THE SCIENCE WORLD
A Monthly e Magazine
ISSN:2583-2212
Sept, 2023; 3(09), 2462-2466

Popular Article

Effect of Fertilizer Toxicity on Central Nervous System in Domestic Animals

Dr. Kamal Sarma[1], Dr. Jonali Devi[2] and Dr. J.S. Sasan[1]
Faculty of Veterinary Sciences & A.H., SKUAST-J, R.S. Pura, Jammu (J&K)
https://doi.org/10.5281/zenodo.8392943

Almost all fertiliser products have an N-P-K, or nitrogen, phosphorus, and potassium (potash) ratio, which can be found on both organic and non-organic goods. The majority of ready-to-use fertiliser products marketed to the general public are not very toxic, although they can cause irritating effects such as skin irritation, vomiting, diarrhoea, and loss of appetite. Poisoning is more likely in companion animals exposed to industrial, concentrated, or undiluted products, while such incidents are infrequent. The following article goes over some of the more frequent and problematic fertiliser ingredients.

Bone Meal: This is made up of defatted, dried, and flash frozen animal bones that have been ground into a powder and then repurposed as a calcium or phosphorus-rich mineral supplement. Small amounts of the substance may cause vomiting and diarrhoea, while excessive amounts may cause an FBO as the product congeals in the GIT. To lessen the likelihood of blockage and the necessity for surgery, prompt emesis (within 60 minutes) is critical. Supportive therapy may include digesting diets, hydration supplementation, anti-emetics (only if the obstruction is minor), and, in rare cases, surgical removal of the FBO.

Blood Meal is a supply of nitrogen and phosphorus that is made up of dried, powdered, flash frozen blood. Vomiting, foetid diarrhoea, and pancreatitis may occur after ingestion.

Iron: Fertilizers containing iron can cause iron poisoning, depending on the type of exposure and dose, as well as the type of iron in the product. Iron toxicosis begins with vomiting and diarrhoea, mimicking the irritating effects of general fertiliser intake. Hematemesis, melena, fatigue, tachycardia, acidosis, and effects on the liver and kidneys may be more severe in advanced cases or massive ingestions.

Nitrates: Despite the fact that urea has essentially replaced ammonium nitrate in most household fertilisers, nitrate fertilisers are still widely used on farms, ranches, and commercial sites. Because nitrates are water soluble, they can contaminate both natural and treated water supplies. Intoxication is caused by the conversion of nitrate to the more toxic nitrite in the body, which causes vasodilation

and methemoglobinemia.

Urea: In many home fertilisers, urea is often utilised as a nitrogen source. Monogastric species, such as dogs and cats, tolerate ingestions well, with gastrointestinal symptoms being the most common. Methemoglobinemia can occur after a large meal.

Insecticides: Insecticide and/or fungicide chemicals may be present in some fertilisers. Among the most regularly utilised chemicals are imidacloprid and tebuconazole. Ingestion might cause minor irritability, as well as vomiting and/or diarrhoea.

UREA TOXICITY

In patients undergoing neurosurgical treatment, evidence has been given that intravenous injection of hypertonic urea solutions reduces intracranial pressure and causes brain shrinkage (Stubbs & Pennybacker, 1960). Because of the increased osmotic pressure of the blood plasma, urea may cause water to be withdrawn from the brain tissue. However, believing such an explanation in the case of urea is challenging due to the fact that this material is often believed to freely distribute itself throughout the human body's intracellular and extracellular fluids (McCance & Widdowson, 1951) and, in particular, it does not appear to behave as an osmotically active solute in relation to the membrane of the antidiuretic hormone receptor organs, which are thought to be located in the hypothalamus area of the brain (Verney, 1954).

AMMONIUM NITRATE TOXICITY

In childhood, the brain is more vulnerable to the harmful effects of ammonium than in maturity. Hyperammonemia causes irreversible damage to the developing central nervous system, resulting in cognitive impairment, seizures, and cerebral palsy due to cortical shrinkage, ventricular hypertrophy, and demyelination. Ammonium exposure impacts various amino acid routes and neurotransmitter systems, as well as cerebral energy metabolism, nitric oxide generation, oxidative stress, and signal transduction pathways, according to recent studies.

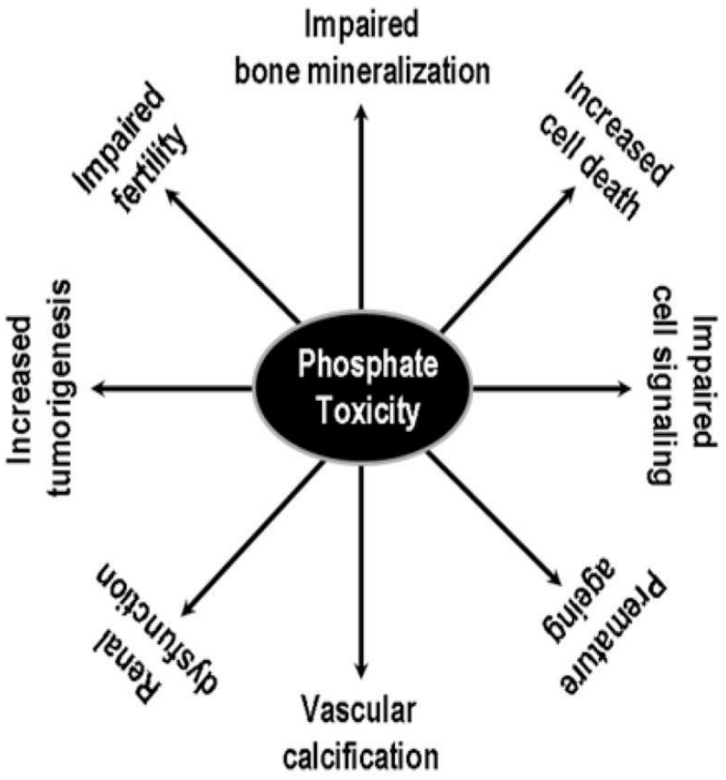
CADMIUM TOXICITY

Chronic cadmium exposure produces central nervous system problems, such as olfactory impairment. The mobility of cadmium in synapses was investigated utilising in vivo micro dialysis to elucidate cadmium toxicity in synaptic neurotransmission in the brain. The release of (109)CdCl(2) into the extracellular space was aided by stimulation with high K(+) one and 24 hours after injection into the amygdala of rats, showing that cadmium taken up by amygdalar neurons is released into synaptic clefts in a calcium- and impulse-dependent way. Furthermore, the amygdala was perfused with artificial cerebrospinal fluid with 10-30 microM CdCl to investigate the function of cadmium in synapses (2). During cadmium perfusion, the release of excitatory neurotransmitters such as glutamate and aspartate into the extracellular space was reduced, while the release of inhibitory neurotransmitters such as glycine and gamma-amino butyric acid (GABA) into the extracellular space was enhanced. These findings imply that cadmium released from amygdalar neuron terminals influences the degree

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published: **28.09.2023**

and balance of synaptic excitation-inhibition.

PHOSPHATE TOXICITY



Potash toxicity

In experimental settings, elevated brain potassium levels are linked to neuronal injury. Synaptic transmission and the normal functioning of the neuron-glia signalling network require brain ion homeostasis. Several mechanisms may contribute to transient or prolonged accumulation of brain extracellular potassium (K+) after aneurysmal subarachnoid haemorrhage (aSAH): erythrocytolysis, unspecific membrane breakdown due to parenchymal injury, dysfunction of the Na+/K+ pump, and, on the one hand, compromised glial buffering, and, on the other hand, activation of neuronal ATP–sensitive, or G protein–dependent, calcium-sensitive K. Both blood chemicals and ischemia can cause cortical spreading depolarization during aSAH (CSD). CSD causes a rise in brain extracellular Concentration ([K+]) of up to 60 mmol/L, considerably beyond its natural limit. Extracellular [K+] levels in the brain are only reached at such high levels during ictal epileptic episodes with spreading depolarization. Extracellular [K+] increases from 3 to 12 mmol/L during ictal epileptic seizures, by comparison.

Higher brain extracellular [K+] was linked to higher cerebral micro dialysis (CMD) lactate and CMD glutamate levels, increased intracranial pressure (ICP), and poor functional outcome in patients with severe traumatic brain injury (TBI), implying a link between higher brain extracellular [K+] and brain parenchyma dysfunction and/or damage. Plasma [K+] derangement is linked to muscle weakness, cardiac arrhythmia, and, to a lesser extent, cerebral symptoms like lethargy, irritability, confusion, and coma. Concomitant Na+ and acid-base abnormalities are more likely to cause

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published: **28.09.2023**

neurological symptoms, suggesting that brain extracellular [K+] may be independent of plasma K+ levels. The results of animal investigations have revealed that a disturbed blood–brain barrier might be open to big molecules while maintaining the extracellular [K+] stability. The link between plasma and cerebral extracellular [K+] following acute brain damage in humans is poorly understood.

RADIOACTIVE ELEMENT TOXICITY

DU passes the blood-brain barrier and accumulates in the brain, concentrating in certain areas. At least with oral exposure, the hippocampus and striatum absorb DU more quickly than the cerebellum and cortex. When a dust exposure protocol is used, DU accumulates in the CNS in the olfactory bulb, hippocampus, cortex, and cerebellum, displaying rising DU concentrations in that order. DU not only accumulates in the CNS, but also has physiologic activity there. DU inhibits the development of spikes in the hippocampus of rats. DU also modifies the electroencephalographic architecture of the EEG in free moving rats, resulting in abnormalities in the sleep waking cycle and REM sleep, according to research. The behaviour of rats in the open field and the Y maze is affected by DU exposure, implying that DU has neurophysiologic consequences. A number of behavioural consequences associated with DU exposure have also been documented in this lab, including impaired development and maze activities. Others, on the other hand, discovered that neither DU nor enriched uranium exposure changed sleep wake cycles or spatial behaviour.

TREATMENT

Treatment involves symptomatic and supportive care. Small Ingestions Small ingestions can be managed by the owner at home. If the pet vomits, the owner can withhold food and water for 1 to 2 hours; then gradually reintroduce water to the pet. Only 1 or 2 episodes of diarrhea is expected; no antidiarrheal medication is needed. Large Ingestions When recent large ingestions of fertilizer (> 0.5 g/ kg) occur, emesis is recommended in asymptomatic animals. At Home. Owners can induce vomiting at home with hydrogen peroxide (2.2 mL/kg body weight PO). If a significant amount of fertilizer is recovered, further treatment may not be required.

In Hospital. In the hospital, Apo morphine (0.023 mg/kg IV) can be administered to induce vomiting. Activated charcoal is not recommended because it binds poorly to minerals and is unlikely to be benefi cial. If the elemental iron dose is greater than 20 mg/kg and the animal is asymptomatic, milk of magnesia, which complexes with the iron and decreases its absorption, can be administered (5–15 mL/dog Q 12 H).

Additional Therapy:

- Antiemetic, such as maropitant (1 mg/kg SC Q 24 H) or ondansetron (0.1–0.2 mg/kg IV Q 8–12 H), are recommended if significant vomiting develops.
- Gastrointestinal protectants may be used as needed, including sucralfate (0.25–1 g/kg Q 8 H for 3–5 days), omeprazole (0.5–1 mg/kg Q 24 H), and/or a histamine-2 blocker, such as famotidine (0.5–1 mg/kg Q 12 H).

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published: 28.09.2023

- Metronidazole (10–20 mg/kg Q 8–12 H for 3–7 days as needed) is recommended when large ingestions result in bloody diarrhea.
- Fluids are recommended if an animal seems to show signs of shock or has significant vomiting and diarrhea.

Official Website
www.thescienceworld.net
thescienceworldmagazine@gmail.com
Published: **28.09.2023**