

A Report of an Outbreak of Acute Enteritis in Swine with Hepatic Lesions

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INTRODUCTION: This was a collaborative investigation between SMRL; the Faculty of Veterinary Medicine, Kasetsart University; and the Tub Kwang Animal Husbandry Section, Kasetsart University.

OBJECTIVE: To determine the etiology of an epizootic of acute enteritis in swine with pre-existing hepatic lesions.

BACKGROUND: The Tub Kwang pig farm located in Saraburi province maintains approximately 80 adult breeding sows and 50 young adult swine (40–50 Kg.). The animals are housed in covered open barns with concrete floors. The basic diet consists of a locally grown grain mixture with added vitamin, mineral and protein supplements. Fresh drinking water is supplied from automatic waterers. In early November 1972 a large number of pigs became sick. Within two weeks 65 animals were sick and 21 died. (Table 1). Clinical signs included a slightly increased temperature, icterus, loss of appetite, depression, lameness, and erythematous and edematous skin lesions.

METHODS: A complete necropsy was performed on three dead and four sick animals, and tissues were taken for complete histopathologic examination. Specimens of heart blood, lung, stool, brain, and intestine were cultured for both aerobic and anaerobic bacteria. Virus isolation was attempted in tissue culture from stool, liver, kidney, lung, spleen, heart, and brain. Weanling mice were inoculated with serum and with suspensions of liver, kidney, lung, heart, spleen, and brain and observed for 3 weeks. Specimens of drinking water and food were submitted for bacteriologic, mycologic, and toxicologic evaluation.

RESULTS:

Virology: No viruses were isolated either in tissue culture or by animal inoculation.

Gross Pathology: The skin, mucous membranes and other tissues were markedly icteric on all animals examined. The mesenteric lymph nodes were enlarged and diffusely hemorrhagic. Lung lesions were observed in all the animals, consisting of reddened and consolidated areas with sticky exudate present in the bronchi. The pericardial sac was edematous around the ventricular area, and in one animal 10 ml of clear fluid was present in the pericardial sac. Gastrointestinal lesions were present in all animals, and varied from one animal showing only redness of about 30 cm of the serosal surface of the small intestine with normal stool, to another showing gastritis with reddened areas of gastric mucosa and bloody mucus filling over 6 meters of the small intestine. Generalized subcutaneous and intramuscular hemorrhage was found, but is attributed largely to supportive therapy with subcutaneous and intramuscular injections of fluids. The livers of all animals were completely yellow and fatty. The livers were normal in size and shape, but of a slightly fibrous consistency in two of the animals.

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Microscopic Pathology: Microscopic pathologic examination revealed moderate to severe chronic diffuse hepatitis, diffuse acute hemorrhagic enteritis, and severe acute diffuse hemorrhagic lymphadenitis in all animals examined. All animals showed lung lesions varying from acute bronchiolitis to bronchopneumonia and chronic interstitial pneumonia. Two animals showed colitis, and one animal showed acute splenitis and myositis.

Bacteriology: In the sick animals sacrificed for necropsy, examination of brain, lung and stool showed no pathogens or abnormal flora. The anaerobic bacteria *Clostridium perfringens* was isolated from fresh heart blood of three of the four animals. In the three dead animals examined, *Pasteurella multocida* was isolated from several tissues. No pathogenic bacteria were isolated from the drinking water.

Feed Analysis:

No pathogenic bacteria were isolated from the food, but a variety of fungal organisms were identified, including *Rhizopus sp.*, *Candida sp.*, *Penicillium sp.*, *Mucor sp.*, and *Aspergillus sp.* The chemical examination revealed the presence of chlorinated hydrocarbon compounds in each item of animal feed (Table 2) and in samples of pig tissues taken at necropsy.

Discussion:

The hepatic lesions appear to have developed before the other lesions observed. These liver lesions are consistent with those observed in a wide variety of toxic conditions, and could have been caused by chronic exposure to chlorinated hydrocarbons both in the feed and by frequent applications of chlordane spray in the treatment of sarcoptic mange. The toxins produced by *Aspergillus sp.* have also been reported to cause chronic liver lesions like those described.

The acute enteric lesions observed can be attributed to bacterial infection. *Clostridium perfringens* has been reported to cause an infectious necrotic enteritis in baby pigs, either as a primary entity or secondary to other disease conditions. *Pasteurella multocida* rarely causes a primary disease in swine, but often develops as a disease secondary to other conditions which cause debilitation and decreased resistance. It occurs as a chronic or sub-acute disease of the lungs, and/or as an acute septicemia, but it can often be isolated from the lungs of swine that show no disease or tissue alteration. The simultaneous existence of pasteurellosis and certain other infectious diseases is not uncommon. In one study, *Pasteurella multocida* was isolated in pure culture from 44% of 314 swine showing some lung pathology but no symptoms of disease.

Considering the age of the pigs in this outbreak, the gross and microscopic lesions, and the response to treatment, the illness and fatalities were probably due to the intestinal bacteria. It appears that the overall problem was one of chronic liver toxicity from chemical and/or biological products producing debilitating hepatic lesions which in themselves were not severe enough to produce overt disease, but were contributory in producing an environment for existing opportunistic organisms to establish. *Pasteurella multocida* and *Clostridium perfringens*, present in the food and environment, but needing a stressed animal in which to establish themselves, produced the severe acute hemorrhagic necrotizing enteritis and bronchopneumonia which resulted in death for many of the animals. Suckling pigs under one month of age were refractory to the condition described here. They were not exposed to the toxic products in the feed, and they were receiving substantial amounts of maternal antibody through the milk to provide protection against the bacteria involved.

Immediately after the outbreak, animals were medicated with oxytetracycline or penicillin-streptomycin, and the food supply was changed. There have been no further cases of illness or death since that time. Many animals which appeared clinically normal were examined at slaughter and were found to have yellow livers similar to the animals described here.

Table 1.
Swine Epizootic at Tub Kwang

Age	Total Animals	Sick Animals	Morbidity	Deaths	Mortality
<1 month	100	0	0%	0	0%
1-3 months	200	40	20%	15	37.5%
3-6 months	50	20	40%	3	15%
Adult females	<u>80</u>	<u>5</u>	<u>6%</u>	<u>3</u>	<u>60%</u>
Total	<u>430</u>	<u>65</u>	<u>15%</u>	<u>21</u>	<u>32.3%</u>

Table 2.
Chemical Analysis of Feed

Feed	Chemicals Detected
Corn Meal	DDT 0.5 ppm
Soybean Cake	DDT 0.2 ppm
Fish Meal	Eldrin 6.25 ppm
Meat Scraps	Eldrin 6.43 ppm
Ground Feed	DDT 0.23 ppm