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Blue-green algae (cyanobacterial) toxicity in animals

Dr R D Last – MMedVet(Path) -
Specialist Veterinary Pathologist

Introduction

Cyanobacteria are capable of producing different structural groups of cyanobacterial toxins, namely cyclic peptides (microcystins, nodularins) which target hepatic function; alkaloids (anatoxins, saxitoxins) that target the nervous system, inflammatory agents (lyngbyatoxin-a and aplysiatoxin) which induce skin lesions and endotoxins (lipopolysaccharides) which are cutaneous and gastrointestinal irritants.

Additive, synergistic and/or antagonistic effects among different classes of cyanobacterial metabolites, as well as interactions with other toxic stressors such as metals or persistent organic pollutants can impact on the severity of toxicity.

The occurrence of cyanobacterial mass populations can create a significant water quality problem, especially as many cyanobacterial species are capable of synthesising a wide range of odours, noxious compounds or potent toxins. The cyanotoxins are a diverse group of compounds, both from the chemical and the toxicological points of view. Cyanobacterial toxins are hepatotoxic, neurotoxic, cytotoxic, dermatotoxic and irritant toxins.

Toxic blooms of cyanobacteria have been associated with acute, usually lethal toxicity in a wide range of mammalian species (cattle, sheep, swine, dogs, bats, mice, guinea pigs, humans) and multiple outbreaks in the Kruger National Park have involved a wide range of African wildlife species (zebra, wildebeest, white rhinoceros, impala, lion, cheetah).

Cyanobacterial toxins

Hepatotoxins

- Microcystins (*Microcystis, Anabaena, Oscillatoria, Nostoc, Planktothrix, Anabaenopsis, Hapalosiphon*). Hepatotoxic, protein phosphatase inhibition, membrane integrity and conductance disruption, tumour promoters
- Nodularins (*Nodularia)*. Hepatotoxic, protein phosphatase inhibition, membrane integrity and conductance disruption, tumour promoters, carcinogenic
- Cylindrospermopsins (*Raphidiopsis / Cylindrospermopsis, Aphanizomenon, Anabaena, Raphidiopsis, Umezakia*). Necrotic injury to liver (also to kidneys, spleen, lungs, intestine), protein synthesis inhibitor, genotoxic.

Neurotoxins

- Anatoxin-a (*Aphanizomenon, Anabaena, Raphidiopsis, Oscillatoria, Planktothrix, Cylindrospermum*). Postsynaptic, depolarising neuromuscular blockers
- Anatoxin-a(S) (*Anabaena*). Acetylcholinesterase inhibitor
- Saxitoxins (*Aphanizomenon, Anabaena, Planktothrix, Cylindrospermopsis, Lyngbya*). Sodium channel blockers.

Dermatotoxins

- Lyngbyatoxin-a (*Lyngbya, Schizotrix, Oscillatoria*). Inflammatory agent, protein kinase C activator.
- Aplysiatoxin (*Lyngbya, Schizotrix, Oscillatoria*). Inflammatory agents, protein kinase C aktivators

Endotoxins

- Lipopolysaccharides (all cyanobacteria). Inflammatory agents, gastrointestinal irritants.

1. Hepatotoxins

The **microcystins** are predominantly produced by the genera *Microcystis*, *Anabaena* and *Oscillatoria* and are protein phosphatase inhibitors and potent hepatotoxins.

Toxicity of microcystis in mammals is characterised by fulminant intrahepatic haemorrhage followed by hypovolaemic shock, secondary to massive hepatocellular necrosis and collapse of hepatic parenchyma and death. Hepatic toxicity is dose dependent and so pathology can vary from acute, subacute to chronic. Gross pathology is characterised by body cavity effusions, generalised petechiae and massive hepatic necrosis.

Microcystins absorbed from the gastrointestinal tract are taken up in the blood via a multispecific bile acid transport system. Rounding of hepatocytes occurs concurrently with the loss of normal hepatic architecture, which is the result of cytosolic interaction of microcystin with phosphatases which are essential for maintaining the cytoskeletal intermediate filaments.

The nett result is cell distortion which is visualised histologically as dissociation of the hepatocellular cords with widening of the space of Disse, and such histopathology is considered of significant diagnostic value.

Cylindrospermopsins are toxic guanidine alkaloids produced by blue-green algae (cyanobacteria) with poisonings reported in cattle and humans (Palm Island Disease). In both cattle and man, the condition is characterised by a chronic necrotising hepatopathy, frequently accompanied by severe diarrhoea. Hepatic histopathology is of hepatocellular vacuolar degeneration and necrosis accompanied by portal fibrosis with bile duct hyperplasia, which is quite different from that described with the microcystins.

The cylindrospomopsin producing cyanobacteria do not necessarily accumulate on the surface but can be distributed through the water column and may not be grossly visible. In general, these blooms do not float on the water surface. This is of significance when analysing water sources for potential toxicity in that water samples need to be collected at various levels through the water column and not just from the surface.

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2. Neurotoxins

Neurotoxic blooms (anatoxin, saxitoxin) have been associated with acute deaths in dogs swimming in contaminated rivers. All mammalian species are susceptible to poisoning by these toxins, which act very rapidly, causing paralysis. The clinical coarse is usually per-acute with affected animals usually being found dead near the implicated contaminated water source. Those seen alive usually exhibit severe dyspnoea with sudden collapse and die acutely. Pathology is unremarkable with neurotoxic blooms.

3. Dermatotoxins

Lyngbya, Schizotrix and *Oscillatoria* cyanobacteria produce dermatotoxins (Lyngbyatoxin-a, aplysiatoxin) induce inflammation and irritation to eyes (keratitis, conjunctivitis) and skin, when animals come into contact with cyanobacterial blooms containing these toxins. In some individuals they are capable of inducing allergic skin reactions.

4. Endotoxins

Lipopolysaccharide endotoxins are produced by all species of cyanobacteria. These lipopolysaccharides have irritant effects inducing skin irritation with contact and gastrointestinal irritation with ingestion.

Diagnosis

Confirmation of fatal blue-green algal toxicity requires the following

- Collecting bloom material preserved for identification of the organism (20 ml bloom + 1 ml formalin) and unpreserved (chilled) for toxicity testing (ideally 1 litre, minimum 20 ml). It is important that not only surface bloom but also water from deeper within the water column is included.
- Collection of tissues into 10% formalin for histopathology.

Prevention and Control

- Avoid animal exposure to water containing the algae. Animals should be stopped from drinking from the floating bloom material to prevent poisoning. To prevent intake of intact surface blooms, water supplies should be drawn from sites away from blooms. Remember pumping of surface material bloom to water troughs does not eliminate exposure, as the water remains toxic in the troughs.
- Ideally animals should be moved to another pasture, the affected water supply fenced off and an alternative clean water supply provided.
- Use of algaecide in ponds with cyanobacterial bloom can be considered to destroy algae. This will release the toxins into the water when the cyanobacterial cells die and rupture. Livestock should not be given access to this treated water for at least 7-10 days because it will remain poisonous and needs time to allow for the degradation of the cyanobacterial toxins. It is better to allow blooms to decay and disperse naturally.

Effective management of the conditions that promote algal bloom formation markedly reduce the risk of outbreaks of cyanobacterial poisoning. Reducing the entry of excessive amounts of nutrients (phosphates, nitrates, organic material) into the water body and catchment areas, dams, streams and reservoirs, is central in preventing algal blooms. This is best achieved by

- Minimising fertilizer runoff in the catchment areas
- Fencing livestock out of water sources and pumping drinking water to troughs. This eliminates faecal and urine, which is high in nitrogen and phosphorus, directly entering water sources. In addition, exclusion of livestock prevents soil erosion on the banks of the water source thereby, reducing the amount of organic material entering the water supply.
- Minimise or prevent livestock faecal and human sewage inflows into water sources.
- Locate the supplementary feeding sites for grazing livestock outside of the water catchment areas of stock water supply dams. This will prevent the large amounts of faeces that build up at such places, being washed into the dams by heavy rain. Treated sewage should preferably be used for irrigation instead of being directly discharged into water bodies.

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MULTIPLE-CHOICE QUESTIONS

QUESTION 1

Which of the following chemical compounds are NOT considered cyanobacterial toxins?

a. Nodularins
b. Saxitoxin
c. Methylindole
d. Microcystin
e. Anatoxin

QUESTION 2

Blocking of sodium channels OCCURS WITH which blue-green algal toxin?

a. Saxitoxin
b. Lipopolysaccharide
c. Cylindrospermopsin
d. Lyngbyatoxin-a
e. Anatoxin-a(S)

QUESTION 3

Which of the following cyanobacteria are capable of PRODUCING hepatotoxins, neurotoxins, dermatotoxins and endotoxins?

a. *Microcystis*
b. *Nodularia*
c. *Nostoc*
d. *Planktothrix*
e. *Oscillatoria*

QUESTION 4

On histopathology which of the following hepatic lesions is considered of HIGH diagnostic value for possible microcystin exposure?

a. Vacuolar change of hepatocytes (fatty liver)
b. Hepatocellular cord dissociation
c. Bile duct hyperplasia
d. Portal fibrosis
e. Cholestasis

QUESTION 5

Which of the following statements about the cylindrospermopsins is TRUE?

a. They are pyorrlizidine alkaloids
b. They are rarely associated with clinical diarrhoea
c. They induce acute hepatic necrosis with haemorrhage
d. In general blooms do not float
e. Surface water samples provide the best diagnostic samples

QUESTION 6

What is the MOST COMMON clinical presentation with ingestion of toxic doses of cyanobacterial neurotoxins?

a. Ataxia with low mortality
b. Tremors with chronic course
c. Paralysis with dyspnoea and per-acute mortality
d. Incoordination with subacute presentation
e. Convulsions with acute onset but low mortality

QUESTION 7

Ingestion of lipopolysaccharide endotoxins is associated WITH WHICH of the following clinical syndromes?

a. Hepatotoxicity
b. Paralytic syndromes
c. Renal failure
d. Keratitis
e. Gastrointestinal irritation

QUESTION 8

Which of the following is the PREFERRED SAMPLE for algal identification?

a. 1 ml algal bloom plus 20 ml of formalin
b. 20 ml algal bloom plus 1 ml of formalin
c. 1 litre of fresh bloom material at room temperature
d. 20 ml of fresh bloom material chilled
e. 1 litre of fresh bloom material chilled

QUESTION 9

Following algaecide treatment of a water source with a toxic algal bloom, what is considered a SAFE WAITING period before reintroduction of livestock to the water source?

a. 2 days
b. 3 days
c. 4 days
d. 5 days
e. >7 days

QUESTION 10

Which is considered the MOST EFFECTIVE way of preventing algal blooms in bodies of water?

a. Regular application of algecides
b. Chlorination of the water source
c. Reducing excessive nutrients entering the water source
d. Pumping drinking water to troughs
e. Only allowing treated sewage into water bodies

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