Turned toxic....

How to deal with poisoning in small animals

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Challenges

- Toxicity is often uncertain
Poisoning

• Poisoning is usually accidental
  – Scavenging

• Can be owner-induced
  – Ingestion of human medication

• Malicious poisoning is rare
Clinical Signs

• Can be rapid, severe and potentially fatal
• Non-specific
• Mimic other disease & other toxicities
• Reflect toxin’s target organ / system
  - E.g. Rodenticides $\rightarrow$ coagulation
    • bleeding
Diagnosis

• Index of suspicion
• Thorough history & physical examination
• Toxicological analysis is available for some toxins
  – Collect relevant samples
    • blood, serum, vomit, liver, kidney, brain, urine, food, water
Accurate records
Information Sources

• Veterinary Poisons Information Service
  – 24 hour advice service
  – available only to veterinary practices
  – Cost per case system

• Reference textbooks
Top ten enquiries to VPIS

• Anticoagulant rodenticides
• NSAID’s
• Chocolate
• Paracetamol
• Permethrin

• Metaldehyde
• Lillies
• Grape/raisin/sultana
• Batteries
• Adder bites
Initial management

- Telephone advice
- Stabilise vital signs
- History and physical examination
- Prevent continued absorption
- Increase excretion
- Supportive care and monitoring
- Antidote
Telephone advice

• What are the clinical signs?
  – i) does pet need examining
  – ii) is poisoning a possibility
  – iii) can the owner do anything
Initial management

- Telephone advice
- Stabilise vital signs
- History and physical examination
- Prevent continued absorption
- Increase excretion
- Supportive care and monitoring
- Antidote
Initial Stabilisation

- Airway
- Breathing
- Circulation
- Vascular access/Fluid therapy
- Manage life threatening signs
  - seizures, arrhythmias
- Monitor vital signs
Initial management

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Initial management

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Eliminating further absorption

- Toxin may be absorbed from skin, gut or both
- Buster collar or muzzle
- Skin & coat can be washed or clipped
- Decontamination of the gut
Prevent further GI absorption

- Induce Vomiting
- Gastric Lavage
- Adsorbent
Induce Vomiting

• Indicated
  – within 2-3 hrs from ingestion

• Contraindicated
  – respiratory depression
  – seizures, reduced consciousness
  – ingested caustic substance

• Drugs
  – Apomorphine
  – Alpha-2 agonist
    • medetomidine
    • xylazine
  – 3% Hydrogen peroxide
  – Syrup of Ipecac
  – Washing liquid
  – Soda crystals
Gastric Lavage

- Indicated
  - if vomiting is contraindicated or ineffective
- Risks
  - Aspiration
  - Incomplete evacuation
  - Anaesthetic

- Pre-measured stomach tube placed
- Lavage with warm saline or tap water
- Drain
- Continue until clear lavage fluid
Adsorbent

• Binds toxin and prevents further absorption from gut
• Activated charcoal commonly used
  – Oral or via stomach tube
  – Repeated administration
    • Every 4-6 hours interrupts enterohepatic recycling
  – Not effective for alcohol, corrosives, iron, lithium
WBI?
Initial management

• Telephone advice
• Stabilise vital signs
• History and physical examination
• Prevent continued absorption
• Increase excretion
• Supportive care and monitoring
• Antidote
Hasten Elimination

• Cathartic or laxative

• Increase renal elimination
  – Intravenous fluids ± diuretic agents

• Dialysis
Initial management

- Telephone advice
- Stabilise vital signs
- History and physical examination
- Prevent continued absorption
- Increase excretion
- Supportive care and monitoring
- Antidote
Supportive Therapy

- Fluid therapy
- Maintain body temperature
- Eliminate toxin from environment
- Decontaminate skin/coat if not already done
- Support vital body functions
  - Cardiorespiratory
  - Neurological
  - Renal
  - Hepatic
- Consider nutrition
Initial management

• Telephone advice
• Stabilise vital signs
• History and physical examination
• Prevent continued absorption
• Increase excretion
• Supportive care and monitoring
• Antidote
Treating the effects of the toxin

- **Haemorrhage**
  - Apply pressure
  - Fluids+/- blood
- **Seizures**
  - Diazepam, barbiturates, propofol CRI
- **Respiratory depression**
  - Oxygen
  - Ventilation
- **Hyperaesthesia**
  - Sedatives
- **Tachycardia**
  - Beta blockers, antidysrhythmics
- **Bradycardia**
  - Atropine
- **Hypothermia**
  - External warming, warm water enema or lavage
- **Hyperthermia**
  - Ice packs, muscle relaxants, NSAIDs
Specific toxins
Lily toxicity

• Liliaceae plant family can cause ARF
  – Consider any *Lilium* spp or *Hemerocallis* spp potentially nephrotoxic

• Neither Lily-of-the-Valley (*Convallaria majalis*), the peace lily (*Spathiphyllum* spp) nor *Calla* lily are true lilies
  – Not nephrotoxic
    • Peace and Calla contain oxalates
      – Develop stomatitis which limits ingestion and stops renal failure
    • Lily of the Valley contains cardiac glycosides


Clinical Signs

• Signs develop within 12 hours and progress to renal failure within 2-3 days
• Vomiting
• Inappetance /anorexia
• Depression /lethargy
• Oliguria/Anuria
• CNS signs
  – Ataxia, head pressing, disorientation, tremors, seizures
Clinicopathological findings

• Biochemistry
  – Acute renal failure
    • Azotaemia, hyperkalaemia, hyperphosphatemia

• Urinalysis
  – Epithelial casts
  – Submaximally concentrated urine SG
  – Proteinuria
  – Glucosuria
Treatment

• Induce emesis if < 2 hours from ingestion
  – Adsorbent

• IVFT <6 hours of ingestion
  – Can prevent the development of renal failure
  – Continue for at least 48 hrs

• > 6 hours post ingestion
  – consider dialysis

• IVFT starts > 18 hours after ingestion or anuria
  – Poor prognosis
12 week old FE DSH
48hr history lethargy, anorexia, vomiting and abdominal pain
No improvement with symptomatic therapy
  – Including NSAID’s
Depressed, generalised tremor, ataxia
Abdominal palpation – renal pain and irregularity
Hypothermia (36.7C)
Dehydration
• Urea 129 mmol/l (6.5-10.5)
• Creatinine 1800 umol/l (133-175)
• Sodium 137.3 mmol/l (149-157)
• Potassium 10.16 mmol/l (4-5)
• Calcium 2.21 mmol/l (2.3-2.5)
• Phosphate 4.36 mmol/l (0.95-1.55)
• pH 6.9 (7.3 +/- 0.08)

• Ultrasound
  – Bilateral hyperechoic kidneys
  – Small volume ascites and retroperitoneal fluid
• Treatment
  – Intravenous fluid therapy
  – Diuretics
  – Calcium gluconate and glucose to manage hyperkalaemia
  – Sodium bicarbonate to treat acidosis
  – Indwelling urinary catheter
• Despite 8hrs treatment remained anuric
  – Anuric for 36 hours prior to presentation
  – Further clinical deterioration
• Owner then reported observing kitten to have eaten Lily flowers
Anticoagulant rodenticides

Vitamin K (active)

Epoxide reductase reaction

Vitamin K (inactive)

Clotting factors II, VII, IX, X (inactive)

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Clinical effects may not show for 1-3 days
Diagnosis

• History
  – Possible exposure

• Clinical signs
  – Lethargy
  – Exercise intolerance
  – Respiratory distress
  – Hypovolaemic shock
  – Bleeding
    • Internal
    • External

• Clotting profile
  – Activated clotting time
  – PT and APTT
Clinical signs

• Haemorrhage into major body cavities
Clinical Signs

- Bleeding into lung parenchyma
- Bleeding into sub-mucosa of trachea
- Bleeding into GI tract
- Bleeding from nose
- Bleeding into joints
- Bleeding into subcutaneous space
- Repeated bleeding at venepuncture sites
Other abnormalities

- Anaemia
- Hypoproteinaemia
Management

• Gastric decontamination
  – <3h post ingestion
  – Emesis +/- gastric lavage
  – Adsorbents

• Not if already bleeding
Management (cont)

• Cage rest
• IVFT
• Drainage of cavity bleeds if necessary
• Blood/blood products
  – Fresh or stored blood contain adequate Vitamin K dependent clotting factors
    • Synthesis takes approx 6-12 hours
• Oxygen
Management

• Vitamin $K_1$ (Konakion – 2 or 10mg/ml)
  – S/C (multiple sites) then orally
  – 5mg/kg s/c then 2.5-5mg/kg divided twice daily orally
  – Treat for 7-28 days depending on toxin
  – Check PT 2 days after finishing treatment and continue if necessary

• Avoid Vitamin $K_3$
Monitoring

• PT should normalise within 1 hour of plasma transfusion
• PT will normalise after 12-24 hours of Vitamin K alone
• Re-evaluate PT 2-4 days after stopping Vitamin K
Pyrethrins & Pyrethroids

• Misuse of concentrated products e.g. canine
  – Misadministration of canine product
  – Close contact recently treated dog

• Alter activity of sodium channels
  – Increase duration of depolarisation
Pyrethrins & Pyrethroids

• Clinical signs
  – Vomiting,
    hypersalivation, ataxia,
    mydriasis, tachycardia
    hyperaesthesia,
    hyperthermia, tremor,
    twitching, seizures,
    respiratory distress
  – Death possible
  – Often dermal
Pyrethrins & Pyrethroids

• Treatment
  – Gut decontamination only if oral exposure
    • Activated charcoal
  – Dermal decontamination
    • Copious washing lukewarm water with mild detergent
  – Muscle tremor
    • Diazepam
    • Methocarbamol
  – Seizures
    • Diazepam
    • Phenobarbitone
    • CRI propofol
  – IV fluids
Ethylene Glycol

- Renal & metabolic effects
- Antifreeze & screen washes
- Sweet aromatic smell and taste
- Lethal dose:
  - 4-6ml/kg (dog), 1.4ml/kg (cat)
- Effects are dose dependent
Signs

• Drunk for 1st few hours
• PU/PD and dehydration within 12 hours
• Oliguric renal failure within 24 hours (cats) to 72 hours (dogs)
• Hypocalcaemia, high anion gap acidosis, azotaemia, oxaluria, hyperglycaemia, hyperkalaemia, hyperphosphataemia
• Central nervous signs
• Death
Diagnosis – ETHYLENE GLYCOL TOXICITY

Ethylene glycol → Glycoaldehyde (CNS depression)
  ↓
Formic acid ← Glycolic acid → Oxalic acid
  ↓
Glycine
  ↓
Calcium oxalate
  ↓
Crystalluria and renal mineralisation → renal failure

High anion gap metabolic acidosis
Specific Therapy

- Ethylene glycol metabolised by alcohol dehydrogenase
  - can give ethanol to compete for metabolism
    - Not if renal failure present
  - 4-methylpyrazole (Antizol)
NSAIDs

- Ibuprofen common
- Rapidly absorbed
- Renal /Gastrointestinal effects within 2-12 hours
  - Nausea, Vomiting, gastrointestinal haemorrhage
  - Abdominal pain
  - Anorexia
  - Weakness/Ataxia
  - Acute renal failure (can be delayed up to 5 days)
Ibuprofen
Phospholipids in cell membranes

Phospholipase

Arachidonic acid

COX

NSAIDS
Prostaglandins

LOX
Leukotrienes

NSAIDS
Therapy:

• Gastric decontamination
• Gastric protectants for 14 days
  – Antacids e.g. ranitidine or Proton-pump blocker (omeprazole)
  – Sucralfate
• Anti-emetics
• Misoprostol
Supportive care

• Fluid therapy
• Monitor urine output
• Monitor electrolytes, blood gases and renal function
• Aggressive treatment for acute renal failure if necessary
Paracetamol

• Metabolised in liver by glucuronidation, sulphation or oxidation
  – Glucuronide pathway lacking in cats

• Metabolite *N*-acetyl-*p*-benzoquinone causes
  – severe oxidative stress to hepatocytes and rbc's → MetHb and Heinz body formation

• Particularly dangerous in cats
  – Oxidation of haemoglobin to methaemoglobin

• Main effect in dogs is hepatocyte damage
Signs

- Vomiting, depression, weakness
- Dyspnoea, facial oedema, muddy mucous membranes, tachycardia, tachypnoea (cats), hypothermia
  - Hours to develop signs
- Jaundice (dogs) – liver damage may take 1-5 days to develop
Therapy:

• Gastric decontamination
• N-acetylcysteine – provide cysteine for glutathione synthesis
  – Give orally or i/v
• Manage methaemoglobinemia with vitamin C, sodium sulphate and methylene blue as needed
• Oxygen
• Supportive care e.g. blood transfusion
Chocolate toxicity

- Theobromine
  - Methylxanthine
- Increased muscle activity
- CNS stimulation
- Signs within 4 hrs

- Vomiting
- Abdominal pain
- Hypersalivation
- PU/PD
- Hyperexcitable
- Tachycardia
- Ataxia
- Mild hypotension
- Muscle rigidity, seizures, tachypnoea, hyperthermia, cyanosis, arrhythmia, renal dysfunction
Chocolate toxicity calculator

- VIN.com
  - Vin mobile calculators

- Android and Apple apps
Chocolate poisoning - management

- Gastric decontamination/anti-emetics as appropriate
- Convulsions – diazepam
  - barbiturates or propofol may be necessary
- Treat arrhythmias if necessary
  - Beta blockers
- Supportive
  - Fluids if necessary
  - Anti-emetic
  - Sedation?
  - Monitor temperature, hydration, ECG and renal function
- Consider bladder lavage
Cannabis

• Most common plant poisoning enquiry in dogs
• Ingestion main route of toxicity
  – Occasionally seen in sniffer dogs
• Toxicity can be related to impurities
• Minimum lethal dose
  – 3mg/kg dog
• Majority intoxication have clinical signs
  – No reported fatalities
Clinical signs
Cannabis

- Seen within 1-3 hours, recovery 24-72 hours
- Weakness, ataxia, lethargy
- Dilated pupils
- Nystagmus
- Photophobia
- Tachycardia
- Urinary/faecal incontinence
- Hyperaesthesia & heightened senses
- Behavioural changes e.g. aggression, barking
- Later
  - depressed /stuporous
  - bradycardia
Sometimes

- Dogs can have
  - Heightened sense smell & hearing
- Dogs can obsessively investigate
  - Sights, sounds odours
Treatment
Cannabis

• Gastrointestinal decontamination
  – Emesis if < 1 hour post ingestion
  – Repeated administration activated charcoal q 4hrs
    • THC undergoes enterohepatic recirculation

• General supportive therapy
  – Quiet, dark environment
  – Maintain normothermia
  – IV fluids if hypotensive

• Sedation e.g. diazepam if very agitated