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Monograph

Nitrate Poisoning in Farm Animals

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Introduction:

Nitrate poisoning is a rare but important cause of poisoning in cattle. It can occur as the result of eating crops such as Brassicas, green cereals or sweet clover that contain high levels of nitrate. However, the most common source is inorganic nitrate fertilizer, either directly (straight from an open bag), via grazing an over-fertilized field, or via water run-off from heavily fertilized fields. Spring is the most common season for nitrate poisoning.

(Source:https://agsolutions.com.au/2020/02/06/nitratepoisoning/)

Sources:

a. **Fertilizers:** Nitrogen fertilizer, Manure increases soil nitrate concentration, application of urea to the crops, on a subsequent day if animals are grazed on such crops are succumbed due to nitrate poisoning, not due to urea poisoning

b. **Plants:** Zeamaize, cabbage, Barley, linseed, lucerne, maize, millet, oats, rape, Johnson grass (Sorghum variety), soybean, subterranean clover, Tama ryegrass, wheat. In unfavourable conditions like a sudden change in the weather, low soil pH, low soil temperature, and soil deficient in minerals are contributing to nitrate accumulation in the leaves, and lower parts of crops such as wheat, barley and oats. Mostly lower parts of plants have the highest nitrate content like stem>roots>leaves>seeds.

c. **Industrial effluent:** Effluent from dairy, meat industry and other organic industries may accumulate nitrate in their leaves and stems. Due to its high mobility, nitrate also can leach into groundwater. If people or animals drink water high in nitrate, it may cause methemoglobinemia, an illness found especially in infants (Self and Waskom, 1992).

a. **Water:** Nitrate (NO3) is a naturally occurring form of nitrogen found in soil. Nitrogen is essential to all life. Most crop plants require large quantities to sustain high yields. The formation of nitrates is an integral part of the nitrogen cycle in our environment. In moderate amounts, nitrate is a harmless constituent of food and water. Plants use nitrates from the soil to satisfy nutrient requirements and may accumulate nitrate in their leaves and stems. Due to its high mobility, nitrate also can leach into groundwater. If people or animals drink water high in nitrate, it may cause methemoglobinemia, an illness found especially in infants (Self and Waskom, 1992).
b. Dynamites explosion contains ammonium nitrate

Factors affecting nitrite poisoning:

a) **Species:** Ruminants are highly susceptible due to the rumen bacterial nitro reductase enzyme which converts nitrate to nitrite. In Horses nitrite conversion takes place in the caecum, in the swine some extend in the small intestine. Monogastric-like pigs are less likely to affect by nitrate as no conversion of nitrite and the pigs drink drainage water that has become heavily contaminated. Sometimes nitrate causes irritation to gastric mucosa leads to vomiting, diarrhea, abdominal pain
b) **Age of the plant:** mature plants contain fewer nitrates than growing plants. In mature plants, nitrate is used for protein synthesis and has less chance of toxicity while in growing plants, nitrate is being used for roots and shoots and contains more nitrate likely to cause more toxicity. Plant stressors, such as drought, are associated with increased levels of nitrate in plants. Soils high in nitrogen readily supply nitrate to plants. Additionally, factors such as acidic soils, sulphur or phosphorus deficiencies, low molybdenum and low temperatures are known to increase nitrate uptake by plants.
c) **Dose and duration of ingestion:** large quantity intake in a short time more poisoning
e) **Deficiencies:** Deficiency of Vitamin A exaggerated nitrate toxicity
f) **Adaptability:** lack of prior exposure to nitrate
g) **Poor quality f carbohydrate diet:** as an absence of carbon skeleton which hampers ammonia utilization for protein synthesis
h) **Hypovitaminosis:** Nitrate interfere with the utilization of Vitamin A, D and E, and also Iodine
i) **Other health factor:** Presence of anemia or methaemoglobinurea hasten nitrate toxicity

Toxicokinetic:

If an animal's nitrate intake is too high, or if conditions are not right for the conversion of nitrite to ammonia in the rumen, nitrite will accumulate and may be absorbed into the blood. Nitrite may also be absorbed directly from fodder such as hay if it becomes wet or mouldy. Microbes on the fodder convert nitrate to nitrite under these conditions. Nitrite reduces the ability of the blood to transport oxygen throughout the body of the animal. If the nitrite level is high enough, death can occur through oxygen starvation.

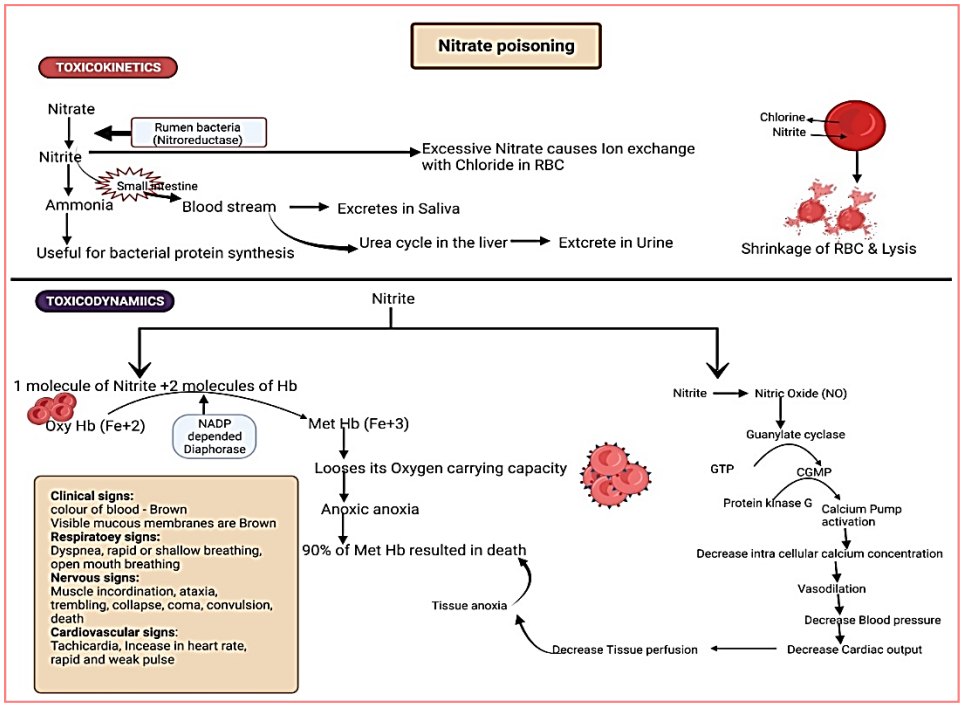
Non-ruminants, such as horses and pigs, have no mechanism for converting nitrate to nitrite in their digestive tracts and so are not in danger of getting nitrite poisoning from an excessive intake of nitrate. However, they are highly susceptible to oral intake of nitrite (for example, in mouldy hay) because they cannot convert the nitrite to ammonia.

Toxicodynamic

Nitrite: effect RBC and vascular endothelial cells after being absorbed in the bloodstream.

a) RBC: nitrite converts oxyHb (Fe+2)to MetHb (Fe+3) by NADP-dependent diaphorase enzyme systems 1 &2 when there is excess nitrite, this enzyme system gets saturated in more concentrated metHb If MetHb % is more than 20 % indicative, not harmful, while 50% and 90% showing signs and death respectively

b) Vascular endothelial cell: Nitrite will convert to NO in the vascular smooth muscle due to the presence of acetylcholine and causes vasodilation through Guanylate cyclase enzyme (Depicted in the picture)



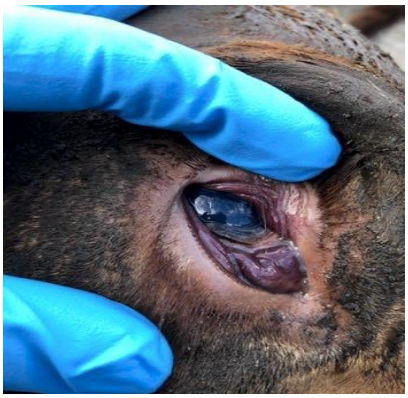
Clinical sings:

1. Acute toxicity:

- GIT effects: Local irritation, vomiting, diarrhea, colic, micturition
- CVS effects: Tachycardia, increase in heart rate, rapid and weak pulse rate
- Respiratory effects: dyspnea, rapid/shallow breathing, open mouth breathing
- Nervous effects: muscle incoordination, ataxia, trembling, collapse, coma, terminal convulsion and death
- Death 12-24 hrs.

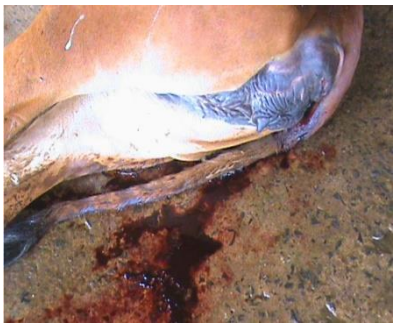
2. Chronic toxicity:

- Early abortions, nitrate interfere with implantation of fertile ova, infertility mostly due to less O_2 supply, anorexia, hypothyroidism, hypovitaminosis of A, D and E
- Mucous membrane: cyanotic
- Post mortem findings:
- Mucous membrane – cyanotic
- Dark chocolate brown color blood, tissues and organs due to methaemoglobin
- Pin-point hemorrhages may be present in the heart and trachea along with general congestion of the blood vessels.



Mud coloured mucous membranes

Source : http://www.flockandherd.net.au/cattle/ireader/nitrate-toxicity-cattle.html



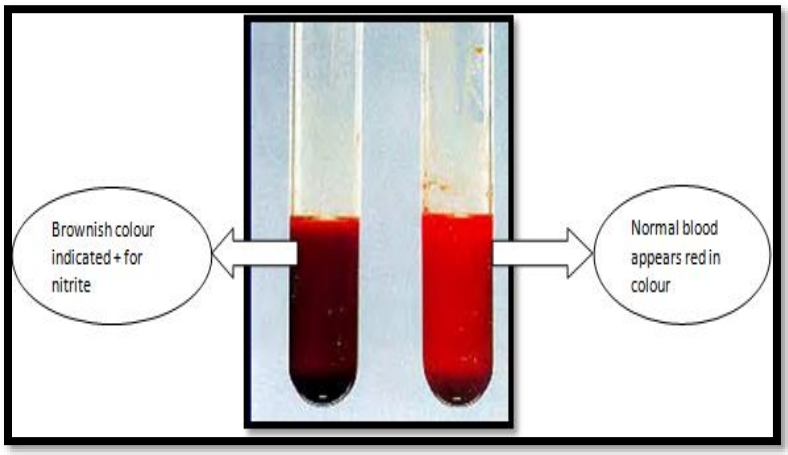
Bloody foamy oozing from nose
Source: Oruc, et al (2010)

Un clotted brown coloured blood
source: Nagarajan et al (2015)

Diagnosis:

a) Based on history

b) Based on clinical signs: cyanotic mucous membrane, presence of chocolate brown color blood (McKenzie et al, 2004; Latimer, 2011)



c) Laboratory diagnosis: estimation of blood concentration of nitrate and nitrite levels. If the nitrate and nitrite levels more than 20 ppm and 0.75 ppm respectively indicated positive

d) Rumen contents can be analyzed for nitrite levels

e) Estimation of percentage of methemoglobin (more than 70% indicated for death)

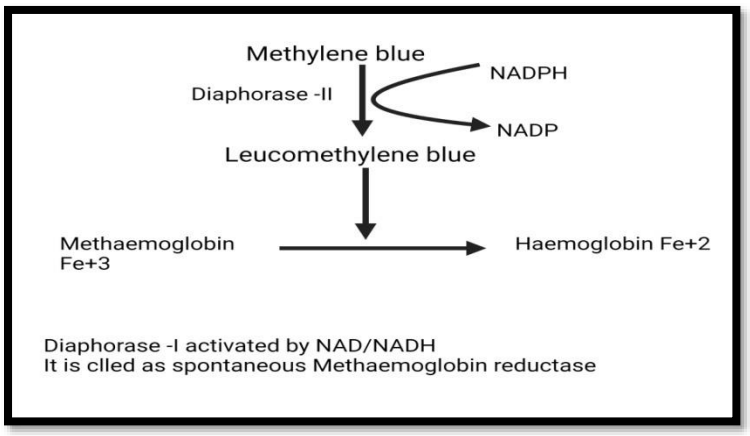
f) Eyeball and aqueous humor under refrigerated conditions which do not undergo bacterial decomposition in dead animals, more than 30 ppm of nitrite in ocular fluid indicative of its poisoning. Also, analysis of the aqueous humor using a Merck quant Strip test which is a field test for veterinarians (Ensley and Rumbeiha, 2012).). after the death of suspected animals with nitrite poisoning the collection of rumen content, urine, serum, plasma is not useful because of bacterial decomposition. Diphenylamine blue test for estimation of nitrate content in plants materials

Differential Diagnosis:

1. **Cyanide poisoning:** death is rapid 1-4 hrs., venous blood color appears as bright red or cherry red (Hemoglobin carries oxygen but is not utilized by the tissue and cherry red colored due to oxyhemoglobin), the bitter almond smell of gastric content.
2. **Hydrogen sulphide (H2S) poisoning:** the color of blood seems to be dark due to sulphides, and stomach content will be dark with rotten egg smell.
3. **Carbon monoxide (CO) poisoning:** the color of blood seems to be bright red in arteries and cyanotic in venous (carboxy hemoglobin formation as CO is 200 times more affinity for hemoglobin than O_2)

Treatment:

1. Animals can be treated by intravenous injections of methylene blue. Commercial preparations intended for the treatment of prussic acid poisoning only should not be used to treat nitrate poisoning. Methylene blue is not approved by the Food and Drug Administration for use in food-producing animals. Methylene blue (Isotonic solution 1%in distilled water) @8.8mg/kg for ruminates IV route 4.4 mg/kg in other animals' species. Methylene blue is a reducing agent and converts methemoglobin to hemoglobin and restores normal oxygen transport by the red blood cells. The half-life of methylene blue is about 2 hours, meaning that small doses can be given repeatedly every few minutes until the animal is not exhibiting severe respiratory distress.



2. Ascorbic acid@5-10mg/kg BW
3. Supportive therapy:

a. Adrenaline infusion 0.05 µg/kg/min
b. Blood transfusion
c. Saline purgative ex: magnesium sulfate (cattle, 250–500 g; horses, 30–100 g; pigs, 25–125 g; dogs, 5–25 g; cats, 2–5 g), sodium sulfate (cattle, 500–750 g; horses, 250–375 g; pigs, 30–60 g; dogs, 5–25 g; cats, 2–5 g), sugar alcohols (mannitol and sorbitol), lactulose (dogs, 5–15 mL, t.i.d., per os).
d. Mineral oil or demulcent to soothe GIT like glycerine, liquid paraffin, egg white, honey gruels administration n(mineral oil or liquid paraffin cattle, 250–500 mL; horses, 250–1000 mL; pigs, 25–300 mL; dogs, 5–30 mL; cats, 2–6 m)
e. Vitamin supplementation
f. Administration of broad spectrum antibiotics in cold water to suppress microflora chlortetracycline @30mg/kg BW for 5 days

Prevention:

✓ Prevent access to nitrate sources. In particular, prevent cattle getting into fertiliser stores. Ensiling high nitrate pastures will usually reduce the nitrate levels to safe levels (as will allowing the pasture to set seed).

- ✓ Feeding a high grain diet alongside high nitrate forage has a protective effect because carbohydrates enhance the conversion process from nitrate to microbial protein.
- ✓ When grazing, feed a dry roughage first to reduce the amount of affected plants ingested by hungry animals.
- ✓ Harvested forages that are high in nitrate often can be fed safely by mixing them with other feeds to reduce the total dietary intake of nitrate.
- ✓ Ruminants can tolerate fairly high levels of nitrate in their diet if the intake is spread over the whole of the feeding day their diet is also high in readily available carbohydrate, which is needed to fuel the microbial activity in the rumen, if these conditions are not met, the nitrate can accumulate, resulting in poisoning.

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