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The Differential Diagnosis of Three Common Swine Problems

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Swine exhibiting nervous disorders or a weak, incoordinated posterior gait can often present a problem in diagnosis. This review will be based mainly on clinical signs and post-mortem lesions in the differential diagnosis of organic arsenic toxicity, water deprivation syndrome, and edema disease. Several conditions may

give similar clinical symptoms but these three may give the most problem in differential diagnosis.

ORGANIC ARSENICAL TOXICITY

Organic arsenicals are used mainly in swine as feed additives, treatment for vibriotic dysentery, other enteritis conditions and blood parasite problems. The two main forms that are used are

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arsenilic acid and the phenylarsonic acids. Toxicity is usually associated with one of the following circumstances:

- 1) Intentional excessive feed levels
- 2) Mistaken feed calculations
- 3) Prolonged and excessive administration of arsenicals in combination with other drugs

The recommended long term feeding level is 100 ppm and used at that level usually causes no problems. When used therapeutically in the feed, levels of from 250 ppm to 400 ppm are fed for 4-5 days. Levels of 450 ppm of arsenilic acid and up, fed daily in the ration will produce symptoms of acute poisoning within a week according to evidence.

It has been noted that toxicity may be increased in animals with gastrointestinal problems. Also organic arsenicals may be more toxic when there is limited water supply. Clinical signs are mainly referable to a motor paralysis of the limbs.

In the acute form the following signs may be seen:

- 1) Loss of locomotion control
- 2) Ataxia may be present
- 3) Blindness may be seen with arsenilic acid, but not with 3-nitro
- 4) Clinical signs are reversible up to a point. Removing excess arsenical results in recovery

Chronic Form

- 1) Weakness of limbs, knuckling of pastern, exaggerated stepping movements, reluctance to rise
- 2) Complete paralysis or severe incoordination, such as walking on knees
- 3) The pigs are aware of surroundings, will continue to eat and drink if provided
- 4) Convulsions are not seen in organic arsenical toxicity

Post-mortem lesions are minimal grossly. Microscopically, demyelinations and gliosis are seen in peripheral nerves. History of excessive arsenical feeding is a main factor in this diagnosis.

WATER DEPRIVATION SYNDROME

Pigs that have had a lack of water may develop a characteristic clinical syndrome

formerly called salt poisoning. Recent evidence shows that the lack of water is the most important factor and not excessive levels of salt. This syndrome has occurred in swine on a ration having only 0.5% salt in it. This condition is actually a sodium ion toxicity only because of the low water intake. This can occur with any sodium ion compound, not just NaCl. With the lack of water, sodium ion concentrates in the brain, forming an osmotic gradient to draw water from the circulatory system with the result being cerebral edema. There is also evidence that indicates sodium ion inhibits anaerobic glycolysis in the brain affecting cerebral function.

Clinical Signs

- 1) Aimless wandering
- 2) Head pushing
- 3) Circling
- 4) Intermittent epileptiform seizures
 - a) Twitching of facial muscles
 - b) Cervical muscle contractions
 - c) Opisthotonus
 - d) Paddling of front limbs
 - e) Twitching of facial contractions
- 5) Death may occur anytime in the course

In the diagnosis of the syndrome, serum and cerebral spinal fluid sodium levels are often helpful. Cerebral spinal fluid sodium levels of 160 mEq./liter or higher and serum sodium levels of 155 mEq./liter or higher is a finding in the syndrome. Changes in the brain and meninges microscopically are considered diagnostic. The lesion is an eosinophilic meningioencephalitis with quite marked edema and malacia often.

Treatment of severely affected pigs is often not very successful but diuretics may be used. The most important factor is to give water slowly to the rest of the pigs in small amounts, otherwise water may precipitate the rest of the herd into the syndrome.

EDEMA DISEASE

Edema disease of swine is also known as gut edema, gastric edema, enterotoxemia, and edema of the bowel. The disease

is an acute, non-contagious condition of young pigs that is often fatal. With the great increase in the pig population, with its accompanying overcrowding and the rushing of pigs through to slaughter weights as early as possible, this condition has once more come into prominence and in a more severe form than was the case years ago.

The etiology of edema disease is believed to be due to endotoxins produced by hemolytic *E. coli*. The disease is most common in young pigs in the 8 to 12 weeks age group. Most often it is seen shortly after weaning, however it can occur in very young pigs and also in older swine up to market weight.

Predisposing factors seem to direct toward nutrition and stress. Usually the fastest-growing, thrifty pigs are affected the most, being on a good plane of nutrition. Stresses which may predispose are weaning, recent transportation, change from starter ration to a grower, and recent vaccinations.

Clinical Findings

- 1) Often find some pigs dead with no clinical signs
- 2) Incoordination of the hind limbs—swaying often from side to side
- 3) An attack of diarrhea may be seen in some of the pigs often before clinical signs of edema disease
- 4) Pigs are usually aware of their surroundings
- 5) Convulsions, which usually are not intermittent in nature
- 6) Edema of eyelids and conjunctiva
- 7) The most thrifty pigs are usually affected

Post-Mortem Lesions

- 1) Edema of eyelids
- 2) Edema in the wall of the stomach
- 3) Ansa spiralis of the colon is often quite edematous in pigs that have died of edema disease
- 4) Often very few post-mortem lesions are seen

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Breaks, Good and Bad: The Inner Life of Research

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Once in a while, if you are lucky, you will get to read a technical research article which has some soul. The rest are corpses. Corpses are fine for their purpose, but they never reveal the inner life, the dynamic mechanism. An outstanding exception is the recent best seller in science, *The*

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Double Helix; which details the very human development of a very important theoretical model (7). My own experience has nothing so singularly grand to offer, but throughout my work there has occurred a string of breaks, both good and bad, which seems to comprise a certain essence of research. Rather than making an organized scholarly report, I will try to tell it like it happened.