Rodenticide toxicity

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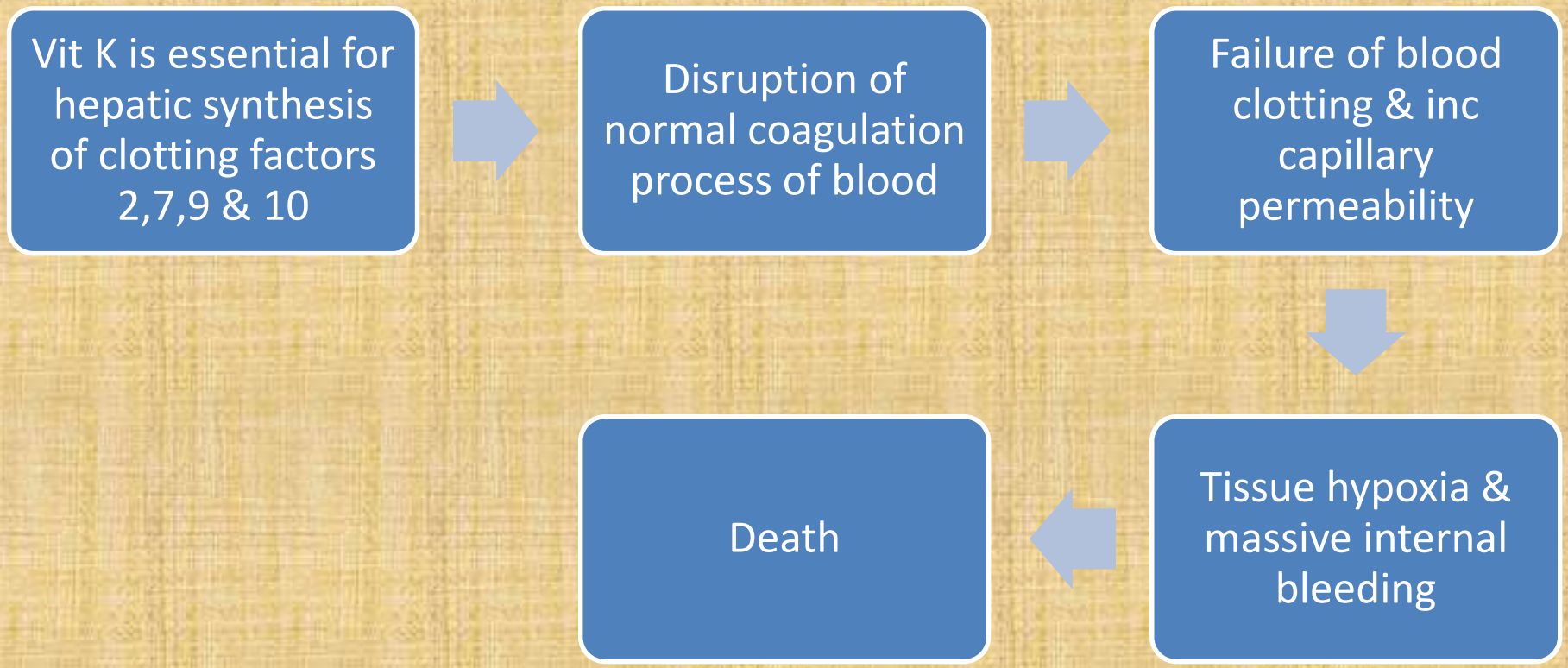
ANTI COAGULANT RODENTICIDES

- 1st generation e.g warfarin,pindone
- Intermediate e.g chlorophalicone
- 2nd generation e.g brodifacoum

MOA

- Direct capillary damage



Clinical signs

- Manifestation of hemorrhage including anemia, epistaxis
- Ataxia,
- Colic
- Weakness
- Polpnea
- Depression & anorexia even before bleeding

Lesions

- Hemorrhage in mm & body orifices
- s/c bleeding
- Blood fails to clot
- Hepatic necrosis

Dx

- On the basis of history
- Increased activated coagulation time of blood

Rx

- Vit K1 (phytonadione)@0.25-5 mg/kg s/c
- Menaphthone @ 0.5- 2 mg/kg i/m
- Fresh frozen plasma @ 9ml/kg i/v
- Whole blood @ 20 ml/kg i/v
- Glucose and saline therapy
- Supplemental oxygen
- Oxalic acid derivatives

ANTU

- Increased permeability of lung capillaries
- Massive pulmonary edema & pleural effusion
- Animal drowns in its own fluid

General considerations

- Abandoned rodenticide
- Ruminants are resistant
- Food increases chances of toxicity

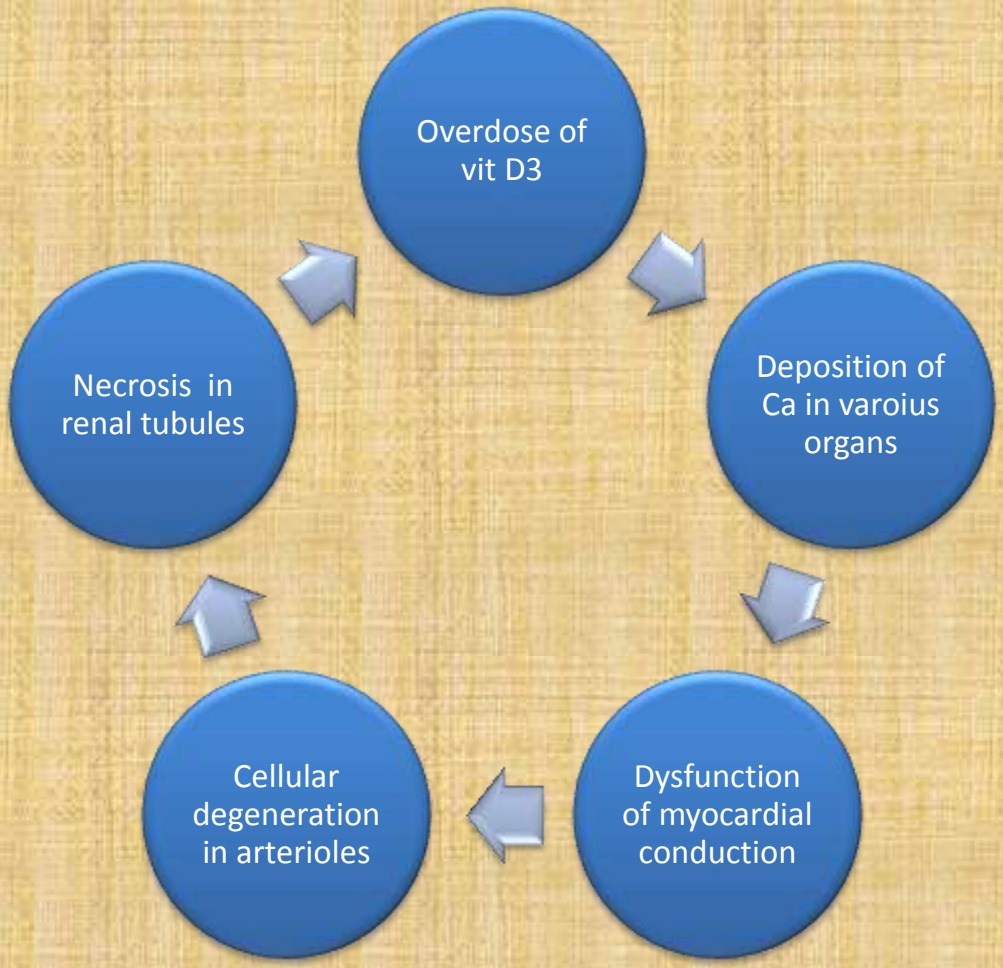
Clinical signs

- Vomition
- Labored breathing & cyanosis
- Incoordination & cyanosis
- Foam from nostrils & mouth
- Coma & terminal clonic convulsions
- Death within 2 hrs

Treatment

- Emetics like apomorphine & gastric lavage if respiratory distress is not evident
- Agents providing sulfahydryl groups eg thiosulphate
- Positive pressure O2 therapy
- Osmotic diuretic eg mannitol
- Atropine @ 0.02 – 0.25 mg/kg may relieve P. edema

Cholecaciferol



Clinical signs

- Develop within 18 – 36 hr of ingestion
- Depression & anorexia
- Polydipsia & polyuria
- Vomition with blood
- Hemorrhagic diarrhea
- GI smooth M excitability decrease results into constipation

Treatment

- Gastric evacuation followed by administration of activated charcoal@ 2-8 g/kg in water slurry
- Prednisolone @ 1-2 mg /kg
- Fluid therapy with NSS
- Furosemide@5mg/kg i/v followed by 3mg/kg tid
- Calcitonin @4-10 IU/kg s/c to reduce serum calcium levels

Strychnine

- Alkaloid from seeds of nux vomica
- Rapidly absorbed and metabolised to strychnine N oxide
- Used as malicious poison to kill stray dogs
- Competitive antagonist of inhibitory neurotransmitter glycine

Mechanism of action

Acts at glycine receptors in spinal cord & medulla

Lowers threshold for stimulation of spinal reflexes

Extensor rigidity & titanic convulsions

Clinical features

- Restlessness , muscle twitching & stiffness of neck
- Intermittent convulsions & relaxation
- Advance signs ar etonic seizures & opisthonous(saw horse posture)
- Spasm of respiratory muscles or paralysis of respiratory centre lead to death

Lesions

- Cyanosis due to asphyxia
- Rapid onset of rigor mortis

Diagnosis

- History and clinical signs
- Detection of strychnine(detected upto long time after death)

Treatment

- Phenobarbitone or chloral hydrate
- Gastric lavage with KmNO4 or tannins
- Acidification of urine & diuresis to promote excretion
- Muscle relaxants like glyceral guaiacolate@ 110mg/kg or methacarbamol @ 150mg/kg followed by 90mg/kg
- Keep animal in warm and quiet environment
- Ketamine and morphine ar econtradicted

Red squill

- One of the safest rodenticide with selective action on rats and mice
- It induces vomition in dogs and cats
- Unpalatable for domestic animals

Mechanism of action

- In small doses – induces convulsions
- In large doses – cardiac arrest

Clinical features

- Vomition
- Ataxia & paralysis
- Alternate period of CNS stimulation & depression
- Bradycardia, aarhythmia & cardiac arrest
- Death occur wuthin 3 days

Lesions

- Gastroenteritis with congestion , swelling & ulceration of mucosa
- Congestion of abdominal & thoarcic organs
- Degenerative changes in kidney , lungs & myocardium

Treatment

- Quinine sulphate or phenytoin @ 35mg/kg to suppress arrhythmia
- Atropine sulphate s/c at 8 hr interval to prevent cardiac arrest
- Other supportive measures

Bromethalin

- Rapid oral absorption
- Metabolized by MFO to more toxic metabolite

  desmethyl bromethalin
- Excreted through bile

Mechanism of action

- Uncouples oxidative phosphorylation
- Loss of Na K ATPase & inhibition of Na pump
- Intracellular edema, swelling, & degeneration of cells
- Also induces cerebral lipid peroxidation
- Increased CSF pressure
- Dec impulse conduction, paralysis & death

Clinical features

Acute toxicity

- Hyper excitability & seizures
- Muscle tremors & hind limb hyperreflexia
- Pyrexia & death within 10 hrs

Sub acute toxicity

- Tremors & ataxia
- Depression, vomition & lateral recumbency
- Effects reversible on termination of exposure

Lesions

- Cerebral edema
- Spongy degeneration of optic nerve & posterior paralysis

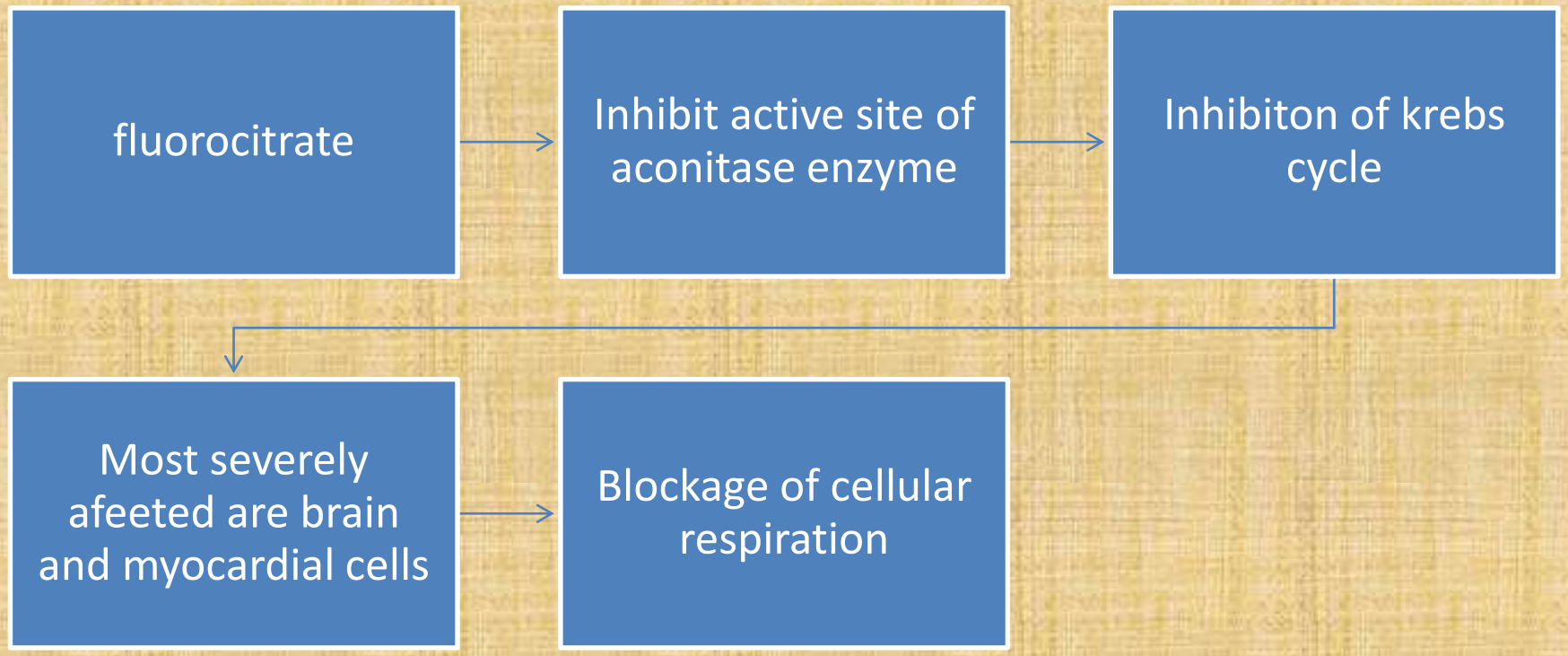
Treatment

- Symtomatic and supportive
- Mannitol @ 250mg/kg i/v 6 hrly to reduce cerebral edema
- Corticosteroids
- Ora fluiod therapy
- Diazepam or phenobarbitone to control seizures

Fluoroacetates

- Most potent rodenticide
- Not specific for rodents
- Preparations are made black to distinguish from other substances

Mechanism of action



Clinical features

- Dogs – exhibit signs of CNS excitation
- Horse, cattle, sheep, goat – cardiac abnormalities
- Cats – combination of both

Lesions

- Severe enteritis
- Cyanosis
- Dark blood
- Organ congestion
- Hemmorhage in heart
- Pulmonary changes

Diagnosis

- History symptoms and lesions
- Detection of excess citrate in kidney tissue

Treatment

- Glycerol monoaceate @ 0.5mg/kg i/m in NSS
- Ethanol 50% or acetic acid 5% @8ml/kg orally
- Supportive treatment for convulsions & arrhythmia
- Emetics are contradicted

Thallium sulphate

- Dec activity of –SH enzymes – succinic dehyrogenase &

  MAO

- Toxicity is due to inhibition of S containing macromolecules

- It also competes with potassium for active transport

Symptoms

- Gastroenteritis & abdominal pain
- Dyspnea & fever
- Conjuctivitis & gingivitis
- Tremors and seizures
- Hyper keratosis & dermatitis

lesions

- Ulceration and hemmorhagic gastroenteritis
- Inflammation of resp mucosa
- Degenerative changes in heart , liver and kidney

Treatment

- Emetics & gastric lavage with 1% sodium iodide solution
- 10 % iodide solution i/v
- Fluid therapy
- Sedatives
- Sod thosulphate/diphenyl thiocarbazone

Zinc phosphide

- Non selective that is why hazardous
- More toxic after meals
- Due to increased acid production

Mechanism of action

- Zinc phosphide + water & acid in stomach
- Production of phosphine gas
- Direct irritant to gut
- Inhibition of cyt c oxidase leads to tissue hypoxia

Clinical features

- Dyspnea
- Abdominal pain
- Vomition of blood
- Ataxia
- Prostation
- Convulsions and coma
- Colic in horses
- Bloat in cattle

Lesions

- Smell of phosphine from stomach contents
- Pulmonary congestion & edema
- Pleural effusions & sub pleural hemorrhage
- Congestion of liver and kidneys
- Gastroenteritis

Diagnosis

- History and clinical signs
- Detection of Zn phosphide in stomach contents
- Analysis of phosphine in tissues

Treatment

- Gastric lavage with 5% sod bicarb to neutralize stomach acidity
- Fluid therapy with cal gluconate or sod. Lactate to overcome acidosis
- 5% glucose i/v to prevent liver and kidney damage
- Demulsants & protectants to treat gastroenteritis
- Barbiturates to control convulsions
- Artificial respiration
- Activated charcoal

Thanks ☺