VETERINARY SCIENCE RESEARCH JOURNAL
olume **6** | Issue 1 | Apr., 2015 | 58-66

DOI : 10.15740/HAS/VSRJ/6.1/58-66
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e ISSN-2230-942X

REVIEW ARTICLE

Work on characterization of the effect of collibacillosis on calves' performances

KINDALEM BAYEW[1], TEWODROS FENTAHUN[1], MUKARIM ABDURAHAMAN[2] AND ADDIS GETU

Members of the Research Forum

Associate Author :
[1]Faculty of Veterinary Medicine, University of Gondar, GONDAR (ETHIOPIA)

[2]College of Agriculture and Veterinary Medicine, Jimma University, JIMMA (ETHIOPIA)

AUTHOR FOR CORRESPONDENCE :
ADDIS GETU
Faculty of Veterinary Medicine, University of Gondar, GONDAR ETHIOPIA

Abstract : Collibacillosis is one of the most common diseases of farm animals caused by pathogenic stains of *E.coli* bacteria. The occurrences of this disease are as Septicemic and enteric forms. The form is caused by strains that are able to invade the extra intestinal tissue, resist the bactericidal effect of complement, survive and multiply in body fluids, escape phagocytosis and induce damage by the release of cytotoxins. Later the strains of *E.coli* have colonize and proliferate the ability in the upper part of the small intestines and produces enter toxins, which causes an increase in net secretion of fluid and electrolytes from the systemic circulation. The enteric Collibacillosis is one of the most common causes of diarrhea in very young calves (in the first week life) and causes high mortality in this age group. Demonstration of enterotoxigenic strains can be done by cultural isolation of the relevant strains, assay of toxins, and histopathological, serological, and molecular techniques. When therapy is to be established, physical examination should be done to determine the current disease and level of dehydration. Although antibiotics are frequently used in the treatment of Collibacillosis, fluid and electrolyte therapy is the major part of treatment to prevent dehydration and electrolyte imbalance. Measures to control the disease includes avoiding group housing, weather, stress and poor hygiene; supplementing nutrition to the pregnant dam; early colostrum feeding to the calves; vaccination of pregnant dams in endemic areas; and effective management of outbreak when it occurs.

Key words : Collibacillosis, Calves, Colostrum

How to cite this paper : Bayew, Kindalem, Fentahun, Tewodros, Abdurahaman, Mukarim and Getu, Addis (2015). Work on characterization of the effect of collibacillosis on calves' performances. *Vet. Sci. Res. J.*, **6**(1) : 58-66.

Paper History : Received : 02.02.2015; Revised : 10.03.2015; Accepted : 19.03.2015

INTRODUCTION

Neonatal calves are characterized with highly exposure for Collibacillosis that causes in a very common and serious bacterial diarrhea. It affects mainly calves at younger age which is caused by *E.coli.* The bacteria causes two common disease of new born calves; coli septicemia in which the bacteria invade the systemic circulation and internal organs, and enteric Collibacillosis in which the bacteria is isolated to the lumen and mucosal surfaces of the small intestine (Hirsh *et al*., 2004).The pathogenesis of these two diseases is markedly different and their causes them are also differs. Each type posses unique attributes of virulence that differentiate it from each other as well as from one

pathogenic group of *E.coli,* which are part of normal digestive flora of healthy calves which affect the overall reproductive performances. Hence, *E.coli* cause septicemia survives and multiplies in the blood and internal organs of calves. Conversely those that cause diarrhea are equipped to survive locally in gastro intestinal tract (Hirsh *et al*., 2004).

Collibacillosis is one of the common diseases of new born farm animals and is a significant cause of highly economic loss in raising livestock. It is particularly important in intensive system of animal production. For example, the morbidity in dairy calves raised under intensified condition may reach up to 70 per cent but it is usually 30 per cent (Radostits *et al*., 1994). Collibacillosis is a disease of intensification involves many epidemiological factors for its causation (Thrusfield, 2005). Its control requires for understanding of all aspects of the disease (Radostits *et al*., 1994). The objective of this paper was to review and compile available information on different aspects of this important disease on calves' performances.

General feature of *E. coli* :

The bacteria *E.coli* is an important pathogens of animals. It is a gram negative, facultative anaerobic, rod-shaped bacterium that is commonly found in the lower intestine of warm blooded organisms (endotherms). Most *E.coli* strains are harmless, but some serotypes can cause serious food poisoning in their hosts and food contamination. The harmless strains are part of the normal flora of the gut, and preventing colonization of the intestine with pathogenic bacteria. It is the major facultative gram negative species comprising the normal flora of the GIT but may also be the cause of septicemic disease in foals, calves, piglets, puppies, and lambs; of enterotoxigenic *E.coli* diarrhea in new born farm animals and edema disease in pigs enterotoxigenic *E.coli* (Hirsh and Zee, 1999; Hirsh *et al*., 2004). Colonization of mammalian intestinal tract by *E.coli* from the environmental sources occurs shortly after birth and these organisms persist as an important member of the normal flora of the intestine throughout life. Most strains of *E.coli* are low virulence. Pathogenic strains have virulence factors, which allow them to colonize mucosal surfaces and subsequently produce disease. Predisposing factors, which permit colonization, includes age, immune status, nature of diet, and heavy exposure to pathogenic strains (Quinn and Markey, 2007). Somatic (O), flagellar (H), and capsular (K) antigens are used for serotyping of different strains of *E.coli*. The somatic antigens are lipopolysaccharides (LP). Proteinaceous fimbrial (F) antigens act as adhesions facilitating attachments to mucosal surface. For examples O141:K85:H3 describes an isolate with antigen of 141 serogroup, capsular antigens 85 and flagellar antigen 3 (Hirsh and Zee, 1999).

E. coli is a natural inhabitant of the intestines. However, some types of *E.coli* bacteria are capable of causing disease. The source of infection in an otherwise uninfected herd is usually normal calves and adult cows that serve as reservoirs of infection. These carrier animals can allow the bacteria to persist in a herd by circulating through animals of all ages. The most common route of infection with these pathogenic forms of *E.coli* is ingestion. It is also possible for calves to become infected via the nasopharyngeal mucosa (*i.e.* inhalation), which can lead to meningitis (infection of the tissues around the brain). Calves become exposed to pathogenic *E. coli* in the environment when other infected or carrier calves and cows shed the bacteria in the feces. The calves become ill when they ingest *E. coli* from contaminated bedding and calf pails, dirty calf pens, diarrheic calves in overcrowded calving grounds, and from the skin of the perineum and udder of the dam.

Pathogenic strains of *E. coli* :

Enterotoxigenic E.coli :

Entero toxigenic starins of *E.coli* posses the ability to colonize and proliferate in the upper part of the small intestines and to produceenterotoxins, which causes an increase in net secretion of fluid and electrolytes from the systemic circulations (Acres, 1985). The adhesion of *E.coli* to the intestinal epithelial cells is mediated by bacterial pilli and the mechanism of attachment to the receptor is complex (Radostits *et al.,* 1994). Calves affected by enterotoxigenic *E.coli (*ETEC) are depressed, anorexic, rapidely dehydrated and more than 90 per cent of the cases are caused by *E.coli* containing the k99 pillus attachment fimbriae types. In addition to k99 antigen several other fimbriae antigens designated as F41, Fg26, Att 25 and F210 have been identified on calves ETEC. Among fimbrial

adhesion F41 is the best characterized (Acres, 1985). Villous attachment and colonization by strains of ETEC having multiple types of appear to be effectively prevented by vaccination with bacteria that have only a single pillus antigen common with challenges strains (Smith, 2002). ETEC produces two types of toxins (heat stable and heat labile entero toxins) that can be important in causing the disease (Smith, 2002).

Enter pathogenic *E. coli* :

They are absorbed as attaching and effacing *E.coli* because they attach to the brush border, distort, and destroy the microvilli in severe cases. They produces the micro haemorrhagic colitis with petechial or chymotic haemorrhages in the wall of colon and rectum. They produce a toxin known as shiga like toxins (SLT) or verotoxins. An *E.coli* that carries this toxin is k-99 negative and may belong to O serotype 5, 26, 111 (Smith, 2002). Shiga toxin producing *E.coli* (STEC) produces cytotoxin identical at the genetic and protein level to the shiga toxins produced by shiga dysenteria. Enterohaemorrhagic *E.coli* is subset of STEC. EHEC is divided into typical and atypical EHEC strains. Typical EHEC strains cause haemorrhagic colitis and haemorrhagic uremic syndrome (HUS) in human (Tutenl *et al.*, 2002). There are reports of outbreaks of bloody diarrhea in calves from 2 days to 4 days old associated with enterohaemorrhagic *E. coli*. In some of the outbreak, other agents also involved (Smith, 2002). STEC of serotype O157:H7 carries at the high frequencies by cattle but have not been implicated in natural disease, although they may be used to experimentally reproduce disease in calves (Carter, 1979). Calves naturally or experimentally infected with STEC that induces enteric disease show no systemic sign. This has been attributed to lack of the Gb3 receptor in the blood vessels of calves. The most common clinical sign is diarrhea but dysentery and dehydration are seen in some cases (Smith, 2002). Diarrhea has been associated with certain STEC particularly of O118 serogroup. The STEC that can implicate, are typically Shiga toxin-1 (Carter and Changapa, 1989).

Enter invasive *E. coli* :

The enter invasive strains adhere to cells of the distal small intestine, invade the enterocytes and deeper layers of the intestinal mucosa (Quinn *et al.*, 1994). Invasive strains of *E.coli*, invade the tissue and systemic circulation via the intestinal lumen, nasopharyngeal mucosa and tonsillar crypts or umbilical vessels (Radostits *et al.*, 1994). These strains are able to invade the extra intestinal tissues, to resist the bacteriocidal effect of compliment, to survive and multiply in the body fluids, to escape phagocytosis and intercellular killing by phagocytes and to invade tissue damage by the release of cytotoxins (Radosits *et al.*, 1994). The virulence factors such as capsules, adhesions, sideronores and alpha hemolysins are important as survival factors for these invasive strains, which are responsible for colisepticemia (Quinn *et al.*, 1994).

Forms of collibacillosis :

Septicemic collibacillosis (Colliform septicemia) :

This result from the invasive strains of *E.coli* invading the tissues and systemic circulations via the intestinal lumen, nasopharyngeal mucosa and tonsillar crypts or umbilical vessels (Smith, 2002). These strains are able to invade the extra intestinal tissues to resist the bactericidal effect of compliment to survive and multiply in body fluids to escape phagocytosis and intracellular killing by phagocytes to induce damage by the release of cytotoxins. Calves and piglets, which are deficient in colostral, immunoglobulin is highly susceptible to septicemia. Colostrum provides protections against collisepticemia, but may not prevent diarrhea caused by *E.coli*. Although colostrums fed calves are much more resistant to endotoxins that colostrums deprived calves; calves, piglets, and lambs; which normal level of serum immunoglobulin are generally protected from septicemia (Andrew *et al*., 2004).

Septicemic invasion can produce shock with a low grade diarrhea in young calves; most commonly in those that are less than 4 days old (Smith, 2002). The clinical finding and lesions in septicemic collibacillosis are attributable to effects of endotoxins, which causes shock (Andrew *et al.*, 2004). Differentiations of these diseases from simple enteropathies depend on demonstration of signs of bacteremia such as arthititis, or hyperesthesia and neck rigidity associated with meningitis. Fever is not a consistent feature of septicemia in neonates and cannot be used to rule out septicemia. Diarrhea is not profuse reasonably secondary to endotoxemia. Low grade bacteremia does not produce

obvious signs, but the organism can localize in joints and the development of septicemic arthritis in older calves (Smith, 2002).

Enteric collibacillosis :

Enteric collibacillosis is caused by Enterotoxigenic *E.coli* characterized by profuse watery diarrhea (Smith, 2002). ETEC most commonly causes diarrhea in calves under 4 years old; although, they can produce diarrhea in older calves (Andrew *et al.,* 2004; Smith, 2002). In experimentally infected new born calves, the incubation period is 12-18 hrs (Andrew *et al*., 2004). Enterotoxigenic strains of *E.coli* posses the ability to colonize and proliferate in the upper part of small intestine and to produce enterotoxins, which causes an increase in net secretions of fluid and electrolytes from the systemic circulation .The enterotoxigenic form of colibacillosis occurs most commonly in calves and piglets and less commonly in foals and lambs (Radostits *et al*., 1994). Enterotoxigenic *E.coli* is often haemolitic and usually belongs to O group 8, 9, 20, 101 or 141 (Smith, 2002). ETEC posses two virulence attributes (determinants) that distinguish them from non pathogenic strains; ability to adhere to the mucosal surface of electrolytes and ability to produce enterotoxins. Adhesions is mediated by filamentous protein structures called fimbriae, sometimes called pilli or adhesions which bind to the specific receptors on the enterocytes cell membrane. Adhesion mediated by fimbriae does not bring the bacterium in close contact with the electrolyte luminal surface as in the EPEC and EHEC. The microvilli are unaltered and "caps" and "pedstals" are not seen (Andrew *et al.,* 2004).

Antigens that bind to the calves electrolytes include k99, k88 and F41 (Smith, 2002). But the adhesions antigen commonly found in ETEC of calves are k99 and f41. They often occur together but may present independently. Their adhesion ability allows ETEC to overcome the peristalsis of the small intestine by sticking to the mucosal surface. Adhesions of the bacterium to the electrolyte surface confer other advantages to the bacteria because enterotoxins are released close to their receptor sites. The ability of ETEC to produce fimbriae and endotoxins is determined by genes that are usually carried on a single plasmid and thus, occurs together. It is adequate, therefore to diagnose ETEC infection by detecting either the neurotoxin or adhesion (Andrew *et al*., 2004). Calves infected with ETEC have a mild inflammatory reaction in the small intestinal wall and some villous atrophy. In fresh specimens, sheets of gram negative bacilli can adhere to the small intestinal mucosa (Smith, 2002). The production of neurotoxins by the *E.coli* results in net secretions of fluid and electrolytes from the systemic circulation into lumen of the gut resulting in varying degrees of diarrhea, dehydration, electrolyte imbalance, acidosis, hyperkalemia, and when acidosis is severing circulatory failure, shock and death. The hyperkalemia in calves with neonatal diarrhea and acidosis has been associated with cardiac rate and rhythm abnormalities including bradycardia and atrial stand still. The response to *E.coli* enterotoxins in calves and piglets is similar to neurotoxins in man and take place through an intact mucosa (Radostits *et al*., 1994).The course is rapid and the calf may progress from health to recumbence and death within 6-12 hrs. Weakness may be noticed before diarrhea is observed. When the diarrhea breaks, it is profuse and watery and is passed with straining (Smith, 2002). Special culture media may be required to encourage ETEC to express adhesions, because bacteria in general are poor producer of K99 and F41, even though those growing in the gut readily produce them. Once expressed, they can be detected using specific antisera in agglutination, hemagglutination or ELISA test.

Epidemiology and transmission :

Transmission of pathogenic *E. coli* often occurs via fecal-oral transmission. Common routes of transmission include- unhygienic food preparation, farm contamination due to manure fertilization, irrigation of crops with contaminated grey water or raw sewage, feral pigs on cropland, or direct consumption of sewage-contaminated water. Dairy and beef cattle are primary reservoirs of *E. coli* O157:H7, and they can carry it asymptomatically and shed it in their feces. Food products associated with *E. coli* outbreaks include cucumber raw ground beef, raw seed sprouts or spinach, raw milk, unpasteurized juice, unpasteurized cheese and foods contaminated by infected food workers via faecal–oral route (Radostits *et al*., 1994). According to the USA food and drug administration (FDA), the faecal-oral cycle of transmission can be disrupted by cooking food properly, preventing cross-contamination, instituting barriers such as gloves for food workers, instituting health care policies so food industry employees seek treatment when they are ill, pasteurization of juice or dairy products and proper hand washing requirements (Radostits *et al.,*1994).

Shiga toxin-producing *E. coli* (STEC) specifically serotype O157:H7, have also been transmitted by flies, as well as direct contact with farm animals, petting zoo animals, and airborne particles found in animal-rearing environments (Tutenel *et al*., 2002). Enter pathogens 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 (Carter, 1979). The progression of infection, the severity of lesions produced, and the severity of the diarrhea can be modulated by immunoglobulins received via colostrum. Immunoglobulins act directly on pathogens in the intestinal lumen during the period of colostrum ingestion, because significant amounts of circulating immunoglobulins are re-secreted into the intestine, especially when the concentration of circulating immunoglobulin is high (Radostits *et al.*, 1994). The lack of specific antibodies in dams that have not been exposed to specific pathogens, and the use of specific vaccines, further modulate this influence. Stress caused by a poor environment, inadequate protection from the weather, or an insufficient or inappropriate diet also increases the risk for disease (Smith, 2002). With all of the enteropathogens, healthy adult cattle may be carriers and periodically excrete the organism in feces. Excretion may increase with the stress of parturition and be more frequent in primiparous cows. This can lead to contaminated calving areas and infection of the udder and perineum of the dam. Other sources of infection include the feces of healthy calves and the feces of diarrheic calves, which contain large numbers of organisms early in the course of infection. A few scouring calves can result in severe contamination of the calf-rearing area. Transmission is by fecal-oral contact, fecal aerosol, and, in the case of corona virus, also by respiratory aerosol (Radostits *et al.*, 1994).

Pathogenesis :

The major factors which are important in the understanding of collibacillosis are immune status of animal and properties of strains of *E.coli,* its capacity to invade tissues and produces a septicemia or to produce enterotoxin (Radostits *et al.,* 1994). ETEC enter the animal by oral route when present in sufficient amount; colonize the small intestines following attachment by fimbrial adhesions to receptors on the small intestinal epithelium or in the mucous coating epithelium (Gyles *et al.,* 2004). ETEC adhering closely to the intestinal epithelium produces enterotoxins that stimulate the secretions of water and electrolytes into the intestinal lumen. This leads to diarrhea, if the excess fluid from small intestine and large intestine is not absorbed. ETEC causes severe watery diarrhea, which mean lead to dehydration, listlessness, metabolic acidosis and death (Gyles *et al*., 2004).

Diarrhea in neonatal ruminants is usually associated with disease of the small intestine and can be caused by either 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 (Gyles *et al*., 2004). 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 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 (Smith, 2002).Inflammation contributes to the pathophysiology of diarrhea in most intestinal infections, and mediators of inflammation can affect ion flux within the intestine. Inflammation also leads to vascular and lymphatic damage and to structural damage of the crypt-villus unit. Most infectious forms of diarrhea have hypersecretory, inflammatory and malabsorptive components. These lead to a net loss of water, sodium, potassium, and bicarbonate. If severe, the calf develops hypovolemia, hyponatremia, acidosis, and prerenal azotemia (Radostits *et al*., 1994).

Enterotoxigenic *E.coli* produces the enterotoxin, which stimulates marked hypersecretion by activating guanylate cyclase and by inducing a net secretion of sodium and chlorine. The membrane-bound sodium-glucose co transport system remains functional. Inflammation, leading to necrosis of the enterocyte, submucosal inflammatory infiltration,

and villous atrophy is a major component of the pathophysiology of diarrhea produced by *E.coli*. Infections with verotoxin-producing enteropathogenic *E.coli* result in accumulation of fluid within the large intestine and extensive damage to the large intestinal mucosa with edema, hemorrhage, and erosion and ulceration of the mucosa which results in blood and mucus in the lumen (Gyles *et al*., 2004).

Inappropriately formulated milk replacers produce diarrhea by 2 mechanisms, both associated with malabsorption. Vegetable (especially soybean) products are commonly used as protein sources in the manufacture of milk replacers. Depending on the degree of refinement, these products may contain carbohydrates that are indigestible in young calves. Such carbohydrates are not absorbed in the small intestine and may contribute to diarrhea via colonic fermentation. In addition, most calves <3 week old appear to have an allergic reaction to soy proteins that results in villous atrophy, leading to diarrhea that is probably malabsorptive (Smith, 2002).

Clinical findings :

The major signs are diarrhea, dehydration, profound weakness, and death within one to several days of onset (Radostits *et al.,* 1994). Diarrhea due to enterotoxigenic *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. Profuse amounts of liquid feces are passed, and the calves rapidly become depressed and recumbent. Calves may lose >12 per cent of body weight in fluid, and hypovolemic shock and death may occur in 12–24 hr (Smith, 2002). 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 months old and may manifest with diarrhea or primarily as dysentery with blood and mucus in the feces. The clinical course is short (Radostits *et al.,* 1994). Dietary diarrheas are seen in calves <3 week old and are characterized by voluminous feces of pasty to gelatinous consistency. Initially, the calves are bright and alert and have good appetites. Eventually, they become weak and emaciated if the diet is not corrected. Infectious forms of diarrhea are often complicated by poor-quality diets or insufficient nutritional intake (Acres, 1985).

Diagnosis :

It is difficult to make a definite etiologic diagnosis 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 (Radostits *et al.,* 1994). 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 (Acres, 1985). Complete laboratory examination can be expensive, and it has also been argued that there is little value in expending large amounts of money on diagnosis unless there is specific control procedures that can be implemented based on the information gained. In all cases, information on total milk or milk replacer consumption should be obtained. When milk replacer is being fed, the composition of the diet should be evaluated. Nonspecific immunity should be assessed by determining immunoglobulin and vitamin A concentrations in serum (Smith, 2002).

Differencial diagnosis :

Diarrhea due to *Salmonella* spp. usually is not seen in calves <14 days old. It is characterized by feces that foul smell and contain blood, fibrin and copious amounts of mucus. Septicemia, with high fever and depression progressing to prostration and coma, is the salient manifestation of salmonellosis in calves and, although diarrhea is present, death is usually from septicemic rather than from hypovolemic shock. Calves with salmonellosis usually lose weight rapidly

and often die in spite of vigorous therapy (Radostits *et al.*, 1994). Hemorrhagic enterotoxemia due to *C. perfringens* type B or C is characterized by acute onset of depression, weakness, bloody diarrhea, abdominal pain, and death within a few hours. It usually develops in vigorous calves that are just a few days old that have large appetites and a ready source of milk. Calves affected with *C.perfringens* usually die before treatment can be instituted. Cryptosporidiosis is seen in calves 5–35 days old but most commonly in the second week of life. It is characterized by persistent diarrhea that does not respond to therapy. Diarrhea due solely to *Cryptosporidium* spp. is often mild and self-limiting, although the severity may be related to the general strength of the calf and to the intensity of challenge with the organism. Combination infections with cryptosporidia, rotavirus, and corona virus are common and result in persistent diarrhea often characterized by emaciation and death. Death from hypoglycemia also occurs as a sequela of cryptosporidiosis in calves 3–4 wk of age that have recovered from diarrhea but are still emaciated. Death often occurs during a bout of cold weather and is more likely to occur on farms where there is a policy of reducing the amount of milk fed to calves during periods of diarrhea (Radostits *et al.*, 1994). Diarrhea due to rotavirus, corona virus, and other viruses usually is seen in calves 5–15 days old, but can affect calves to several months of age. Affected calves are only moderately depressed and often continue to suck or drink milk. The feces are voluminous, soft to liquid, and often contain large amounts of mucus. Diarrhea commonly persists for 3 to several days, with some cases of corona viral diarrhea becoming chronic (Smith, 2002).

Lesions :

Significant lesions may be absent, but in those which have died of colisepticaemia there may be subserous and submucosal petechial haemorrhages and enteritis. In cases of pneumonia the lungs may show areas of congestion and necrosis. The spleen and lymph nodes of mesentery are sometimes enlarged and congested, and joint infections develop as synovitis (Radostits *et al.*, 1994).

Treatment :

Many of the factors involved in disease resistance are nonspecific; thus, important preventive measures can be taken and therapy can be initiated before an etiologic diagnosis has been established. Treatment includes fluid and electrolyte replacement, alterations of the diet, antimicrobial and immunoglobulin therapy and use of antidiarrheal drugs and adsorbents (Radostits *et al*., 1994). Fluid and electrolyte therapy is most important and should be instituted as soon as possible regardless of whether clinical evidence of dehydration has developed. Calves that are still able to stand and that are willing and able to suck can often be treated with oral electrolytes alone. Fluids for oral rehydration should promote the co-transport of sodium with glucose and amino acids and should contain sodium, glucose, glycine or alanine, potassium and either bicarbonate or citrate or acetate as a bicarbonate precursor (Gyles *et al.*, 2004). These can be administered by nipple bottle or, if necessary, by stomach tube. The solutions should be used liberally until the animal is rehydrated (Smith, 2002). Whether or not milk should be fed during the rehydration period is controversial. Feeding milk may increase fecal volume, but it provides energy to the calf and may promote gut healing. Calves have large energy requirements and little reserve. Electrolyte solutions do not meet calf energy requirements and milk should not be withheld for >24–36 hr (Smith, 2002). Calves that are recumbent, weak and show evidence of water loss >8 per cent of their body weight require IV fluid and electrolyte therapy. These calves are usually acidotic, and the fluid and base deficits can be corrected initially by administering an isotonic (13 g/l) solution of sodium bicarbonate, ideally at 100 ml/kg over 4–6 hr. Because of the calves are frequently hypoglycemic, addition of 25–50 g of dextrose to the bicarbonate solution is often beneficial. The bicarbonate solution should be followed by continuous IV fluid therapy with a physiologically balanced electrolyte solution administered at 5–8 ml/kg/hr for the next 20 hr; higher rates may be necessary depending on the severity of diarrhea. Oral electrolyte solutions should probably be used concurrently with IV therapy (Hirsh *et al.*, 2004).

The use of antimicrobials is not supported by most clinical trials and not indicated in diarrhea induced by viruses or protozoa. Antibiotics may be of value in treating diarrhea associated with enterotoxigenic or attaching and effacing *E.coli*. The route of administration should be oral, and the choice based on sensitivity testing. When septicemia disease, due to inadequate transfer of colostral immunoglobulins, is suspected as a complication, parentally administered

antibiotics are also indicated. Salmonellosis should be treated with parenteral antimicrobials (Radostits *et al*., 1994). Several drugs, such as flunixin, meglumine, indomethacin, loperamide, diphenoxylate, and bismuth subsalicylate, have antisecretory and anti-inflammatory activity and are used in treatment, but there are no clinical trials of their efficacy in calves. Intestinal gels and adsorbents, such as kaolin and pectin, are in general use, but their only established effect is to increase fecal consistency; they do not reduce the loss of water and ions (Radostits *et al.,* 1994).

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 labour and general management objectives make it impossible to recommend specific management procedures that are applicable to all situations (Smith, 2002). However, 3 broad principles apply in all herds, –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; –nonspecific resistance should be maximized by providing good nutrition to the dam and neonate and assuring that newborn calves consume >5 per cent of their body weight of high-quality colostrum, preferably within 2 hr and certainly within 6 hr of birth, followed by equivalent amounts at 12hr intervals for the next 48 hr; and –the specific resistance of the newborn should be increased by vaccinating the dam or the newborn (Radostits *et al.*,1994). A significant portion of both naturally sucking dairy calves and calves handfed colostrum do not acquire adequate amounts of immunoglobulin because of delayed sucking or feeding, ingestion of an inadequate volume of colostrum, or ingestion of colostrum of inferior immunoglobulin concentration. When time constraints on labour preclude an ensured intake of colostrum by nipple-bottle feeding, administration of 3.8 l of colostrum by esophageal feeder within the first 2 hr of life can be the best colostrum feeding policy (Thomas, 2000). Immunization of calves against colibacillosis by vaccination of pregnant dams can control enterotoxigenic colibacillosis. The pregnant dam is vaccinated 6 and 2 weeks before parturition to stimulate antibodies to strains of enterotoxigenic *E.coli*; these antibodies are then passed on to the newborn through the colostrum. A single booster is given in subsequent years (Quinn *et al*., 1994).

Zoonotic risk :

Several of the agents that produce diarrhea in calves can also produce diarrheal disease in people. These organisms are commonly present as subclinical infections in the gut of calves and lambs; immune compromised people should avoid contact with young ruminants and possibly all farm animals (Radostits *et al.*, 1994). Cattle, including calves, are one of the reservoirs for the verotoxic *E.coli* serotype O157:H7 that is associated with human hemorrhagic colitis and the hemolytic uremic syndrome. Infection in people is usually acquired by consumption of contaminated food, but the infective dose is low, and the possibility of infection by direct contact exists (Smith, 2002). Other verotoxic *E.coli* associated with human disease can also be isolated from the feces of healthy cattle. Human disease from infection with enteric livestock pathogens has occurred after seemingly trivial contact associated with visits to livestock fairs, petting zoos, and farm educational tours. Hand cleansing and disinfection should be a component of these visits (Carter and Changapa, 1989).

Conclusion and recommendation :

Collibacillosis is an important disease in intensive production. There are two forms of the disease caused by different strains of *E.coli*. The systemic form is caused by invasive strains and the enteric form is caused by enterotoxigenic strains. The enteric form is the most common cause of diarrhea and death in every young calf. The main factors for the occurrence of the disease are poor hygiene, group housing, and whether stress. Treatment of enteric collibacillosis is complex involving antibiotic and fluid therapy and other nursing measures. The maxim ounce of prevention is more worth than pound of treatment. The prevention measures including good calf management, increasing non–specific resistance of the calf by appropriate colostrum feeding practice and by increasing specific resistance through vaccination of either the dam or the calf.

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