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Lecture title: Poisoning by Aflatoxins (Aflatoxicosis)

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Summary:

•Aflatoxins (AFs) are metabolites produced by fungi growing on spoiled feeds. Elevated relative humidity (95%–97%) levels and warm temperatures (25°C–37°C) are associated with fungal growth.

•Eighteen AFs have been isolated with AFB1, AFB2, AFG1, AFG2, and the second-generation metabolites M1 and M2 are the most widely studied.

•AFB1 is generally recognized as one of the most potent hepatic carcinogens in the world.

•Aspergillus flavus, A. nomius, and A. parasiticus are the most commonly recognized species that produce AFs. Other less common AF-producing species include A. niger, A. ruber, A. wentii, Penicillium citrinum, and P. frequentans.

•In sheep, the oral LD50 is 5 mg/kg; a dose rate of 4 mg/ kg is associated with death at 15 to 18 hours caused by acute hepatic insufficiency.

•The estimated LD50 for AFB1 in calves is estimated to be 0.5 to 1.5 mg/kg.

•The oral LD50 of AFB1 for most species is in the range of 0.03 to 18 mg/kg.

Epidemiology

*Occurrence

•Aflatoxicosis has been reported in most countries and on many spoiled feeds, especially:

-harvested peanuts -peanuts in shells - cottonseed meal - sorghum grain

-corn - moldy bread - green chop sorghum

•All animal species are susceptible, but outbreaks occur mostly in pigs, sheep, and cattle.

•beef and dairy cattle are more susceptible than sheep or horses.

•Young animals of any species are more susceptible than adults.

•Because the toxin is excreted in cows' milk it has public health importance.

Pathogenesis

•AFs absorbed from the gastrointestinal tract, entering the portal blood system in a short period and concentrating in the liver.

•Cytochrome P450 is actively involved with the transformation of AFB1 to the toxic metabolite AFB1-8-9-epoxide.

•The toxic effects of AFs are most pronounced in the liver where the metabolism of carbohydrates, lipids, and proteins is impaired.

Clinical Findings

***Cattle**

1- blindness, walking in circles.

2- ear twitching, teeth grinding, frothing at the mouth.

3- photosensitive dermatitis and keratoconjunctivitis.

4-diarrhea.

5-abortion, and anal prolapse.

6- terminal convulsions. and dead within 48 hours.

***Horses**

Clinical signs are nonspecific but include :

depression, anorexia, fever, tremors, ataxia, icterus, and hemorrhage.

Clinical pathology

1-Acute elevations in serum hepatic enzymes with γ-glutamyl transpeptidase, AST, and SDH.

2- Elevations in prothrombin time and serum bilirubin .

3-Laboratory assay methods include chromatography/mass spectrometry and immunoassays for Analysis and quantitation of AFs in feed materials, urine, blood, and tissues.

Necropsy Findings

In all species are those of hepatosis, multiple foci of necrosis and fibrosis.

Differential diagnosis list:

• Cyanobacteria (blue-green algae) toxicosis

• Fascioliasis

• Primary hepatic disease (neoplasia, bile duct obstruction)

Treatment

Symptomatic treatment of hepatic insufficiency