CASE REPORT

Caecal coccidiosis in commercial male turkeys

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ABSTRACT

An outbreak of coccidiosis with high mortality is reported in 30-day-old commercial turkeys. Grossly, a severe typhlitis with a large fibrino-necrotic core was present. Large numbers of oocysts were observed in caecal smears. The location and the severity of the lesions and the oocyst morphology were strongly suggestive of Eimeria adenoeides infection. This species has already been reported in turkey flocks in Italy, but it is rarely responsible for clinical coccidiosis and severe lesions with high mortality. Other caecal parasitic infections are considered in differential diagnosis.

Key Words: Turkey, Typhlitis, Coccidiosis, Eimeria adenoeides

INTRODUCTION

Coccidial infections in turkeys are sustained by seven Eimeria species (E. adenoeides, E. gallopavonis, E. melagrimitis, E. dispersa, E. innocua, E. meleagridis and E. subrotunda) (McDougald, 2003). The most pathogenic species are E. melagrimitis, E. adenoeides and E. gallopavonis and they have been recently included in the guidelines for evaluating efficacy of anticoccidials in poultry (Holdsworth et al., 2004). Turkey coccidiosis is commonly regarded as a minor economical issue that affects birds younger than 6-8 weeks (McDougald, 2003). These infections are widely present but rarely associated with clinical signs (Trees, 2002) and disease outbreaks remain poorly characterized. However coccidia can interact with bacteria, viruses and mycotoxins often exacerbating a number of enteric diseases (Witlock et al., 1984; Norton et al., 1993; Droual et al., 1994). Consequently, anticoccidials are routinely used in turkeys to prevent reductions in the weight gain and feed conversion. The aim of this paper is to describe a field case of caecal coccidiosis recently observed in a commercial turkey flock in Northern Italy.
Material and methods

In October 2004 an outbreak of disease characterized by high mortality and enteric disorders was reported in a commercial turkey farm located in Northern Italy. This farm housed 13,000 male birds in four houses and the enteric syndrome affected only one of them with a flock of 4000 birds. Monensin was used as coccidiostat in the feed. Five 30-day-old male turkeys were submitted for necropsy. Parasitological examination was carried out on caecal contents and mucosal scrapings. Samples of duodenum, caecum, pancreas, spleen, bursa of Fabricius, thymus and heart were collected in 10% buffered formalin for histopathology. Formalin-fixed tissues were embedded in paraffin wax, sectioned at 4 µm, and stained with Haematoxylin and Eosin. Digital micrographs of the caecal fresh smears were acquired (20x mag) and morphometric analysis (length and width) of 50 oocysts was performed by means of an image analysis software (Image Pro.Plus 5®). The average length and width and the shape index (length/width) were calculated.

Results and discussion

The outbreak of disease involving one of the four turkey houses was characterized by enteric disorders and mortality starting from the 4th week of age. A first attempt in controlling the syndrome with antibiotics (amoxicillin, colistin and flumequine) was unsuccessful. Histomoniasis was then suspected as field necropsy examinations of dead turkeys revealed severe caecal lesions in most of the birds. At slaughter time (140 days) the mortality in the affected flock reached 26% with an average weight of 18.8 Kg. The total mortality on the farm and the average slaughter weight were respectively 17% and 19.3 Kg with a feed conversion of 2.50.

The submitted turkeys were in poor condition and at necropsy the most striking feature was a moderate to severe typhlitis. The caeca were distended, thin-walled and contained a large grayish-white fibrino-necrotic core. A mild catarrhal enteritis was also present. The spleens appeared mildly smaller than normal and no liver lesion was present. The microscopic examination of mucosal scrapings of affected caeca revealed a small number of ellipsoidal oocysts. Conversely, a huge number of oocysts was ascertained in the smears of the caecal fibrino-necrotic cores. The oocysts appeared uniform in shape and size. The average oocyst dimensions were 24.5 x 16.1 µm and the average shape index was 1.53. These data are consistent with the morphologic characteristics of *E. adenoeides* (McDougald, 2003).

Histologically, a mild, chronic, catarrhal enteritis was detectable. The caecal lesions varied from a mild to moderate, multifocal sloughing of the villus epithelium with intact crypts to a diffuse disruption of the caecal mucosa. In the luminal exudate, large bacterial aggregates characterized by a mixed morphology were evident in association with parasites in different stages of development. Sections from spleens of 2 birds showed mild to moderate lymphocytic depletion, as well as one section from the bursa of Fabricius. Pancreas, thymus and heart were normal.

Enteric disorders, increased mortality and poor flock performances are commonly observed in commercial turkeys in association with a number of diseases. In the present case the early suspected bacterial involvement was excluded since antibiotics had failed to control the outbreak. The nature and the severity of the caecal lesions in the dead birds led the veterinary practitioners to suspect histomoniasis. However, the myriad of oocysts ascertained in the fibrino-necrotic caecal cores of the turkeys submitted to our laboratory was diagnostic of clinical coccidiosis. Histomoniasis was ruled out as no liver lesions were present and no histomonads were evident in the caecal smears and in the histologic sections. Moreover, microscopic examination revealed the caecal epithelium to be mostly affected and the lamina propria showed only a mild inflammatory involvement, whereas in histomoniasis it is usually severely affected and filled with numerous protozoa.

As for the etiology of this outbreak of coccidiosis, we assume that the unique involvement of the caeca, the severity of the lesion and the morphology of the oocysts are strongly suggestive of *E. adenoeides*. However, *E. meleagridis* and *E. gallopavonis* have been considered in differential diagnosis. Although the former replicates in the caeca it is regarded as non-pathogenic. E. gallopavonis is
more pathogenic, its oocysts closely resemble those of E. adenoeides but it affects primarily the lower ileum adjacent to the ileocecal junction and caecal lesions can very rarely occur (McDougald, 2003; Holdsworth et al., 2004). Moreover E. gallopavonis does not yet appeared to be recognized in Europe (Trees, 2002).

Eimeria spp. infections have already been reported in turkey commercial flocks in Italy (Grilli et al., 2001). Neither enteric signs nor intestinal lesions were present in the monitored flocks though the presence of E. adenoeides was suspected in the caecal content of 4-to-8-week-old birds from 3 of the 4 flocks examined. In the present case, however, the amount of oocysts was dramatically higher. Moreover the infection should have been very precocious or its course very rapid as clinical signs and mortality started from the 4th week. We cannot explain why the other 3 flocks on the farm remained unaffected as well as the previous and the following broods did.

Conclusions

This case was the most severe outbreak of turkey coccidiosis occurred in our diagnostic routine. The caeca were the only affected intestinal tract and contained myriads of oocists. Other parasites as Histomonas were undoubtedly excluded. Only E. adenoeides can be culprit for such severe caecal lesions. The early age of the birds may have played an essential role in the occurrence of the disease.

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