ACUTE HEPATIC NECROSIS

Common effects of acute hepatotoxins from plants, mycotoxins, cyanobacteria, macrofungi and sawfly larvae

Core data

Common sources:
- furanosesquiterpenes from *Eremophila deserti* and some *Myoporum* spp.
- diterpenoid (kaurene) glycosides from *Cestrum parqui* and *Xanthium occidentale*
- microcystin from various cyanobacteria including *Microcystis aeruginosa*
- nodularin from cyanobacterium *Nodularia spumigena*
- amatoxins in *Amanita* spp. macrofungi
- lophryotomin and pergidin in sawfly larvae
- aflatoxins
- methylazoxymethanol in cycads
- *Trema tomentosa* (uncharacterised toxins)

Animals affected: ruminants, horses, pigs, dogs, deer, camels
Mode of action: various
Poisoning circumstances: hungry animals with access to large amounts of plant
Main effects:
- acute hepatocyte coagulation necrosis (extent and distribution varies)
- hepatoencephalopathy

Diagnosis: pathology + toxin source access
Therapy:
- prognosis is poor to grave in most cases
- no specific therapy is available
- use general decontamination techniques including activated charcoal PO

Prevention: deny access ; see individual toxins

The major toxic causes of acute hepatic necrosis in domestic animals from biological sources include
- furanosesquiterpenes (*q.v.*)
- diterpenoid (kaurene) glycosides - atractyloside, carboxyatractyloside, parquin, wedeloside (*q.v.*)
- cyanobacterial hepatotoxic cyclic peptides – microcystin (*q.v.*) and nodularin (*q.v.*)
- macrofungal peptides (amatoxins) in *Amanita* spp. (*q.v.*)
- sawfly larval peptides lophryotomin and pergidin (*q.v.*)
- aflatoxins (*q.v.*)
- methylazoxymethanol (MAM) (*q.v.*) in cycads
- gossypol (*q.v.*)
- plants with unknown or uncharacterised toxins including
  - *Trema tomentosa* (*q.v.*)
  - *Cynosurus echinatus* (rough dog's-tail grass) (*q.v.*)
  - *Lythrum hyssopifolia* (lesser loosestrife) (*q.v.*)

Minor toxic causes of acute hepatic necrosis in domestic animals from biological sources include
- *Argentipallium blandowskianum* (*q.v.*)
- *Ozothamnus diosmifolius* (*q.v.*)
- pulegone [pennyroyal oil] (*q.v.*)

Minor toxic causes of acute hepatic necrosis in domestic animals from non-biological sources include
- iron (*q.v.*)
- coal tar products (*q.v.*)
- molybdenum – acute (*q.v.*)
- phosphorus (*q.v.*)
- paracetamol (cats + other species) (*q.v.*)

- aspirin (acetylsalicylic acid) (*q.v.*)
- other NSAIDs (ibuprofen, naproxen, phenylbutazone) (*q.v.*)
- imidocarb (*q.v.*)
- gaseous anaesthetic agents – dogs (*q.v.*)

Toxicity:

Domestic **ruminants** are the animal species affected most commonly, but cases are also known in

- dogs (cycads, aflatoxins, macrofungi, cyanobacteria)
- pigs (*Xanthium occidentale, Perreyia flavipes* sawfly larvae)
- horses (*Trema tomentosa*) (Hill *et al.* 1985)
- deer (*Trema tomentosa*) (McKenzie 1985, 1992)
- camels (*Trema tomentosa*) (Trueman & Powell 1991)

Conditions of poisoning:

hungry animals (e.g. travelling livestock, newly-introduced livestock) with access to significant populations of the plants

many of the hepatotoxic flowering plants listed are not hazardous to livestock under normal paddock conditions, often because of unpalatability or insufficient population density

Clinical signs:

Ruminants – signs of liver dysfunction

anorexia
depression
rumen stasis
abdominal pain (teeth grinding, saw-horse stance, kicking at the belly)
± jaundice
recumbency
coma, death within 24 hours.

Ruminants – nervous signs (**hepatoencephalopathy** originating from hypoglycaemia and hyperammonaemia, but probably multifactorial)

muscle tremor
hyperexcitability
↑ aggression
mania
blindness
lack of response to external stimuli
convulsions
coma.

Monogastrics

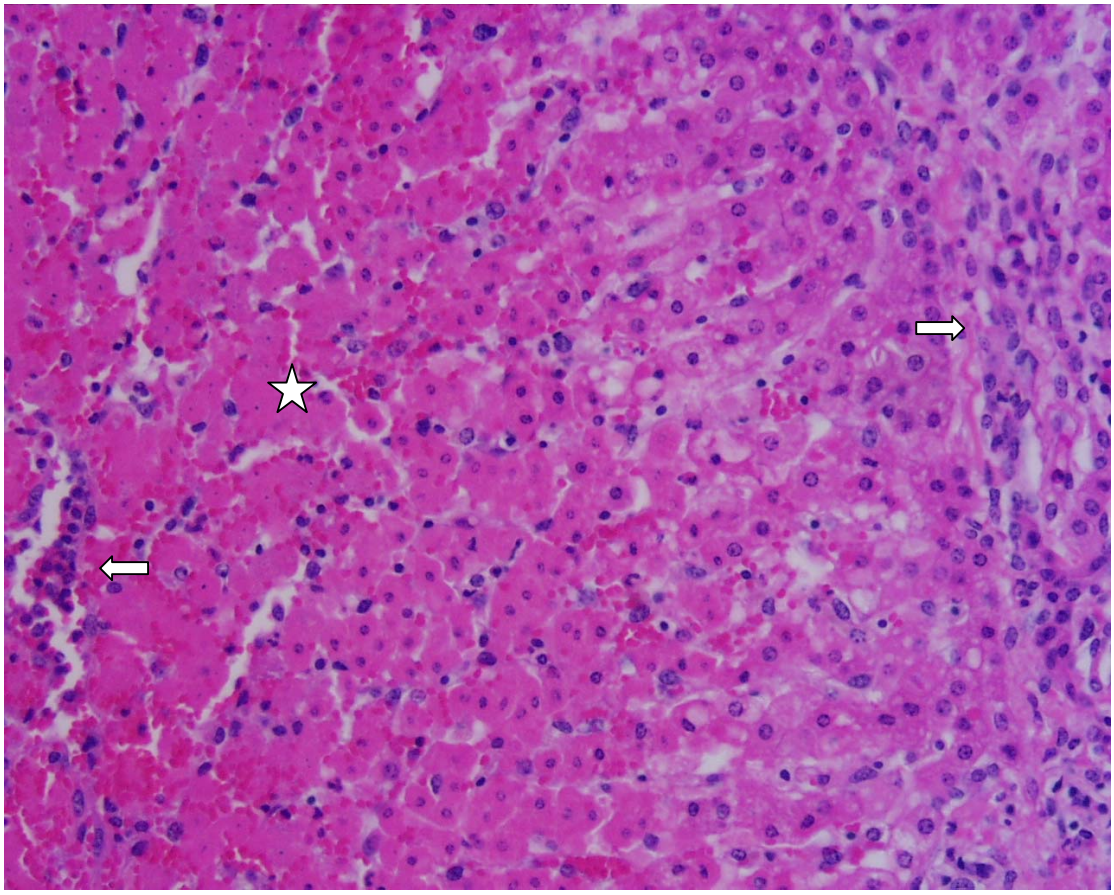
anorexia
depression
abdominal pain (teeth grinding, saw-horse stance, kicking at the belly)
± jaundice
± vomiting
± diarrhoea
recumbency
coma, death within 24 hours.

Pathology:

↑ liver-associated serum enzymes (AST, GLDH), ↑ serum bilirubin

necropsy/histopathology

± jaundice
liver swollen, congested, zonally mottled
coagulation necrosis of hepatocytes (periacinar; periportal; rarely midzonal), ± haemorrhage
gall bladder oedema
haemorrhage in the lower alimentary tract (?portal hypertension) and elsewhere (?exhaustion of clotting factors in damaged liver)



Cestrum parqui (green cestrum) periacinar hepatocyte coagulation necrosis. H&E x . Central vein (left arrow); portal triad (right arrow); necrotic hepatocytes (star) [RAM Photo]

Diagnosis:

liver pathology/clinical pathology

access to plant, cyanobacteria, mycotoxin-containing feed or insect larvae

detection of the plants responsible at or near the site of mortality – This can be difficult

examination of rumen or stomach contents for recognisable plant fragments – The success rate is low for this procedure.

Therapy:

prognosis is poor to grave in most cases

no specific therapy is available

use general decontamination techniques (*q.v.*) including activated charcoal PO

Prevention & control:

prevent access