***Ministry of Higher Education and scientific research***

***UNIVERSITY OF kerbala***

***College of Veterinary Medicine***

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**الكوكسيديا في العجول**

***Supervision أشراف***

***Assistant Professor Dr. Hayder Badri Abboud***

***الأستاذ المساعد الدكتور حيدر بدري عبود***

***By***

***Haider Abd Alamir Mahmoud***

***Fourth year 2014-2015***

***حيدر عبد الأمير محمود***

***المرحلة الرابعة 2014-2015***

Coccidiosis in cattle

***Introduction***

Coccidiosis is caused by infection by protozoan parasites called Eimeria spp. which parasitize the lining of the intestinal tract. Infection causes a loss of absorptive capacity of the gut with consequent diarrhoea and possibly dysentery. Outbreaks of disease are commonly seen 3-4 weeks after mixing groups of dairy calves.(1)

***Etiology***

Twelve *Eimeria spp* have been identified in the feces of cattle worldwide, but only 3 (*E zuernii*, *E bovis*, and *E auburnensis*) are most often associated with clinical disease. The other *Eimeria spp* have been shown experimentally to be mildly or moderately pathogenic but are not considered important pathogens.(2)

***Epidemiology***

Coccidiosis is commonly a disease of young cattle (1–2 mo to 1 yr) and usually is sporadic during the wet seasons of the year. “Summer coccidiosis” and “winter coccidiosis” in range cattle probably result from severe weather stress and crowding around a limited water source, which concentrates the hosts and parasites within a restricted area. Although particularly severe epidemics have been reported in feedlot cattle during extremely cold weather, cattle confined to feedlots are susceptible to coccidiosis throughout the year. Outbreaks usually occur within the first month of confinement. Cows may contribute to environmental contamination of *E bovis* oocysts through a periparturient increase in fecal oocyst counts. Time to onset of diarrhea after infection is 16–23 days for *E bovis* and *E zuernii* and 3–4 days for *E alabamensis*; clinical disease due to coccidiosis does not typically occur in the first 3 wk of life. Coccidiosis is therefore not considered part of the neonatal diarrhea complex in calves.(3)

***Life cycle***

******Coccidiaoocyst (eggs) are ingested by susceptible animals when they consume contaminated feed or water, graze contaminated pasture or lick a dirty hair coat. The oocysts release sporozoites (larvae) that multiply asexually in the cells lining the wall of the small intestine and releasing thousands of merozoites (2nd generation larvae). The merozoites then enter the large intestine and go through a sexual reproductive cycle to produce thousands of oocyst. These oocyst pass out with the manure to further contaminate soil, feed, water, bedding, etc. and begin the cycle again.

Each oocyst contains eight sporozoites. Each sporozoite enters multiple cells in the lining of the small intestine and destroys the cell as it forms a schizont (packet) of merozoites. When it ruptures, it releases more than 100,000 new merozoites and it has been estimated that ingestion of only 125 sporulated oocysts will subsequently cause damage to more than 6 billion intestinal cells. This interferes with digestion and absorption of nutrients. About 71% of the life cycle occurs in the small intestine. Rarely do you see any clinical signs during this time in the small intestine. When they go to the large intestine around day 16 and the oocyst breaks out of the cell following the sexual reproduction around day 18, it causes diarrhea and blood in the feces from the damaged gut lining. Oocysts can be detected in the manure around 21 days.

Oocysts passed in manure need moisture and mild temperatures to sporulate. High temperatures and dryness impede sporulation. They can survive freezing (down to about 18 degrees F) for a couple of months, but temperatures below minus 22 degrees F will usually kill them. In both cases the manure or straw pack may give them the protection to survive even the harshest environmental conditions.(4)

***Clinical signs***

The most typical syndrome of coccidiosis is chronic or subclinical disease in groups of growing animals. Calves may appear unthrifty and have fecal-stained perineal areas. In light infections, cattle appear healthy and oocysts are present in normally formed feces, but feed efficiency is reduced. The most characteristic sign of clinical coccidiosis is watery feces, with little or no blood, and animals show only slight discomfort for a few days. Severe infections are rare. Severely affected cattle develop thin, bloody diarrhea that may continue for >1 wk, or thin feces with streaks or clots of blood, shreds of epithelium, and mucus. They may develop a fever; become anorectic, depressed, and dehydrated; and lose weight. Tenesmus is common because the most severe enteritis is confined to the large intestine, although pathogenic coccidia of cattle can damage the mucosa of the lower small intestine, cecum, and colon. During the acute period, some calves die; others die later from secondary complications (eg, pneumonia). Calves that survive severe illness can lose significant weight that is not quickly regained or can remain permanently stunted. Calves with concurrent enteric infections (eg, *Giardia*) may be more severely affected than calves with coccidia infections alone. In addition, management factors, such as weather, housing, feeding practices, and how animals are grouped, are important in determining the expression of clinical coccidiosis in cattle.

Nervous signs (eg, muscular tremors, hyperesthesia, clonic-tonic convulsions with ventroflexion of the head and neck, nystagmus) and a high mortality rate (80–90%) are seen in some calves with acute clinical coccidiosis. Outbreaks of this “nervous form” are seen most commonly during, or following, severely cold weather in midwinter in Canada and the northern USA; there are no reports of the “nervous form” outside this geographic location. Affected calves may die <24 hr after the onset of dysentery and nervous signs, or they may live for several days, commonly in a laterally recumbent position with a mild degree of opisthotonos. Nervous signs have not been reported in experimental clinical coccidiosis in calves, which suggests that the nervous signs may be unrelated to the dysentery or, indeed, even to coccidiosis.(5)

***Diagnosis***

Veterinary diagnosis is based upon typical clinical findings affecting a large number of calves in the group. Interpretation of faecal examinations is not simple because there are low numbers of oocysts present in the faeces of many normal calves. The stage of infestation also greatly influences the number of oocysts present in faeces. So the demonstration of large numbers oocysts in faecal samples is helpful but speciation to determine whether they are pathogenic (capable of causing disease) is rarely undertaken in field outbreaks. There is a good response to specific anticoccidial therapy.Histopathology findings of coccidiosis in the gut of a dead calf confirms the clinical diagnosis.(6)



***Differential diagnosis(7)***

* parasitic gastroenteritis
* salmonellosis
* persistent infection with BVD
* necrotic enteritis
* ragwort poisoning
* lead poisoning
* peritonitis

***Necropsy finding***

* Fecal staining of hindquarters and tissue pallor of the carcasses are common.
* The pathogenic coccidia of cattle can damage the mucosa of the lower small intestine, cecum, and colon causing congestion, hemorrhagic enteritis, and thickening of the mucosa. Ulceration or sloughing of the mucosa may occur in severe cases.
* The first-generation schizonts of E bovis appear as white macroscopic cyst-like bodies in the villi of the terminal ileum.(8)

***Treatment***

Drugs that can be used for therapy of clinically affected animals include sulfaquinoxaline (6 mg/lb/day for 3–5 days) and amprolium (10 mg/kg/day for 5 days). Sulfaquinoxaline is particularly useful for weaned calves that develop bloody diarrhea after arrival at a feedlot. For prevention, amprolium (5 mg/kg/day for 21 days), decoquinate (22.7 mg/45 kg/day for 28 days) and lasalocid (1 mg/kg/day to a maximum of 360 mg/head/day), or monensin (100–360 mg/head/day) can be used. The major benefits of coccidiostats are through improved feed efficiency and rate of gain.(9)

***Prevention andcontrol***

Coccidiosis has been difficult to control reliably. Overcrowding of animals should be avoided while they develop an immunity to the coccidial species in the environment. Calving grounds should be well drained and kept as dry as possible. All measures that minimize fecal contamination of hair coats and fleece should be practiced regularly. Feed and water troughs should be high enough to avoid heavy fecal contamination. Control of coccidiosis in feeder calves brought into a crowded feedlot depends on management of population density, presence of appropriate feed bunks, or use of chemotherapeutics, to control the numbers of oocysts ingested by the animals while effective immunity develops.

Control of infection should include changes in management factors that contribute to the development of clinical disease. Inadequate housing and ventilation should be corrected, feeding practices adopted that avoid fecal contamination of feed, calves grouped by size, and an “all-in/all-out” method of calf movement from pen to pen adopted.(10)

***Reference***

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