cream cheese. Mycopathologia. 1979;67(2):107–109. https://doi.org/10.1007/BF00440681
Richard JL, Bacchetti P, Arp LH. Moldy walnut toxicosis in a dog, caused by the mycotoxin penitrem A. Mycopathologia. 1981;76(1):55–58. https://doi.org/10.1007/BF00761899
Sobotka TJ, Brodie RE, Spaid SL. Neurobehavioral studies of tremorgenic mycotoxins verruculogen and penitrem A. Pharmacology. 1978;16(5):287–294. https://doi.org/10.1159/000136781
Uhlig S, Ivanova L, Voorspoels P, Fæste CK. *In vitro* toxicokinetics and phase I biotransformation of the mycotoxin penitrem A in dogs. Toxins (Basel). 2020;12(5):293. https://doi.org/10.3390/toxins12050293
Walter SL. Acute penitrem A and roquefortine poisoning in a dog. Can Vet J. 2002;43(5):372–374
Warantuke K. Tremorgenic mycotoxin intoxication in dogs. Today's Vet Practitioner. 2017;7(3):85–88
Young KL, Villar D, Carson TL et al. Tremorgenic mycotoxin intoxication with penitrem A and roquefortine in two dogs. J Am Vet Med Assoc. 2003;222(1):52–53. https://doi.org/10.2460/javma.2003.222.52

KEY POINTS

- Tremorgenic mycotoxicosis is relatively common in dogs because of their indiscriminate eating, scavenging and bin-raiding habits.
- Common sources are mouldy foods, particularly mouldy dairy products and bin waste.
- Typical signs of tremorgenic mycotoxicosis are rapid onset tremors, vomiting, pyrexia and convulsions.
- Symptomatic animals are also at risk of aspiration pneumonia.
- Treatment is aimed at controlling increased muscle activity and seizures.
- The tremorgenic mycotoxins, penitrem A and roquefortine are lipophilic and lipid emulsion is recommended in the management poisoning.