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**colibacillosis in newly born farm الاسهال الابيض في صغار العجول**

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Escherichia Coli in new born calves
Diarrhea is common in newborn calves, lambs, and kids. The clinical presentation can range from mild diarrhea without systemic disease to profuse, acute diarrhea associated with rapid dehydration, severe disturbance of acid-base and electrolyte balance, and death, sometimes in as few as 12 hr. This discussion emphasizes the disease in calves, but the principles of pathophysiology and treatment apply to lambs and kids as well.

Etiology

Several enteropathogens are associated with diarrhea in neonates. Their relative prevalence varies geographically, but the most prevalent infections in most areas are *Escherichia coli*, rotavirus, coronavirus, and *Cryptosporidium parvum*. Cases of neonatal diarrhea are commonly associated with more than one of these agents, and the cause of most outbreaks is multifactorial. Determining the particular agents associated with an outbreak of diarrhea can be important, because specific therapy and prophylaxis are available for some. Also, some agents have zoonotic risk. Diarrhea is also present in septicemic colibacillosis[1].

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*E coli* is the most important bacterial cause of diarrhea in calves during the first week of life; at least two distinct types of diarrheal disease are produced by different strains of this organism. One type is associated with enterotoxigenic *E coli*, which has two virulence factors associated with production of diarrhea. Fimbrial antigens enable them to attach to and colonize the villi of the small intestine of neonatal calves in the first days of life. Strains in calves most commonly possess K99 (F5) or F41 fimbrial antigens, or both. These antigens are the focus of immunologic protection. Enterotoxigenic *E coli* also elaborate a thermostable, nonantigenic enterotoxin (Sta) that influences intestinal ion and fluid secretion to produce a noninflammatory secretory diarrhea. Diarrhea in calves and lambs also has been associated with enteropathogenic *E coli* that adhere to the intestine to produce so-called attaching and effacing lesions, with dissolution of the brush border and loss of microvillous structure at the site of attachment, a decrease in enzyme activity, and changes in ion transport in the intestine. These enteropathogens are also called “attaching and effacing *E coli*.” Some produce verotoxin, which may be associated with a more severe hemorrhagic diarrhea. The infection most frequently is in the cecum and colon, but the distal small intestine can also be affected. The damage in severe infections can result in edema and mucosal erosions and ulceration, leading to hemorrhage into the intestinal lumen. Diarrhea caused by enterotoxigenic Escherichia coli is an infectious bacterial disease of calves that occurs during the first few days of life. The Escherichia coli that cause the disease possess special attributes of virulence that allow them to colonize the small intestine and produce an enterotoxin that causes hypersecretion of fluid into the intestinal lumen. These enterotoxigenic Escherichia coli are shed into the environment by infected animals in the herd and are ingested by newborn calves soon after birth. There is some natural immunity to enterotoxigenic Escherichia coli; however, it often fails to protect calves born and raised under modern husbandry conditions. Hence, methods have been developed to stimulate protective immunity by vaccination of the dam. The protective antibodies are transferred passively to calves through the colostrums[2].

Epidemiology and Transmission

Enteropathogens associated with diarrhea are commonly found in the feces of healthy calves; whether intestinal infection leads to diarrhea depends on a number of determinants, including differences in virulence of different strains of a pathogen and the presence of more than one pathogen. The resistance of the calf is of major importance and is largely determined by successful passive transfer of colostral immunoglobulins. Colostrum-deprived calves are highly susceptible to infection with enteropathogens and develop severe and often fatal disease.

Pathogenesis

Diarrhea in neonatal ruminants is usually associated with disease of the small intestine and can be caused by hypersecretion or malabsorption. Hypersecretory diarrhea develops when an abnormal amount of fluid is secreted into the gut, exceeding the resorptive capacity of the mucosa. In malabsorptive diarrhea, the capacity of the mucosa to absorb fluid and nutrients is impaired to the extent that it cannot keep up with the normal influx of ingested and secreted fluids. This is usually the result of villous atrophy, in which the loss of mature enterocytes at the tips of the villi results both in a decrease in villous height (with a consequent decrease in the surface area for absorption) and in loss of the brush border digestive enzymes. The extent and distribution of villous atrophy varies with different pathogens and can explain variation in the severity of clinical disease. Malabsorptive diarrhea may be aggravated by the colonic fermentation of nutrients that normally would have been absorbed in the small intestine. Fermentation products, especially lactic acid, appear to draw water into the colon osmotically, which contributes to the severity of diarrhea.

Clinical Findings

The major signs are diarrhea, dehydration, profound weakness, and death within one to several days of onset.

Diarrhea due to enterotoxigenic (K99-bearing) *E coli* is seen in calves <3–5 days old, rarely later. However, the age of susceptibility may be extended in the presence of other pathogens. Onset is sudden. Profuse amounts of liquid feces are passed, and the calves rapidly become depressed and recumbent. Calves may lose >12% of body weight in fluid, and hypovolemic shock and death may occur in 12–24 hr. Body temperature may be increased but is commonly normal or subnormal. If fluid and electrolyte therapy is administered early, response is usually good. Disease produced by attaching and effacing *E coli* is seen predominantly in calves from 4 days to 2 mo old and may manifest with diarrhea or primarily as dysentery with blood and mucus in the feces. The clinical course is short.

Diagnosis

difficult to make a definite etiologic diagnosis for diarrhea based solely on clinical findings. However, the history, age of the animal(s) affected, and clinical signs may permit a presumptive diagnosis. Fecal samples can be submitted for isolation and characterization of the common enteropathogens. Samples should be taken from several untreated calves in the early stages of diarrhea. Special techniques are necessary for the demonstration of viruses, cryptosporidia, and K99-bearing *E coli*. The interpretation of fecal microbiology can be difficult because of mixed infections and because enteropathogens are commonly present in the feces of healthy calves.

The best diagnostic information is usually obtained by submitting untreated, acutely affected animals for necropsy. This allows examination of intestinal mucosa for evidence of diagnostic lesions and for the presence of enteropathogens such as cryptosporidia. It may be the only way that disease such as that associated with attaching and effacing strains of *E coli* can be diagnosed. The diagnostic value of a necropsy diminishes quickly with time after death; important lesions can disappear within minutes due to autolysis.

Treatment

Many of the factors involved in disease resistance are nonspecific; thus, important preventive measures can be taken and therapy can be started before an etiologic diagnosis has been established. Treatment includes fluid therapy for water and electrolyte replacement and correction of acid-base disturbances, alteration of the diet, and antimicrobial and anti-inflammatory therapy.

Fluid and electrolyte therapy is most important and should be started as soon as possible regardless of whether clinical evidence of dehydration has developed (clinical signs of dehydration are not apparent until the calf has lost at least 6% of its body weight in fluid). Calves still able to stand and willing and able to suck can often be treated with oral electrolyte solutions alone. Fluids for oral rehydration should promote the cotransport of sodium with glucose and amino acids and should contain sodium, glucose, glycine or alanine, potassium, and either bicarbonate or citrate or acetate as alkalinizing agents. Several commercial preparations are available. These can be administered by nipple bottle or, if necessary, by stomach tube. The solutions should be used liberally until the animal is rehydrated.  keep the calf hydrated, fluids must be replaced at the same rate of loss . As far as antibiotic there are quite a few , Scour Halt, SMZ-TMP, Vetisulid, Quatra Con, to name a few . The main thing is to replace the fluids .[3]

Prevention and Control .

Because of the complex nature of diarrhea in neonates, it is unrealistic to expect total prevention—economical control is the major objective. The incidence of clinical disease and the case fatality rate depend on the balance between the levels of exposure to infectious agents and the resistance in the calf. Differences in herd size; availability of facilities, land, and labor; and general management objectives make it impossible to recommend specific management procedures applicable to all situations. However, three broad principles apply in all herds: 1) the degree of exposure of neonates should be reduced by isolating diseased animals or by moving calving and calf rearing to a separate area, and by practicing good general hygiene; 2) nonspecific resistance should be maximized by providing good nutrition to the dam and neonate and assuring that newborn calves consume ≥5% of their body wt of high-quality colostrum, preferably within 2 hr and certainly within 6 hr of birth, followed by equivalent amounts at 12-hr intervals for the next 48 hr; and 3) the specific resistance of the newborn should be increased by vaccinating the dam or the newborn Vaccination of pregnant cows with rotavirus and coronavirus vaccines increase the amount of specific antibody in colostrum and milk, but the concentration of antibodies in milk may be insufficient to provide local antibody in the intestinal lumen during the period of peak prevalence of infection, which, in calves, is 5–15 days of age. Controlled trials of commercial vaccines have shown variable results. The addition of small amounts of immune colostrum to milk fed during the period of susceptibility can provide some protection against disease.

REFRENCES

[1]<http://www.merckmanuals.com/vet/digestive_system/intestinal_diseases_in_ruminants/diarrhea_in_neonatal_ruminants.html> [2]<http://www.ncbi.nlm.nih.gov/pubmed/2579990> [3]http://www.cattletoday.com/forum/viewtopic.php?f=7&t=55558