An unusual presentation of suspected oedema disease of swine in Kenya

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ABSTRACT

From a group of 11 recently weaned pigs, 4 were reported to be sick. Clinical examination of the sick pigs revealed marked dyspnoea, bluish-red discolouration of the skin, incoordination and difficulty in walking. Bacteriological examination of the gut contents of 2 pigs that had died earlier yielded pure cultures of haemolytic *Escherichia coli. Post mortem* examination of the remaining 2 pigs that died subsequently revealed progressive pulmonary collapse. One of these also showed subcutaneous oedema of the head and marked oedema of the mesentery of the spiral colon and oedema of the brain. Microscopically there was pulmonary alveolar collapse and degenerative changes in the liver. On the basis of the clinical signs, isolation of haemolytic *E. coli* and the *post mortem* findings, a diagnosis of oedema disease was made.

Key words: *Escherichia coli,* oedema disease, pigs.

Mulei C M, Ngatia T A **An unusual presentation of suspected oedema disease of swine in Kenya**. *Journal of the South African Veterinary Association* (1999) 70(2) 100–101 (En.). Department of Clinical Studies, Faculty of Veterinary Medicine, University of Nairobi, PO Box 29053, Nairobi, Kenya.

INTRODUCTION

Oedema disease of swine is an enterotoxaemia associated with a large number of predominantly haemolytic *Escherichia coli* in the intestines of affected pigs[2,7]. The haemolytic *E.coli* produce Shiga-like toxin (SLT IIV), which causes damage to the small arteries and arterioles, especially in the gastric and colonic submucosa and the brain, and subsequently oedema formation[3,5–7].

The disease occurs most commonly in rapidly growing, recently weaned (5–14 days after weaning) pigs, especially those in good bodily condition, or after a change in feed[2,6,9]. The major clinical signs observed in sick pigs include dullness, incoordination, difficulty in getting up and walking, and oedema of the head, eyelids and conjunctiva[2,6,9,10].

This communication reports unusual cases of suspected oedema disease characterised clinically by marked dyspnoea and bluish-red discoloration of the skin and at necropsy by pulmonary collapse.

CASE HISTORY

Four sick, recently weaned pigs were presented to the Veterinary Clinic, University of Nairobi. The affected pigs were in good bodily condition but were showing marked dyspnoea, incoordination, and difficulty in walking and had a slightly elevated body temperature (40 °C). Bacteriological examination of the colonic and rectal content of 2 pigs that had died earlier with similar symptoms yielded pure cultures of haemolytic *E.coli.* It was not possible to type the organism. Based on the clinical findings and the laboratory report, a presumed clinical diagnosis of oedema disease and pneumonia was made.

The sick (4) and the healthy (7) in-contact pigs (in the same pen) were treated with an intramuscular injection of a combination of procaine penicillin G (200 mg) and dihydrostreptomycin sulphate (250 mg) (Combiotic®, Pfisher Kenya Ltd) per kilogram body weight and oral administration of furazolidone (20 mg/kg body weight) for 4 days. Despite treatment, the 4 sick pigs died, but none of the healthy in-contact pigs became sick or died.

Two of the 4 dead pigs were sent (at different times) for *post mortem* examination to the Department of Veterinary Pathology and Microbiology, University of Nairobi. At necropsy a thorough examination was conducted, tissues for histopathology were taken from various organs and fixed in 10 % formalin. After proper fixation, the tissues were routinely processed, sectioned at a thickness of 6 µ and stained with haematoxylin and eosin (HE).

POST MORTEM FINDINGS

Both the pigs were in good bodily condition but showed collapsed dark red lungs. In 1 pig the collapse was total but in the other pig, small portions of the apical lobes were unaffected. One of the pigs also revealed patchy congestion of the gastric mucosa, marked oedema of the mesentery of the spiral colon, subcutaneous oedema of the head and congestion of the brain.

Microscopic examination revealed alveolar and bronchiolar collapse, varying from slight to complete. There was no exudate in the alveoli or the bronchioles in any of the collapsed areas.

The liver revealed diffuse mild granular and vacuolar hepatocytic degeneration. In the brain cerebral oedema was observed, while in the kidneys degenerative changes were observed in the tubular epithelium and the glomeruli. No significant lesions were observed in the other organs examined.

DISCUSSION

The 4 sick pigs showed marked dyspnoea, bluish-red discolouration of the skin, incoordination and difficulty in walking. Except for dyspnoea, the other features, together with the occurrence of the disease soon after weaning, were highly suggestive of oedema disease of swine as described by various authors[2,6,9,10]. The isolation of pure cultures of *E.coli,* although not typed, from the colonic and rectal content of 2 pigs that died first, supported the diagnosis of oedema disease of swine[2,7]. At necropsy, the significant lesions observed that supported a diagnosis of oedema disease included oedema of the spiral colon and subcutaneous oedema of the head in 1 of the pigs[1,4,6–10].

The immediate cause of death of the 4 pigs was respiratory failure. This was attributed to pulmonary collapse, which was the cause of the marked dyspnoea observed clinically. This has not been described in association with oedema disease of swine. Instead, pulmonary oedema has been associated with some cases of this disease[1,4,6,8]. Whether the pulmonary atelectasis was a result of this disease or part of another disease occurring concurrently could not be ascertained.

The pulmonary atelectasis observed in the pigs in this study was not associated with any exudative blockage of the air

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Received: August 1998. Accepted: March 1999.

passages, as these were devoid of any substantial exudate. Since the cause of atelectasis was not established, its pathogenesis can only be surmised. On the basis of clinical signs and morphological observation, it appears that the atelectasis developed progressively. This was supported by the microscopic examination of the lung tissue, which revealed areas in different stages of development of atelectasis. The lung collapse appeared to be of central nervous system origin. It is possible that the brain oedema affected the respiratory centre, with subsequent pulmonary atelectasis and hence the observed dyspnoea.

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