


Outbreak of edema disease in piglets in the Sertão da Paraíba

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ABSTRACT

Edema disease is a worldwide toxi-infection, which mainly affects recently weaned piglets, and is caused by verotoxigenic strains of *Escherichia coli* bacteria. The epidemiological, clinical and anatomopathological aspects of an outbreak of edema disease in pigs in the hinterland of Paraíba are described. Piglets of both sexes were affected in the post-weaning period. The clinical course was acute, characterized by neurological manifestations and edema. At necropsy, edema of the eyelid, stomach wall, mesocolon and ascites were observed. Histopathology of the nervous system showed an increase in perivascular spaces associated with edema. There were also occasional neurons located in the frontal, temporal and occipital cortices and basal ganglia with cytoplasmic hypereosinophilia and nuclear pyknosis. The diagnosis was established through epidemiology, clinical signs, anatomopathological findings, and microbiological isolation of the agent. Some epidemiological factors are capable of predisposing animals to *E. coli* infections. Thus, it is necessary to implement preventive measures in order to minimize risk factors that are linked to adequate nutritional and sanitary management.

Keywords: Swine disease, Toxi-infection, *E. coli*.

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INTRODUCTION

Edema disease is a worldwide toxi-infection, which mainly affects recently weaned piglets, and is caused by burial-hemorrhagic (EHEC) or verotoxigenic (VTEC) strains of *Escherichia coli* bacteria, which produce *Shiga-like* toxin 2e (verotoxin 2e) and fimbriae F18 (Ribeiro *et al.*, 2016).

Most affected pigs have an acute clinical course, with death within 24 hours (Borowski *et al.*, 2002). Animals that recover usually become refuse and the recurrence of the disease is frequently described in herds (Mello; Lot 2009).

The presumptive diagnosis of the disease can be established based on epidemiological, clinical and necropsy data of the animals. Confirmation of the diagnosis is established through the association of the isolation of the bacterium with the characterization of the gene responsible for the production of the toxin, and the anatomopathological findings (Filippsen *et al.*, 2001). The objective of this work is to describe the main epidemiological, clinical and anatomopathological aspects of an outbreak of edema disease in pigs in the hinterland of Paraíba.

MATERIAL AND METHODS

An outbreak of edema disease in pigs diagnosed at the Animal Pathology Laboratory (LPA) of the University Veterinary Hospital Prof. Ivon Macêdo Tabosa of the Federal University of Campina Grande, Patos Campus, Paraíba, was reviewed. In the clinical and necropsy protocols, information was collected regarding epidemiological data (age, sex, breed, litter size and origin of the animal), clinical signs and anatomopathological findings.

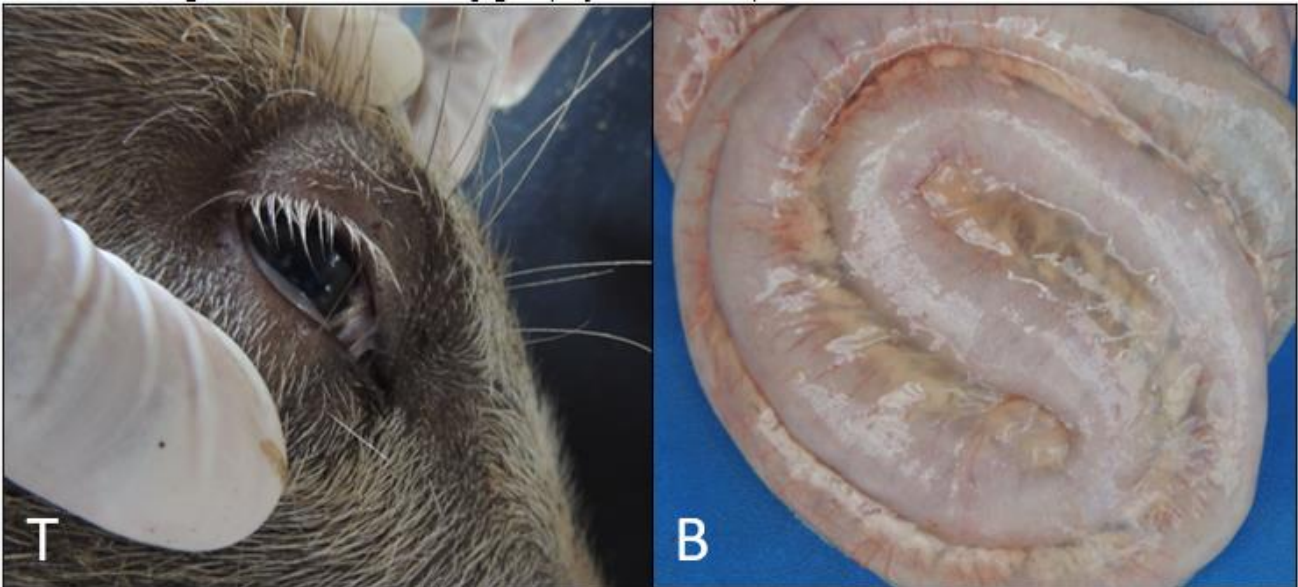
RESULTS AND DISCUSSION

On a property in the rural area of the city of Patos, Paraíba, 18 piglets of both sexes, approximately three months old, were kept in a semi-intensive regime and belonging to two different litters. The animals began to manifest apathy, tremors of intention, ataxia, difficulty in staying in station and lateral decubitus, and 14 animals died in a period of six days. All affected animals were in the post-weaning period. The food consisted of leftover food from restaurants and corn bran. One of the dead animals was sent for an autopsy.

Macroscopically, eyelid edema, ascites, diffuse mesocolon edema (Figure 1) and stomach wall edema were observed. Histopathology of the nervous system showed an increase in perivascular spaces associated with the presence of mildly eosinophilic, proteinaceous, and amorphous material (edema) (Figure 2). There were also occasional neurons located in the frontal, temporal and occipital cortices and basal ganglia with cytoplasmic hypereosinophilia and nuclear pycnosis. In the stomach, the submucosa is marked and diffuse distension due to edema associated with hypereosinophilia and

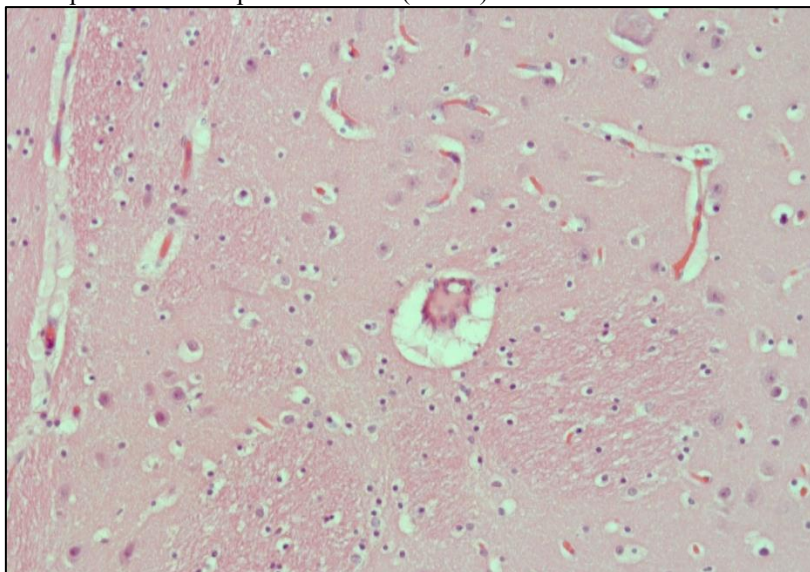
thickening of the media layer of the blood vessels (fibrinoid necrosis), with occasional thrombi in the vascular lumen.

Figure 1: Edema disease in pigs. A) Eyelid edema. B) Diffuse edema of the mesocolon.



Source: LPA-FOCG.

Figure 2: Edema disease in pigs. In the brain, there is an increase in perivascular spaces associated with the presence of mildly proteinaceous, eosinophilic and amorphous material (edema).



Source: LPA-FOCG.

Intestine fragments were submitted to culture and microbiological isolation using *Blood agar*, *MacConkey agar* and *Sabouraud dextrose agar* with chloramphenicol, incubated at 37°C for 24 hours in aerobiosis resulting in the growth of colonies compatible with *Escherichia coli*.

The diagnosis was established by epidemiological and clinical findings, and confirmed by anatomopathological examination and isolation of the agent. The reduction of lactogenic immunity and the increase in stressors related to changes in feeding and management (separation from the



mother, piglet mixing, change of environment), represent a critical time for the establishment of infections. This period was previously the one with the highest incidence of the disease (Brum *et al.*, 2013; Coelho *et al.*, 2017). Food and sanitary management measures were indicated to improve immunity and reduce cases. The main clinical manifestations presented consisted of neurological signs associated with edema. Neurological signs of ataxia, seizures, incoordination, and tremors, along with edema of the face and ventral region of the neck are described as features in the disease (Helke *et al.*, 2015; Santos and Alessi, 2016). The development of neurological alterations is related to circulatory disorders, mainly represented by edema and hypoxia (Fairbrother; Gyles, 2012).

CONCLUSION

The disease of edema has an important frequency, predominantly in the form of outbreaks in piglets in the post-weaning period. Neurological signs followed by facial edema in this period of life are suggestive of the disease. Some factors are capable of predisposing animals to infection by *E. coli*, thus, it is necessary to implement preventive measures in order to minimize risk factors such as sudden change of feed, supply of high-protein diets, piglet stress due to maternal separation, mixture of litters in the same stall, overcrowding, poorly performed disinfection in the facilities, absence of sanitary void between lots, temperature and humidity variations in the stalls.

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