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Acute Atypical Bovine Pneumonia Caused by Ascaris lumbricoides.

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ABSTRACT

A case of acute atypical pneumonia (bovine asthma, pulmonary emphysema, or pulmonary adenomatosis) occurred in a group of cross-bred beef type yearlings in late autumn. Clinical signs included a forced expiratory grunt, excessive salivation, ruminal stasis and, on auscultation over the lungs, pulmonary emphysema and oedema. The cattle had been brought in from a poor summer pasture and housed in a pig pen heavily contaminated with Ascaris lumbricoides eggs as it had contained unwormed feeder pigs all summer.

Fifteen out of seventeen head were affected ten days following housing and all within twenty-four hours. One steer of the group died and at necropsy fourth stage A. lumbricoides larvae were isolated from lungs showing profuse oedema and some emphysema. Histopathological examinations of the lungs showed a diffuse interstitial pneumonia. All remaining animals appeared clinically normal six days following the outbreak.

Introduction

Acute atypical pneumonia (bovine asthma, acute pulmonary emphysema or pulmonary adenomatosis) occurs in cattle of all ages over three months of age. It commonly occurs in autumn ten to twelve days after the cattle have been put on new pasture. **Dictyocaulus viviparus** [1], **Clostridium welchii**[2], phosphorus deficiency, calcium phosphorus imbalance in the soil, alkaline water and protein allergy have all been incriminated as possible aetiological agents for this condition.

The purpose of this paper is to report an outbreak of acute atypical pneumonia caused by the migration of **Ascaris lumbricoides** larvae in yearling cattle. A limited search of the literature has failed to reveal reports of this condition in naturally occurring cases.

Clinical History

A group of yearling cross-bred beef animals in only fair condition were transferred from poor summer pasture to housing late in November. The pen into which the cattle were placed, had, throughout the summer, contained unwormed feeder pigs and had not been cleaned out prior to admitting the cattle. Feed for the cattle consisted of poor quality hay fed on the ground.

On the tenth day following housing, fifteen of the seventeen yearlings, all within twenty-four hours, showed anorexia and acute respiratory distress. They were treated with penicillin-streptomycin intramuscularly on the first day of the outbreak, four ounces of sodium sulphamethazine intravenously on the second day, and three hundred cc. sulphabrom intravenously, one gram chloramphenicol and antihistamines intramuscularly on the third day, with no apparent clinical improvement.

On the third day of illness clinical examination of two of the affected animals revealed the following:

Steer I — Forced expiratory grunt; ruminal atony; pulse: 120 beats per min.; respirations: 60 per min.; temperature: 105 °F. On auscultation, the sounds typical of pulmonary oedema could be heard over the left lung and emphysema over the right lung.

Steer II — Forced expiratory grunt;

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excessive salivation; slow ruminal movements; mild moist coughing; temperature: 104.6 °F.; pulse: 100 per min.; respirations: 48 per min. No pathological lung sounds were audible on auscultation.

A diagnosis of acute atypical pneumonia was made at this time and specimens were collected so that an attempt could be made to determine the specific cause of the condition.

Blood samples from the above two animals showed the total white and the differential white blood counts as well as the haemoglobin to be within normal ranges. Faecal samples from the above two steers as well as from two other affected animals revealed evidence of the presence of a few gastrointestinal helminths. Samples of the hay fed were negative for **Dictyocaulus** larvae. Random samples of manure from the pen showed a high count of **A. lumbricoides** eggs.

On the third, fourth and fifth days following the outbreak all animals in the affected group received neomycin to protect them from secondary infections. On the sixth day they appeared clinically normal.

Pathology

On the fourth day one of the group, a steer not examined clinically, died and was brought to the Ontario Veterinary College. Lesions found at necropsy were as follows: The trachea and bronchi contained copious pink foam. The lungs which did not collapse, showed numerous minute petechiae beneath the pulmonary pleura. Bronchiectasis and atelectasis of the right apical, cardiac and azygos lobes were present. The remainder of the lung was erect, congested and slightly oedematous without consolidation. The abomasum was thickened as a result of submucosal oedema and hyperaemia. Fresh blood was found in the lumen of the duodenum while the rest of the intestine was slightly oedematous and congested. Histopathological examination of the lung showed diffuse interstitial pneumonia with focal hemorrhages and diffuse oedema. A few larvae, apparently **Ascaris**, were detectable in the bronchi and in the atelectatic areas surrounding the bronchiectasis.

The lungs were reexamined for parasites using various parasitological techniques. Random scrapings of the mucous membranes of the bronchi showed larvae in quite large numbers. Five to six gram random samples of lung were then cut into thin slices, placed in a pocket of surgical gauze and emersed in artificial gastric juice (Kennedy [3]). After four hours incubation at 30 °C. the larvae which had settled to the bottom of the gastric juice were placed on wet mount slides, stained with iodine and measured. These larvae agreed in general with the characteristics of the fourth stage. **A. lumbricoides** larvae described by Roberts [4] as well as with the characteristics of larvae isolated from a local case of "Thumps" in young pigs [5].

Discussion

The life cycle of **A. lumbricoides** as described by Lapage [6] is as follows: Soon after ingestion of infective **Ascaris** eggs, hatching of second stage larvae occurs in the intestine. Within twenty-four hours the larvae have migrated via the peritoneal cavity and/or the portal circulation to the liver. Reentering the blood stream the larvae lodge in the capillaries of the lungs. The majority then migrate to the alveoli and undergo their second moult. Following a third moult they travel up the trachea to the pharynx and pass down the esophagus to the intestine where they moult again. Here, in swine, they mature to the adult stage. In other species except man and some apes they never reach sexual maturity [7].

The eggs of **A. lumbricoides** may remain viable in organic matter such as manure from one to five years. The eggs require at least thirty days outside the host before coming infective. According to Campbell [5], respiratory signs attributed to the larval migration in the lungs of pigs occur around ten days post ingestion.

Different species of experimental animals vary widely in their susceptibility to the lung migration of **A. lumbricoides** [8]. Ransom [7] reported the condition causing pneumonia in sheep and goats while Kerr [9] described short term resistance in guinea pigs following single and multiple sublethal doses. **A. lumbricoides** migration in cattle has been studied in both naturally occurring and experimentally induced infections [3].

Kennedy [3] dosed **per os** each of four calves two to three days of age with

10,000 infective **A. lumbricoides** eggs. After eight days the only clinical sign of respiratory tract pathology was a mild dry cough in one calf. Two one month old calves, A and B, were also initially inoculated **per os** with 3,000 infective eggs. Calf A received an additional dose of 10,000 eggs 20 days following the first dose while calf B received 100 eggs on both the 20th and 27th day, 1,000 eggs on the 36th day, 500 eggs on the 42nd day and 15,000 eggs on the 46th day. Neither of the calves showed any signs of disease during the migration of the larvae resulting from the first inoculum. Five days following reinfection calf A developed a slight dry cough, a slight increase in normal lung sounds and a temperature rise (103 to 103.5 °F.). These signs persisted until the 8th day. An eosinophilia developed in calf B on the 13th day following its second exposure. This eosinophilia fluctuated but remained high until the calf was slaughtered 17 days later. The respiratory signs were less marked in this animal than in calf A and it remained afebrile throughout the experimental period. Kennedy's interpretation was that the host is actively sensitized during the first exposure and that the lesions resulting from a re-exposure are due to this altered state of sensitivity.

The gross and histological lesions of the above calves reported by Kennedy [3] were similar to the lesions of the affected yearlings reported in this paper.

It is postulated that the pathogenesis of the disease noted in this paper is as follows: The affected cattle received a very high dose of infective **A. lumbricoides** eggs **per os**, became sensitized, and shortly thereafter developed signs of acute atypical pneumonia.

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Bovine Mycotic Mastitis

A survey is given of the literature concerning yeast mastitis in cows. Routine mycological examinations of 980 milk samples from clinically normal quarters indicated that yeasts do not belong to the normal microflora of the udder. Five cases of mycotic infections were found in 480 samples from abnormal quarters. A description is given of a total of seven cases of udder infections from which the following agents were isolated: *Saccharomyces marxianus, S. fragilis, Candida Krusei, C. parapsilosis var. intermedia* and a *Trichosporon sp.*

Experimental infection of cows with two of the isolated strains produced distinct local and general symptoms. In vitro sensitivity tests revealed no effect of penicillin, streptomycin, chloramphenicol, auromycin and terramycin on any of the mastitis strains. The two latter antibiotics showed a marked growth stimulating effect. Polymynin B, nystatin and trichamycin proved to have an effect on all strains. A list of yeast species reported from mastitis cases demonstrates the rich variety of agents implied, the habits and the pathogenic properties of which are discussed. Attention is called to the role of the extensive use of antibiotics as a factor explaining the increasing number of mycotic mastitis. (authors' summary).

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