Bacterial Causes of Sudden Death in Farm Animals

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Abstract: Sudden death is a serious problem in farm animals. It may occur due to microbial or non-microbial causes. Non-microbial causes may occur sporadically due to internal hemorrhage, severe trauma, rupture of the gut. Farm animals subjected to stressful conditions are highly susceptible to microbial causes of sudden death. It may occur due to bacterial infection as *C. chauvoei* with spread of vegetative form, producing active toxin. *C. tetani* produce tetanospasmin which attack CNS causing deaths due to respiratory failure. *C. botulinum* infection end with complete loss of muscle tone. *B. anthracis* infection proceeds rapidly showing bleeding from the natural orifices after death. MRSA cause mortality in infected horses. *S.equi* infected horses show abscesses which obstruct breathing. Salmonella infection shows fever, bloody diarrhea, acute respiratory disease and deaths. Multifactorial diseases causing sudden death in newborn animals include colitis-X and calf scour. The impact of bacterial causes of sudden death in farm animals results in high economic losses due to costs of eradication, decontamination and vaccination programs. Accurate and rapid diagnosis of sudden death cases is essential for setting of control programs to improve animal husbandry.
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1. Introduction

Sudden death is a syndrome in which animals under frequent observation die, after a period of illness lasting only a few hours following the onset of non-observed clinical signs. Bacterial causes of sudden death lead to great economic losses in cattle due to medication costs, deaths and production loss. Different infectious agents may be involved in sudden death syndrome in cattle (Maas, 2008). Horses subjected to stressful conditions such as transportation, harsh environmental conditions or nutritional disorders are highly susceptible to microbial causes of sudden death (McGorum and Anderson, 2002). In farm animals, sudden death may occurs due to bacterial infection as clostridium, *B. anthracis*, *L. interrogans*, MRSA, *S. equi* and *M. paratuberculosis*, *P. multocida, P. pneumonia,* pathogenic strains of *E.coli*, *S.* Typhimurium or *Y. pestis* (Quinn *et al.,* 1994). Some multifactorial diseases may cause sudden death in young animals such as colitis-X, calf scour (Woodlcock, 1991). Sudden death may occur due to overfeeding in cattle which found dead without any signs of illness. In equine, it may occur due to septicemia caused by anthrax or toxemia due to *C. perfringens* and colibacillosis causing peracute deaths (Sting, 2009). The following review article will throw light on the bacterial causes of sudden death in farm animals.

2. Review

Sudden death in farm animals may occur due to several causes, among which, bacterial causes are the most predominant. Age, immune status, pregnancy, nutritional and managemental factors may affect the response of animal when being subjected to bacterial causes of sudden death (Andersen, 2003; Nagaraja and Titgemeyer, 2007). Bacterial causes of sudden death include Gram positive bacteria; Clostridium species, *Bacillus anthracis (B. anthracis)*, Leptospira *interrogans*, Methicillin Resistant *Staphylococcus Aureus* (MRSA) and *S. equi*. Acid fact bacilli; *Mycobacterium paratuberculosis (M. paratuberculosis)* and Gram negative bacteria; *P. multocida, P. pneumonia, E.coli*, *S.* Typhimurium and *Y. pestis* (Quinn *et al.,* 1994).

2.1. Gram Positive Bacteria

2.1.1. Clostridium

2.1.1.1. Aetiological Agent

They are Gram-positive bacteria, obligate anaerobes capable of producing endospores. Individual cells are rod-shaped or spindle. *Clostridium* consists of around 100 species that include common free-living bacteria and most important pathogens (ESR, 2009; Lewis, 2011). Species affecting farm animals are *C. botulinum*, *C. chauvoei*, *C. novyi*, *C. oedematiens*, *C. paraputrificum*, *C. perfringens*, *C. septicum*, *C. sordellii* and *C. tetani* (Ryan and Ray, 2004; Bruggemann and Gottschalk, 2009).

C. perfringens is present in nature and can be found in the intestinal tract of human, animals,

insects and in soil. It is classified into 5 toxinotypes (A, B, C, D and E) according to the production of 4 major toxins, namely alpha (CPA), beta (CPB), epsilon (ETX) and iota (Rood, 1998). *C. perfringens* can produce up to 15 toxins including lethal toxins such as perfringolysin O (PFO), enterotoxin (CPE) and beta2 toxin (CPB2) (Gkiourtzidis *et al*., 2001).

C. chauvoei is the causative agent of blackleg, a severe disease affecting cattle and sheep with high mortality rate (Sathish and Swaminathan, 2008; Vilei *et al.*, 2011).

C. novyi is found in the soil and faeces. It is pathogenic, causing a wide variety of diseases in man and animals. It comes in three types; A, B and a non-pathogenic type C distinguished by the range of toxins they produce. Some authors include *C. haemolyticum* as *C. novyi* type D (Sasaki *et al.,* 2001).

C. septicum are motile bacteria using peritrichous flagellae. They form terminal spores giving them their drumstick-like shape. Their anaerobic nature creates susceptibility in areas of decreased blood flow (Liechti *et al*., 2003). *C. septicum* produces four toxins; alpha, beta, gamma and delta which are responsible for a number of diseases; fatal braxy malignant oedema and black quarter (gas gangrene) in farm animals (Cortiñas *et al*., 1997).

C. tetani is found as spores in soil and in the gastrointestinal tract of animals. During vegetative growth, the organism cannot survive in the presence of oxygen. As the bacterium matures, it develops a terminal spore which gives the organism its characteristic appearance, resembles tennis rackets or drumsticks. *C. tetani* spores are extremely hard as they are resistant to heat and most antiseptics (Ryan and Ray, 2004; Madigan and Martinko, 2005). The spores are distributed widely in manure-treated soils (Atkinson *et al*., 2006).

C. botulinum causes botulism in equine feeding on haylage containing toxin or other forms of forage. The risk of botulism increases by changing from feeding dry hay to wet form. Horses are susceptible to botulinum toxin at two parts per trillion (Johnson *et al.,* 2010).

2.1.1.2. Susceptible Animals

Bacterial causes of sudden death in farm animals are primarily disease of herbivores which are most susceptible animals including cattle, sheep and goats, also camelids can be infected. Other animals, including horses, pigs can get infected (Radostits *et al.,* 2007).

2.1.1.3. Mode of Infection and Transmission

Ingestion of *C. chauvoei* spores is probably the most common form of exposure and infected ruminants do not directly transmit the disease to other animals. The endospores of *C. chauvoei* can lie dormant in the soil for years. Cases of blackleg often increase when animals are moved to new pastures. *C. chauvoei* spores can enter the body of an animal through skin wounds and contaminated needles or equipment. *C. tetani* usually enters a host through skin wound (Woodlcock, 1991). Most sporadic cases of botulism in cattle have been associated with poultry litter spread onto pasture. The feeding of poultry manure has caused serious losses in cattle. Bird carcasses in silage have been implicated in sporadic cases of botulism. Botulism is caused by ingestion of pre-formed toxins of *C. botulinum* where clinical disease varies from apparent sudden death to recovery after 14-21 days (Johnson *et al.,* 2010).

2.1.1.4. Pathogenicity

Clostridium organisms are normal flora of cattle and only become problematic with dietary stress, injury, changes in management, parasitism result in production of potent toxins (Popoff and Bouvet, 2009) illustrated that clostridia produce the highest number of toxins of any type of bacteria and are involved in severe diseases in animals. Most of the clostridial toxins are responsible for gangrenes and gastrointestinal diseases. Presence of the organism in the intestine is not sufficient to cause diseases. Diseases caused by clostridia in sheep include; Hemorrhagic Bowel Syndrome (HBS), abomasal ulcers and tympany, abomasitis, gas gangrene, sudden death due to enterotoxemia (Lewis, 2011).

C. perfringens is part of the normal intestinal flora. However, it causes enterotoxemia syndromes of cows and calves. The role of some of these toxins in the pathogenicity of disease has not been clarified yet. *C. perfringens* strains harboring cpb2 have frequently been associated with enterotoxaemia in sheep and goat (Uzal and Songer, 2008), in camels (Wernery and Kaaden, 2002). Infections due to *C. perfringens* show clostridial myonecrosis revealed as tissue necrosis, bacteremia, emphysematous cholecystitis and gas gangrene. The toxin involved in gas gangrene is known as α-toxin, which inserts into the plasma membrane of cells, producing gaps in the membrane that disrupt normal cellular function (Warrell *et al*., 2003). *C. perfringens* can participate in polymicrobial anaerobic infections (Brook, 2007).

After ingestion of *C. chauvoei*, they are assumed to cross over the gastro-intestinal tract, enter the bloodstream and finally migrate in various organs and muscles, where they remain dormant until stimulated to cause disease (Sathish and Swaminathan, 2008). These spores are only activated in a low oxygen environment, such as that of damaged tissue in cattle. In sheep, blackleg is frequently associated with wound, such as shearing cuts, tail docking and castration. Then the disease progresses rapidly, with the infected animal dying within 12 to 36 h after the appearance of the first symptoms (Songer, 1998).

C. septicum unlike other clostridium species not require trauma at the site of the infection. It is thought that the infection is established by hematogenous spread from the gastrointestinal tract. Gas gangrene caused by *C. septicum* is associated with colorectal cancer and other defects of the bowel (Larson *et al*., 1995). *C. septicum* causes myonecrosis through the release of exotoxins such as alpha toxin, lethal toxin and hemolytic toxin (Mahon and Manuselis, 2000). Myonecrosis is characterized by extensive tissue destruction, edema and thrombosis (Hickey *et al*., 2008).

C. tetani produces a potent biological toxin; tetanospasmin which is the causative agent of tetanus, a disease characterized by painful muscular spasms that can lead to respiratory failure and death of up to 40% of infected cases (Ryan and Ray, 2004; Atkinson *et al*., 2006). Once an infection is established, *C. tetani* produces two exotoxins, tetanolysin and tetanospasmin. Tetanolysin serves no known benefit to *C. tetani*. Tetanospasmin is a neurotoxin that causes the clinical manifestations of tetanus. Tetanus toxin is generated in living bacteria and is released when the bacteria lyse (Ken, 2005). Tetanospasmin is one of the most potent toxins known. It is released in the wound and absorbed into the circulation and then it reaches the ends of motor neurons all over the body. The toxin acts at several sites within the central nervous system, including nerve terminals, spinal cord and brain and within the sympathetic nervous system (Atkinson *et al*., 2006).

C. botulinum exists in high numbers in soils where cattle graze, they are ingested while eating or enter through a wound and all of them produce toxins that kill cattle very rapidly (Quinn *et al.,* 2002).

C. novyi primary infection is intestinal and transferred by the faecal-oral route. Spores of *C. novyi* escape from the gut and lodge in the liver where they remain dormant until some injury creates anaerobic conditions for them to germinate causing local necrosis and widespread damage to the microvascular system resulting in subcutaneous bleeding and blackening of the skin, hence the common name, "black disease" (Quinn *et al.,* 2002).

2.1.1.5. Clinical Sign

Clostridial diseases progress rapidly and sudden death is often the first and the only sign of disease. *C. perfringens* type C causes necrotic enteritis in newborn calves. Calves are suddenly depressed, weak, may be distended or show abdominal pain. If diarrhea develops, it may have blood and tissue streaks. Affected calves may die before they develop diarrhea (Quinn *et al.,* 2002).

C. perfringens type D produces the classic overeating disease, a syndrome more important in sheep and goats called "overeating disease" or "pulpy kidney disease") than in calves (Wells and Wilkins, 1996). The disease is characterized by sudden death of well-fed calves. Other affected calves may show neurologic, signs, incoordination, trembling and recumbent with head back or convulsing with focal symmetrical encephalomalacia in sheep and goat (Uzal and Songer, 2008).*C. perfringens* type E represents 50% of enteritis in neonatal calves (Songer and Miskimmins, 2004).

C. chauvoei affected cattle are often found dead. Peracute evolution with severe edematous lesions restricted to the local area, fever (>41°C), loss of appetite, lameness and depression observed in blackleg outbreaks, virulence of *C. chauvoei* is caused by rapid spread of the activated, vegetative form of the bacterium in the infected tissue followed by production of potent toxins (Useh *et al*., 2003).

C. novyi infection leads to sudden death as they produce Alpha-toxin is characterized as lethal, necrotizing and oedematising. It causes morphological changes to all cell types especially endothelial cells resulting in breakdown of cytoskeletal structures (Müller *et al*., 1992). The cells of the microvascular system become spherical and the attachments to neighbouring cells are reduced to thin strings. This results in leakage from the capillaries, leading to oedema. Beta-Toxin and Gamma-Toxin are characterized as haemolytic, necrotizing lecithinase. Delta-Toxin and zeta-Toxin are characterized as oxygen labile haemolysin. Epsilon-Toxin characterized as lecithino-vitelin, responsible for pearly layer in cultures (Schmidt *et al*., 1996).

C. tetani infection is followed by production of toxin which attacks the nervous system. Clinical signs are most frequent following puncture wounds or infection of the castration site or contamination of the surgical site. Signs include muscle spasms, rigid limbs, extreme sensitivity to touch and sound and death within 3-10 days (Wells and Wilkins, 1996). Affected calves show hind leg stiffness and difficulty walking. They have bulging eyes with the ears held backwards and nostrils flared. The animal is unable to open its mouth "lock jaw" and there is moderate bloat. Very often an infected animal will show a raised tail head. Despite treatment, in some cattle the condition progresses over two to five days such that the animal is unable to raise itself. Seizure occurs, at first in response to loud noises then occurs spontaneously. Death occurred from respiratory failure (Radostits *et al.,* 2007).

C. botulinum are confined to the nervous system with muscle weakness affecting the hind legs, weakness progresses over four to seven days to involve the forelegs, head and neck. Affected cattle have difficulty chewing and swallowing and there is paralysis of the tongue. Involvement of respiratory

muscles and diaphragm causes death. Cattle botulism show complete loss of muscle tone, head is averted against the chest. In Equine, *C. botulinum* is characterized by muscle weakness, tremors, difficulty swallowing, drooping eyelids and dilation of the pupils, occasionally leading to recumbency and death due to cardiac and respiratory failure (Radostits *et al.,* 2007).

2.1.1.6. Post Mortem Examination

Enterotoxemia associated with CPB2-positive *C. perfringens* in goat kid show hemorrhagic enteritis, congested and edematous small and large intestine distended with gases and hemorrhagic fluid. Dark red necrotic mucosa and large amount of fibrin present in the lumen. Heart show, pericardial sac with a large amount of pericardial fluid and fibrin strands. *C. perfringens* type D show signs of interstitial lung edema with widened interlobular septa and large amount of froth in the airways. Brain show cerebellar vermis hernia, extends backward into the foramen magnum of the occipital bone, Focal symmetrical encephalomalacia in the brain (Quinn *et al.,* 1994).

Blackleg caused by *C. chauvoei* affecting muscles of the neck and hind leg with extensive swelling and black muscle in ruminant (Useh *et al.*, 2003).

C. novyi infection cause necrotic hepatitis, especially in sheep. The disease is associated with liver fluke results from interaction of *C. novyi* and *Fasciola hepatica*. Sheep die from infectious necrotic hepatitis. Peritoneal, thoracic cavities and pericardial sac contain a great quantity of fluids. The liver is swollen with perihepatitis and necrotic foci. Hepatic lymph nodes are enlarged. The gall bladder is full. Subcutaneous tissues are full of cyanotic venous blood. They may darken the hide, hence the name “black disease” (Ken, 2005).

2.1.1.7. Diagnosis

Diagnosis of sudden death in farm animals should include case history, clinical signs, postmortem examination as well as histopatholodical changes. Lab diagnosis is required for confirmation; samples should be collected into transport media then incubated at the conditions best for isolation of suspected bacteria, then Gram stained followed by biochemical identification are essential for traditional diagnosis (Quinn *et al.,* 1994).

For clostridia, samples should be collected in cooked meat broth or thioglycollate broth media then anaerobically incubated. Gram staining illustrated Gram positive spore forming rods. Biochemical analysis is required to differentiate between different species, such as cultivation onto Egg Yolk agar for lecithinase and lipase activity, testing for hydrolysis of gelatin, digestion of casein, Indole production, formation of acid from glucose-lactose-sucrose-maltose fermentation test. Animal inoculation test by I/M injection in mice or guinea pig for toxin identification by neutralization test using polyvalent antitoxin, followed by specific monovalent antitoxin. *C. perfringens* is also identified by Nagler test and CAMP test. Florescent antibody technique for identification of *C. novyi* in acetone fixed liver impression smear (Quinn *et al.,* 1994). *C. perfringens* toxins were detected using quantitative culture followed by genotyping. Toxin detection can be performed by several techniques including an enzyme-linked immunosorbent assay (ELISA) that detects CPA, CPB, ETX and *C. perfringens* (Uzal and Songer, 2008). Multiplex PCR assay provide a useful alternative to *in vivo* toxin neutralization tests for typing of *C. perfringens* isolates (Meer and Songer, 1997). The presence of the toxin in affected tissue suggest an association between the beta2 toxin and the post-mortem findings has been demonstrated by immunohistochemistry in horses that died from typhlocolitis (Vilei *et al*., 2005).

C. chauvoei strains grow anaerobically on Tryptic Soy Agar (TSA) medium containing 5% sheep erythrocytes or in Tryptic soy broth with 2% glucose, 1% yeast extract and 0.1% L-cysteine at 37°C for up to 72 hrs. DNA sequences of *C. chauvoei* have been deposited in GenBank databases including the 16S and the 23S rRNA genes and the flagellin gene (Sasaki *et al.*, 2002). Sialidase activity and cloning of the NanA protein of *C. chauvoei* were analyzed (Vilei *et al.*, 2011). Thirty four isolates were identified by conventional techniques and 16S rRNA gene sequencing using restriction endonuclease analysis and protein analysis showing production of alpha and beta toxins (Sathish and Swaminathan, 2008).

Diagnosis of *C. tetani* depends on the history of previous wound or bite as predisposing cause. Clinical signs found on infected animal and Gram stain smear from deep wound for confirmation (Quinn *et al.,* 1994).

C. botulinum accurate diagnosis requires knowing history about the type of feedstuff and the clinical signs as well as demonstration of the toxin in serum by mouse inoculation test or ELISA (Quinn *et al.,* 1994).

2.1.1.8. Prevention and Control

Intensive care with antitoxin, fluids, antibiotics and anti-inflammatory drugs is necessary for treatment but frequently unsuccessful (Quinn *et al.,* 2002). Synergism between proplis and antibiotics for treatment of enterotoxaemia in calves due to *C. perfringens* type A and C show great results (Masoud *et al.,* 2008). Commercial toxoids available for vaccination against *C. perfringens*, is not effective against type E infections (Songer and Miskimmins, 2004). Clostridia mode of action is to produce one or more potent toxins. Therefore, the best program is obtained by the use of toxoid vaccines, it allow

protection to pass to the lamb via the colostrum (Lewis, 2011).

C. chauvoei could be treated with penicillin (44,000 IU kg^{-1}) but it is unlikely to be effective unless started in the very early stages. When blackleg is a problem on the farm vaccination is essential. The best protection is a two-dose course followed by annual revaccination (Hirsh and Zee, 1999).

There is no treatment for *C. novyi*. An appropriate fluke control plan combined with an appropriate clostridial vaccination programme to prevent black disease (Hirsh and Zee, 1999).

Immunity to these diseases is mediated mainly by antitoxin raised against *C. septicum* alpha toxin. In addition, the immunogenicity of native toxic filtrates is weak, which results in poor antibody response in animals (Cortiñas *et al*., 1997).

There is no effective treatment for *C. botulinum*. Disease can be controlled by specific vaccination, but it is not included in standard multivalent clostridial vaccines. Vaccination for clostridial diseases has been proven to be extremely effective. In areas of high exposure, young calves should be vaccinated around 60 days and the vaccination repeated after the calves are four months old. In areas of low incidence, vaccination when calves are four to six months is usually effective Alum and gel adjuvenated vaccine gave satisfactory antibody titres in rabbit, sheep and cattle which were higher than the minimum protective levels of both improved polyvalent clostridial vaccine (Abdalla *et al.,* 2011).

Vaccinate equine for botulism, before feeding horses haylage/silage. The vaccine only protects against type B, which is the most common type. Avoid feeding on hay fields fertilized with poultry manure and increase moisture levels (16 - 30%) (Hirsh and Zee, 1999).

2.1.2. Bacillus Anthracis

2.1.2.1. Aetiological Agent

B. anthracis is an aerobic, spore-forming bacillus, found in two forms; vegetative form and spore form. It releases highly resistant spores, which contaminate the environment and cause spreading of the disease. Anthrax causes sudden death in cattle, sheep and goats and can severely affect humans. The organism is a common inhabitant in alkaline soils. It can survive in wet and in dry conditions; it forms spores, which are very resistant to environmental conditions. The spores can survive for more than 50 years in the environment, waiting for the next favorable opportunity to multiply once again. Most cases occur from July to September, during warm, dry conditions (Sun *et al.,* 2012).

2.1.2.2. Mode of Infection

Most animals get anthrax disease orally through soil contaminated with anthrax spores while grazing of abrasive forages, which allow penetration of the spores through the lining of the mouth, then anthrax enters blood stream and multiplies followed by fatal infection that occurs rapidly. Carnivores can get the disease by eating animals infected with anthrax (Pilo and Frey, 2011).

2.1.2.3 Pathogenicity

B. anthracis is an obligate pathogen that multiplies only in animals and if an infected carcass is opened, it sporulates resulting in contamination of soil. In unopened carcasses the organism does not sporulate and is destroyed by putrefaction. The disease is not directly transmissible from animal to animal and infection is associated with ingestion of soil or infected material contaminated with spores. Blowflies may be important in the spread of the disease after feeding on infected carcasses (De Vos and Turnbull, 2004). The incubation period ranges from 1-14 days. In the peracute form, animals may die without showing signs or may die in 1-3 days after developing subcutaneous swellings on various parts of the body (Fowler, 1998). Anthrax lethal toxin has specific proteolytic activity that causes the disruption of intestinal epithelial integrity, characterized by mucosal erosion, ulceration and bleeding (Sun *et al.,* 2012).

The poly-D-glutamyl capsule is itself nontoxic, but functions to protect the organism against bactericidal components of serum and against phagocytic engulfment and destruction. Anthrax toxin is a diffusible exotoxin plays a major role in the pathogenesis of anthrax. It has a lethal mode of the action that is not entirely understood at this time. Death is apparently due to oxygen depletion, secondary shock, increased vascular permeability, respiratory failure and cardiac failure. Formation of a poly-D-glutamyl capsule, mediates the invasive stage of the infection mediates the toxigenic stage. All virulent strains form a single antigenic capsule of a poly-D-glutamate polypeptide. Production of capsular material is associated with the formation of a characteristic mucoid or "smooth" colony type. “Rough” (R) colonial variants are relatively avirulent (Fowler, 1998).

2.1.2.4. Clinical Sign

When cattle become infected with *B.anthracis*, the disease usually proceeds rapidly. Most often, the cattle appear to be normal one day and are found dead the next. The most common sign of anthrax infection in animals is sudden death. Prior to death, animals may have a fever, muscle tremors, staggering, trembling, convulsions and difficult breathing. There may be bleeding from the mouth, nose and anus after death (Pipken, 2002).

2.1.2.5. Post Mortem Examination

No post mortem examination should be carried out in Anthrax suspected cases (Quinn *et al.,* 1994).

2.1.2.6. Diagnosis

Anthrax diagnosis requires collection of blood sample from the jugular vein, ear or eye. It is important not to open the carcass. The anthrax organism will die out in an unopened carcass in a few days. If the carcass is opened to the air, billions of spores will be formed and survive in the environment for decades. Incubation requires 5% CO_2 enhancing production of the poly-D-glutamyl capsule and accounts for the mucoid colony type. It is non hemolytic on blood agar (Quinn *et al.,* 1994).

2.1.2.7. Prevention and Control

Anthrax is a treatable disease if diagnosed early. Penicillin and oxytetracycline are effective antibiotic treatments for cattle. There is a vaccine available for livestock and vaccination is effective if used 2-4 weeks before the season when outbreaks are expected; particularly in the summer and fall months. Anthrax vaccine for use in livestock has been available for more than 40 years. It is made from a nonpathogenic strain of the organism. Efficient live spore vaccines are available for Control of the Disease (CDC, 2006). The vaccine strain developed by (Sterne, 1937) is used for most animals including camelids. It is a rough strain that has lost plasmid pX02 which codes for the bacterial capsule. The vaccine is non-pathogenic in most animal species and provides good immunity (De Vos and Turnbull, 2004). The live cattle at risk will be moved away from the suspected area of spore contamination. They will be fed hay or put onto irrigated pasture. Dead cattle are buried and covered with quicklime. Quarantine and disinfection of infected areas and vaccination of animals should be carried out in an outbreak. Anthrax should be a differential diagnosis for any case of sudden death in endemic areas. Cases of anthrax are reported worldwide. Anthrax spores are quite resistant to disinfectants, so bedding and in-contact materials should be considered infectious and burned. Articles or instruments that cannot be burned should be soaked with 5-10% bleach solution (CDC, 2006).

2.1.3. *Leptospira Interrogans*

2.1.3.1. Aetiological Agent

Leptospira cause great economic losses as a result of calf mortality (Ismail *et al.,* 2006). Leptospirosis is a bacterial disease of a number of species caused by serovars of *L. interrogans*. It is prevalent worldwide (Levett, 2001). In Egypt the incidence reaches 19.05 and 12.5% for *L. interrogans* serovars *Icterohaeorrhagiae* and it reaches 9.52 and 12.5% for *L. interrogans* serovar *Pomona* in cattle and buffaloes, respectively. This may be attributed to the widespread of wild rats in the area from which animals were under examination (Hassan, 2007). A surveys in Europe recorded prevalence in cattle of 10.4% (Espi *et al.,* 2000).

2.1.3.2. Mode of Infection

Leptospirosis can be readily transmitted between species through grazing in a field contaminated with infected urine, contaminated soil or water or other body fluids (Barwick *et al*., 1998).

2.1.3.3. Clinical Sign

Uveitis is the most frequently encountered clinical manifestation of leptospirosis in horses; however, abortion and stillbirth are serious problems (Faber *et al*., 2000). Clinical picture shows renal dysfunction in a stallion, neonatal mortality as well as fever, jaundice, anorexia and lethargy (Divers *et al*., 1992).

2.1.3.4. Diagnosis

Diagnosis of leptospirosis can be difficult and may involve antigen detection (PCR), serological evaluation, histological examination, culture and dark field microscopy (Dehkordi *et al.,* 2011; Toyokawa *et al.,* 2011).

2.1.4. Methicillin-resistant *Staphylococcus aureus*

2.1.4.1. Aetiological Agent

Methicillin-resistant strains of *Staphylococcus Aureus* (MRSA) are nosocomial pathogens of serious concern because of their antimicrobial resistance (Hartstein *et al*., 1995). MRSA infection has been reported in hospitalized horses (Seguin *et al*., 1999; Omer *et al.,* 2008). It causes mortality rate reach 18.83% in Egyptian horses (Abd El-Moez *et al.,* 2009).

2.1.4.2. Mode of Infection

MRSA is the causative agent of nosocomial infection in stressful horse (Abd El-Moez *et al.,* 2009).

2.1.4.3. Pathogenicity

MRSA is an opportunistic pathogen. It is a nosocomial disease that cause severe infection in equine subjected to multiple stressful condition. Severity of the condition is related to massive misuse of antibiotics which cause immunosuppression of the affected horses and causes toxic shock syndrome (Abd El-Moez *et al.,* 2009).

2.1.4.4. Clinical Sign

MRSA is an emergency infection in horses (Hartmann *et al.,* 1997). Infected cases show acute watery diarrhea, severe colic, sweating, tremors followed by death. However, the animals were treated with multiple broad spectrum antibiotics. The outbreak occurred in Arabian and foreign breed equine farm with a mortality rate reaches 18.82%. (Abd El-Moez *et al*., 2009).

2.1.4.5. Post Mortem Examination

MRSA in equine show generalized toxemia in all vital organs illustrated in the form of severe congestion, pneumonia, endocarditis, gastroenteritis and nephritis. Post mortem finding and histopathological findings show severe congestion and hemorrhages in the intestine, caecum and heart with severe degenerations and interstitial hemorrhage in the kidney tissue. Lung tissue show alveolar emphysema,

interstitial edema and hemorrhage Abd El-Moez *et al.* (2009).

2.1.4.6. Diagnosis

Accurate diagnosis of MRSA requires isolation of the pathogen on Nutrient agar and Mannitol salt agar and identification using Gram staining, biochemical identification including coagulase test, DNase test and using commercial biochemical analysis kits (API Staph-Ident), Antibiotic sensitivity test and phage typing. Bacteriological swabs examination and internal tissue of dead horses revealed isolation and complete identification of *S. aureus* from all cases. Antibiotic sensitivity test of isolates show 100% resistance of all isolates to all tested antibiotics with an exception for vancomycin which was successfully used for treatment and control the progress of cases in the farm (Abd El-Moez *et al*., 2009).

Molecular analysis of the isolates proved to be an accurate tool as a quantitative-PCR analysis for detection of *mec-A* gene, to confirm it's accusation as a direct cause of the fatal toxic shock syndrome in the equine subjected to multiple stressful conditions. Case history of excessive nonspecific antibiotics treatment helps in propagation of opportunistic multiple drug resistant *S. aureus* which release enterotoxins leading to toxic shock syndrome that end fatally. *mec-A* gene responsible for drug resistance of MRSA strains was detected in all isolates isolated from the equine outbreak in equine subjected to stressful conditions and showing symptoms of generalized toxemia (Abd El-Moez *et al*., 2011).

2.1.4.7. Prevention and Control

Vancomycin is the drug of choice for treatment of infected horses together with good nursing and fluid therapy (Abd El-Moez *et al*., 2009). *In vitro* studies proved that *L. acidophilus* isolated from healthy mare and goat colostrum showed great hindrance capabilities against MRSA (Abd El-Moez *et al*., 2011).

2.1.5. *Streptococcus equi*

2.1.5.1. Aetiological Agent

Strangles is a highly contagious and serious infection of horses caused by *Streptococcus equi* subspecies *equi*, known as *S. equi*. They are Gram positive cocci arranged in the form of chain (Radostits *et al.,* 2007).

2.1.5.2. Mode of Infection

Direct contact with horse that is incubating strangles or has just recovered from the infection, or with an apparently clinically unaffected long-term carrier. Indirect contact occurs when an animal comes in contact with a contaminated stable (buckets, feed, walls, doors) or pasture environment (grass, fences, but almost always the water troughs), or through flies (Timoney, 1993).

2.1.5.3. Pathogenicity

Strangles is most common in animals less than 5 years of age and especially in groups of weanling foals. Foals under 4 months of age are usually protected by colostrum-derived passive immunity. Horses of all ages are susceptible (Timoney, 1999).

2.1.5.4. Clinical Sign

S.equi infected horses show typical generalized signs of infection (depression, inappetence and fever of 39-39.5°C). Horses develop nasal discharge (initially mucoid, rapidly thickening and purulent), soft cough and slight swelling between the mandibles but very painful, with swelling of the submandibular lymph node. With the progression of the disease, abscesses develop in the submandibular and retropharyngeal lymph nodes. The lymph nodes become hard and very painful and may obstruct breathing ("strangles") (Timoney, 1993).

2.1.5.5. Post Mortem Examination

Classic strangles is a severe infection that can be fatal, abdominal or lung lymph nodes may develop abscesses and rupture, brain abscess may rupture causing sudden death or a retropharyngeal lymph node abscess may burst in the throat and the pus will be inhaled into the lung. Purpura haemorrhagica, which is an immune-mediated acute inflammation of peripheral blood vessels occur in infected equines (Newton *et al*., 1997).

2.1.5.6. Diagnosis

Diagnosis of strangles can be confirmed by culturing pus from the nose, abscessated lymph nodes or from the throat of clinically affected horses. Although *S. equi* isolates are thought to be genetically identical, isolates may vary in virulence (Quinn *et al.,* 1994).

2.1.5.7. Prevention and Control

Treatment of a horse in the early stages of strangles is usually effective using penicillin G (Radostits *et al.,* 2007).

2.2. Acid Fast Bacilli

2.2.1. *M. paratuberculosis*

2.2.1.1. Aetiological Agent

Mycobacterium avium subspecies *paratuberculosis* causes Johne's disease in cattle, sheep and goats worldwide. It is an acid fast bacilli stained with Ziehl-Neelsen stain (Al Hajri and Alluwaimi, 2007).

2.2.1.2. Mode of Infection

M. paratuberculosis could be transmitted by ingestion of fecal material, milk or colostrum is the main route of infection. Infected cattle shed low amount of bacteria during the subclinical stage. However, during the clinical stage the shaded organisms in feces increase dramatically (Quinn *et al.,* 2002).

2.2.1.3. Clinical Sign

M. paratuberculosis in camels causes characteristic clinical illness of severe diarrhoea ending in death

(Wernery and Kaaden, 2002). The course of disease is often rapid in camel (Higgins, 1986).

2.2.1.4. Diagnosis

Jhone's disease diagnosis requires cultivation of *M. paratuberculosis* on Horrold's egg media which need up to 16 weeks incubation. ZN stain of rectal scrap or ileocecal valve mucosal smears show acid fast bacilli (Quinn *et al.,* 1994). PCR is effective only as supportive test to the ELISA due to the great variations in the pattern of *M. paratuberculosis* shedding. Attempt to isolate camel *M. paratuberculosis* for its genotyping was not successful despite the incubation for more than 10 weeks (Alhebabi and Alluwaim, 2010).

2.3. Gram Negative Bacilli

2.3.1. *Salmonella* Typhimurium

2.3.1.1. Aetiological Agent

Salmonella is a Gram negative bacterium that can survive and multiply in the environment as a result of fecal shedding. There are approximately 2,500 known serovars in the *Salmonella* genus (Davies, 2008). The most common serotype isolated is *S*.Typhimurium (ESR, 2009). *S*. Typhimurium DT104 exhibits multiple resistances to the commonly used antibiotics (Jones *et al*., 2002).

2.3.1.2. Mode of Infection

Salmonella spp. infection occurs when a susceptible animal ingest feed or water that has been contaminated with feces from animals shedding the organism. Sources of infection may be rodents, birds, flies, feral cats, dogs. The principal route of infection is fecal-oral. Saliva, nasal secretions, colostrums and milk can also be the source of organisms shed from sick animals and oral transmission from these sources is another way to spread disease on a dairy. Cow to cow transmission is typical but other animals, insects, birds, people, vehicles, medications, equipment and utensils can facilitate transmission of the organisms. It is also possible that aerosol transmission of *Salmonella* may occur under some types of housing conditions (Quinn *et al.,* 1994).

2.3.1.3. Pathogenicity

In cattle, enteric salmonellosis is a very common. Various stress factors influence the outcome of infection (Fenwick and Collett, 2004). After oral infection, salmonellae colonise the distal ileum. Initial infection may be followed by bacteraemia and dissemination to several organs. In pregnant animals, abortion may occur. Animals that recover from *Salmonella* infections may become carriers for life, shedding organisms sporadically in their feaces (Radostits *et al.,* 2007).

2.3.1.4. Clinical Sign

Salmonellosis has a wide spectrum of manifestations in bovine. Asymptomatic, mild clinical bacteremia/septicemia and endotoxemic infections can occur. The manifestations vary with virulence of the strain, infectious dose, age of the animal and immunity of the host. Some dairies first experience salmonellosis as an outbreak of high fevers, bloody diarrhea, acute respiratory disease and deaths. 56% of 16 herds tested had at least one cow with a positive manure culture and about 10% of almost 1000 samples cultured were positive. Almost 90% of dairies had at least one positive cow (Fossler *et al*., 2005). There is little specific information about *Salmonella* infections in camelids (Cebra *et al*., 2003).The first report of enteric salmonellosis in camelids appeared in 2004, a case of suppurative hepatitis in an alpaca associated with *S.* Typhimurium (Saulez *et al*., 2004). Other forms of salmonellosis in camelids include meningitis in a newborn alpaca (D'Alterio *et al*., 2003; Hamouda *et al.,* 2010) and two cases of septicaemic salmonellosis caused by *S.* Choleraesuis and *S.* Typhimurium (Anderson *et al*., 1995). Virulent, multi-drug resistant strains of *S.* Typhimurium DT104 infect horses (Weese *et al*., 2001). This multi-drug resistant bacteria cause high mortality, acute toxic enterocolitis, fever, chronic diarrhea or septicemia in human and farm animals (Fone and Barker, 1994). Horses of all ages can be affected especially young foals showing signs of aneroxia, fever profuse diarrhea and death within 24-48 hrs. Salmonellosis more commonly occurs following antimicrobial therapy, hospitalization, stressors such as shipping or training (El-Bialy and Ahmes, 2008).

2.3.1.5. Diagnosis

Accurate diagnosis of salmonella require cultivation on specific media; XLD, RVS, Brilliant green agar, Triple sugar iron agar, Gram stain show medium sized Gram negative rods. Biochemical tests using API 20. Serotyping using slide agglutination test and antibiotic sensitivity test for detection of R factor plasmid (Quinn *et al.,* 1994).Carriers of infections can be detected by culturing feaces but, because excretion is intermittent, repeated sampling and culture may be necessary. Serology may be useful but is best applied on a herd basis (Veling *et al*., 2002; Davies, 2008). No practical serological method exists for detecting individual carrier animals (Hansen *et al*., 2006). The use of pooled Salmonella enrichment broth cultures of bovine feces and Polymerase Chain Reaction (PCR) for the detection of the invA gene of Salmonella in feces appears to be an efficient method of Salmonella detection (Singer *et al*., 2006). Diagnosis of salmonellosis involves isolation of *Salmonella* sp. fecal cultures should be submitted for *Salmonella* culture in all cases of diarrhea or fever. Because *Salmonella* can be shed intermittently, five negative cultures must be obtained before ruling out salmonellosis. Salmonella Typhimurium recovered from calves and lambs were tested for their virulence using Congo red binding test,

ability to produce hemolysin, adherence assay and HEp2 cell invasion test and detection of inv Agene using PCR (Mohamed and Dapgh, 2007).

2.3.1.6. Prevention and Control

In vitro hindrance of *S.* Typhimurium was sufficiently carried out using ciprofloxacin in camel (Molia *et al.,* 2004) and in poultry (Abd El-Moez *et al*., 2010) also it is highly sensitive to ciprofloxacin coated with gold nanoparticles (Zawrah and Abd El-Moez, 2011). Traditional *S.* Typhimurium and Dublin bacterins were not very effective in preventing infections. Safe and effective vaccines provided Gram negative core antigen vaccines that could be relied upon to attenuate the severity of clinical salmonellosis in adult cows and calves that received colostrum from vaccinated donors. A live vaccine, Entervene-D emerged as an effective way to control *Salmonella* Dublin in young calves but side-effects in young animals is always a potential problem. Recently, *Salmonella* vaccine from Agri-Labs, provide a safe and effective way to block division and survival *of Salmonella* in its host. The vaccine reduced shedding of *Salmonella* in a field trial and appears to be cross-protective against many strains (Fossler *et al*., 2005).

2.3.2. *Pasteurella multocida*

2.3.2.1. Aetiological Agent

Pasteurella is a genus of Gram-negative, facultative anaerobic bacteria, non-motile and pleomorphic. Most species are catalase-positive and oxidase-positive (Kuhnert and Christensen, 2008). *Pasteurella multocida* strains are classified into five capsular antigen types (A, B, D, E and F) and 16 somatic antigen types (De Alwis, 1999). Capsular typing is done by various methods. Namioka-Carter classifications type the causes of haemorrhagic septicaemia by 6.B and 6.E. In the Heddleston-Carter system these strains are B_2 (Asia) and E_2 (Africa) (Srivastava *et al*., 2008).

2.3.2.2. Mode of Infection

Animals that survive infection with *P. multocida* may be active carriers for 4-6 weeks and then become latent carriers with the organism being harboured in the nasopharynx, retropharyngeal lymph nodes and tonsils, from which it is periodically shed when the animal is stressed (Bastianello and Henton, 2004). The organism is excreted in respiratory aerosols, saliva, urine, faeces and milk. Transmission is by the respiratory route or on fomites (Srivastava *et al*., 2008).

2.3.2.3. Pathogenicity

Haemorrhagic septicaemia is predominantly a disease of cattle and buffaloes, in tropical and sub-tropical countries of Asia and Africa (OIE, 2009c). In Africa, it is caused by *Pasteurella multocida* types B and E and in Asia by type B (Bastianello and Henton, 2004). Some cases of natural infection have been described in camel by Werney and Kaaden, (2002).

2.3.2.4. Clinical Sign

P. multocida incubation period in naturally acquired infections is from 1-3 days. The course usually varies from peracute to subacute. Peracute infections are characterized by sudden death, while acute cases show fever, profuse salivation, nasal discharge and rapid respiration. Firm subcutaneous swellings in the submandibular region are seen in subacute cases. Untreated cases usually end fatally (Bastianello and Henton, 2004).

2.3.2.5. Diagnosis

P. multocida isolation requires repeated culturing of tonsillar swabs (OIE, 2009b). Animals become septicaemic a few hours before death and culture from blood is possible only in this period. 6B and 6E strains produce hyaluronidase and can be identified by various PCR methods as well as serological methods using the indirect haemagglutination test; high antibody titers indicate recent infection (Srivastava *et al*., 2008). Accurate diagnosis should be built on history of endemic area, isolation of the pathogen from heart blood, liver, spleen or lymph node followed by serotype identification (Quinn *et al.,* 1994).

2.3.2.6. Prevention and Control

P. multocida resistance to antibiotics has not been described. Treatment with sulphonamides is effective in controlling outbreaks of the haemorrhagic septicemia (Bastianello and Henton, 2004). Both live and dead vaccines have been used. Although vaccination reduces mortality, there is no evidence that suggests it could be used in camelids effectively (De Alwis, 1999).

2.3.3. *Yersinia pestis*

2.3.3.1. Aetiological Agent

Yersinia pestis is a Gram-negative bacterium causing plague resulted in millions of deaths. Modern treatment has reduced the threat but the World Health Organization still reports 1,000 to 3,000 cases of plague annually (CDC, 2009a).

2.3.3.2. Mode of Infection

Plague is primarily transmitted by fleas from rodent hosts, particularly rats and wild rodents. It can also be transmitted by bites and scratches from infected animals or by the respiratory route in cases of pneumonic plague. It is carried by a large number of rodents; about 200 species of rodents had been proved to be naturally infected (Davis *et al.,* 1975). In a plague outbreak, deaths that occurred in dromedary camels were associated with transmission by fleas. The disease was transmitted to humans that ate raw liver from a camel that had died of the infection (Bin Saeed *et al*., 2005). Camelids are not considered to be maintenance hosts for *Y. pestis* (Orloski and Lathrop, 2003).

2.3.3.3. Diagnosis

Y. pestis grow on Nutrient, blood and MacConkey agar as well as Yersinia selective media; CIN agar containing antibiotic supplement cefsulodin (15 µg/litre), irgasin (4 µg/litre) and novobiocin (2.5 µg/litre) with incubation at 22-25°C. Gram stain show small sized Gram negative rods. Biochemical tests using API 20 (Quinn *et al.,* 1994).

(IV) Multi-factorial causes of sudden death in newborn animals.

2.4. Multifactorial causes

2.4.1. Colitis -X

2.4.1.1. Aetiological Agent

Colitis -X causative factor has not been verified and the disease has been attributed by various sources to viruses, parasites, bacteria, use of antibiotics and sulfonamides and heavy metal poisoning (Diakakis, 2008; MVM, 2008). Other possible causes include peracute salmonellosis, clostridial enterocolitis and endotoxemia. Excess protein and lack of cellulose content in the diet is thought to be the trigger for the multiplication of *clostridial* organisms. A similar condition may be seen after administration of tetracycline or lincomycin to horses (MVM, 2008). Changes in the microflora of the cecum and colon that lower the number of anaerobic bacteria, increase the number of Gram-negative enteric bacteria and decrease anaerobic fermentation of soluble carbohydrates resulting in damage to the cecal and colonic mucosa and allowing increased absorption of endotoxins from the lumen of the gut (Srivastava, 2010).

2.4.1.2. Pathogenesis

Colitis X, equine colitis X or peracute toxemic colitis is a various fatal forms of acute or peracute colitis in horses, clinical signs include sudden onset of severe diarrhea, abdominal pain, shock and dehydration. Death is common, with 90 to 100% mortality, usually in less than 24 hours. The causative factor may be *C. difficile*. Horses under stress appear to be more susceptible to developing colitis X, there is an association with prior antibiotic use (Diakakis, 2008). Miss use of antibiotics cause progress of severity of infection in horses (Abd El-Moez *et al*., 2009).

2.4.1.3. Clinical Signs

Colitis-X is a term used for colitis cases in which no definitive diagnosis can be made and the horse dies (MVM, 2008) Clinical signs include sudden, watery diarrhea that is usually accompanied by symptoms of hypovolemic shock and usually leads to death in 3 to 48 hours, usually in less than 24 hours. Other clinical signs include tachycardia, tachypnea and weak pulse. Marked depression is present. An explosive diarrhea develops, resulting in extreme dehydration. Hypovolemic and endotoxic shock are manifest by increased capillary refill time, congested or cyanotic mucous membranes and cold extremities (Diakakis, 2008). Clinical signs are similar to those of other diarrheal diseases, including toxemia caused by *Clostridium*, Potomac horse fever, endotoxic shock and anaphylaxis (MVM, 2008).

2.4.1.4. Diagnosis

Diagnosis of multifactorial sudden death cases depend on case history, clinical picture, postmortem examination and lab diagnosis. Cases of colitis X necropsy show, edema and hemorrhage in the wall of the large colon and cecum and the intestinal contents are fluid and often blood-stained (MVM, 2008). Macroscopic and microscopic findings include signs of disseminated intravascular coagulation, necrosis of colonic mucosa and presence of large numbers of bacteria in parts of the intestine. Histopathological findings in horses show that the mucosa of the large colon is hemorrhagic, necrotic and covered with fibrohemorrhagic exudate, while the submucosa, the muscular tunic and the local lymph nodes are edematous (Diakakis, 2008). Accurate diagnosis requires relating the clinical signs with the postmortem findings and isolation of the causative agents as well as differential diagnosis from salmonella infection and tetracycline therapy (Quinn *et al.,* 1994).

2.4.1.5. Prevention and Control

Treatment for colitis-X usually does not save the horse. The prognosis is average to poor and mortality is 90 to 100% (MVM, 2008). Mortality rate has been fallen to 75% by preventing dehydration by treatment using fluids, electrolytes, blood plasma, anti-inflammatory, analgesic drugs and antibiotics, good nutrition is also important; either parenteral or normal feeding can be used to support sick horse. Finally, the use of probiotics is considered beneficial in the restoration of the normal intestinal flora. The probiotics often used for this purpose contain *Lactobacillus* and *Bifidobacterium* (Diakakis, 2008; Abd El-Moez, *et al*., 2011).

2.4.2. Calf scours

2.4.2.1. Aetiological Agent

Most calf scours are caused by infectious agents; *E. coli* bacteria, rotavirus and cryptosporidium. They occur as an acute fatal septicemia and/or subacute pericarditis. It is a common systemic disease of economic importance worldwide. Many cases of scour proceed very rapidly, causing severe dehydration and metabolic imbalance within a few hours of the onset of disease. *E. coli* (K99 and F41) can cause very severe scour and dehydration in calves of less than one week old. Diarrhea caused by *E. coli* can occur as early as 24 hours after birth, but seldom occurs after three days of age unless it occurs as part of a mixed infection with rotavirus and cryptosporidia. *E. coli* can also invade the bloodstream and cause colisepticaemia (Quinn *et al.,* 1994).

2.4.2.2. Pathogenesis

Virulence factors *Escherichia coli* include the ability to resist phagocytosis, utilization of highly efficient iron acquisition systems, resistance to killing by serum, production of colicins and adherence to respiratory epithelium. Mortality survey confirmed scour as the main clinical sign (48%) in camel calves which were born alive but died within the first month of life (Hamouda *et al.,* 2010).

2.4.2.3. Clinical Signs

Systemic infection occurs when large numbers of pathogenic *E. coli* gain access to the bloodstream from the respiratory tract or intestine. Bacteremia progresses to septicemia and death or the infection extends to pericardium, joints and other organs. Isolation and identification of *E.coli* using traditional method is not sufficient to differentiate between pathogenic and non pathogenic strains. Serotyping is required and the use of mPCR is a useful accurate tool to detect toxic genes; shiga toxin and intamin which are responsible for signs of toxicity (Ahmed *et al*., 2007). Isolation of a pure culture of *E. coli* from heart blood, liver or typical visceral lesions in a fresh carcass indicates colibacillosis. Pathogenicity of isolates is established when results in fatal septicemia or typical lesions. Severe infection results in severe dullness, listlessness and collapse in calves of less than one week old and is often fatal despite therapy. Disease produced by this organism is associated with the feeding of inadequate levels of colostrum. Rotavirus can cause very severe scour and mortality in calves of up to two weeks old. The virus is widespread on farms and calves. *S.* Dublin and *S.* Typhimurium are sometimes implicated in calf scour. These infections are most common in calves bought in from markets. Salmonellosis has been suggested as a cause of camel neonatal mortality (Tibary *et al*., 2006). The incubation period in cattle is as little as 15 minutes in newborn calves. Resistant *Salmonella* infections of calves are very common, so disease caused by *Salmonella* is often very severe (Radostits *et al*., 2007).

2.4.2.4. Diagnosis

Many scours, regardless of cause, show similar clinical picture. However, the severity and character of the scour and the age of the affected calves can all help to make a professional judgement for the cause. Frequently, examination of faeces samples from a group of calves can identify the organisms present in an outbreak. Occasionally however, routine tests fail to identify any specific organism and further examinations are required to make an accurate diagnosis, for example, history of predisposing causes, isolation of *E.coli*, post-mortem examination (Quinn *et al.,* 2002) and the use of PCR to test for enterotoxigenic genes (Ahmed *et al*., 2007).

2.4.2.5. Prevention and Control

Treatment of scour depends on prevention of dehydration. In the early stages of disease, provision of fluids by mouth is very effective buy in sever cases of dehydration; fluids must be given directly into the blood. Antibiotics should only be used for *E. coli* and *Salmonella* infection, after sensitivity test to choose the best drug, as inappropriate use of antibiotics can lead to serious antibiotic resistance problems. Provide careful attention to good husbandry and nursing. Ciprofloxacin and probiotics such as *L. acidophilus* isolated from colostrums of goat and mere are highly effective in treatment of infection thus control of calf scour is based on feeding plenty of colostrum immediately after birth (Abd El-Moez *et al*., 2010). Ciprofloxacin coated with gold nanoparticles showed high hindrance *in vitro* for the growth of *E.coli* and *S.*Typhimurium (Zawrah and Abd El-Moez, 2011). Vaccination is very important in the control of calf scour, vaccines are to protect against *E. coli* and rotavirus. However, it is unlikely to be effective unless used in conjunction with good husbandry (Hirsh and Zee, 1999).

3. Conclusion

Sudden death is a serious problem in farm animals. It may occur due to microbial and non-microbial causes. Animals subjected to stressful conditions may be highly susceptible to microbial causes of sudden death. Accurate and rapid diagnosis of sudden death is essential for setting of control programs. It depends mainly on case history, clinical signs, postmortem examinations and lab diagnosis. It is recommended to develop an accurate control program in cases of sudden death to prevent the spread of disease and prevent progress and severity of cases as well as to decrease mortality rate in susceptible flocks. Such program should be based on accurate diagnosis of the causative agent followed by quarantine measurements to prevent spread of infection, hygienic disposal of infected carcasses. Vaccination is required in most cases but it should include the serotype involved in the outbreak.

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