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A Review Article on Cryptosporidiosis

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Abstract: Cryptosporidiosis is caused by protozoan parasites of the genus *Cryptosporidium*, which are ubiquitous apicomplexan parasite that infects the micro-villous border of the gastrointestinal and respiratory epithelium of wide range of vertebrates including human beings and causing diarrhea in both immune-competent and immune-compromised individuals. These organisms has complex life cycle which requires single host and grow only in living cells but can survive in the environment for long periods without losing its infectivity. Fecal contamination of environment, food and water and contact with infected animals plays vital role in the transmission and spread of the disease. Cell mediated immunity is a corner stone of immune response to infection and diagnosis is mainly based on identification of oocysts from fecal materials.There is evidence of zoonotic cryptosporidiosis associated with farms and exposure to infected livestock, particularly young cattle, animal manure and contaminated water.*Cryptosporidium*have been the cause of diarrhea in man and one of the more common opportunistic pathogen affecting human patient with AIDS. High morbidity which results in production losses and death of live animals are significant problems to farm owners. Because of its resistance to antimicrobials and other anticoccidials, currently no effective therapeutic agent is available. Good hygiene measures are important to control and prevent the disease.

Key words: Animals · Transmission · Cryptosporidium · Diagnosis · Diarrhea · Humans · Oocyst

INTRODUCTION

Cryptosporidiosis is caused by a protozoan parasite of the genus *Cryptosporidium,* family Cryptosporididae, orderEucoccidiorida, classCoccidea and phylum Apicomplexa. The parasite infects epithelial cells in the microvillus border of the gastrointestinal tract of all classes of vertebrates [1] and causes severe chronic and even fatal diarrhea with malabsorpition and dehydration [2].

Currently, there are 16 recognized species and nearly triple this number of unnamed cryptosporidianshas been identified only as genotypes. The hosts ranges and pathogencity is species variable. Among thespecies, *Cryptosporidiumparvum*is most commonspecies of medical and veterinary importance [1]. Cryptosporidiosis is a disease commonly caused by *Cryptosporidium parvum*, [2] causes gastrointestinal illness in a wide variety of mammals, including humans, cattle, sheep, goat, pig and horses worldwide. Most people and animals infected with *Cryptosporidium parvum*develop immunity and recover from that infection. However, the disease is persistent and life threatening if there is immunologic impairment [3].

In addition to specific acquired immunity, adults of several species appear to develop an innate age-dependent resistance to cryptosporidiosis [4]. The disease especially in young animals can cause severe illness or death, resulting in decreased performance and production loss. Calves, lambs, piglets and goat kids can become severely ill following infection, resulting in financial loss to the producers from both extra care and supportive therapy needed and the death of production animals. Thus, young animals probably are a more important source of infection than adults [5].

Animal handlers, medical personnel, human beings living or traveling in developing countries and children in day care centers appear to have the greatest risk of exposure to *Cryptosporidium* and high risk activities would include camping, farming and diaper changing [3]. Cryptosporidiosis can be debilitating to healthy individuals and results in significant morbidity and mortality to special populations, such as children, the elderly and the immune-compromised [6].

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Vertebrate host, including humans and infected individuals show a wide range of clinical presentation, but the pathogenicity of *Cryptosporidium* various with species of parasites involved and the type, age and immune status of the host. In many animals, *Cryptosporidium* infections are not associated with a clinical signs or are associated with only acute, self-limiting illness [5]. In acute caseclinical manifestations of cryptosporidiosis typically include persistent watery diarrhea, nausea, vomiting, abdominal cramps and fever in man, dog and cat [7].

Treatments of cryptosporidiosis consist of supportive (fluid and electrolyte) therapy. Keep affected animals warm, dry, well fed and at a constant ambient temperature, to minimize their energy requirements during the course of clinical disease.Good hygienic measures are important in disease control and prevention. In people with AIDS, the ideal treatment involves partial restoration of immune function with highly active antiretroviral therapy (HAART) and rise in CD4 cell count [8].

Therefore, the objectives of this paper are:

- To highlight the etiology, epidemiology, pathogenesis, clinical signs, diagnosis and treatment of cryptosporidiosis
- To review the public health and economic importance of the disease
- To indicate the important control and preventionoptions.

Cryptosporidiosis

Etiology: *Cryptosporidium* is an obligate, intracellular, extra-cytoplasmic, single celled coccidian parasite somewhat smaller than red blood cell, with monoxenous life cycle. It invades the microvillus border of GIT and respiratory epithelium of a wide range of vertebrate animals and is associated with watery diarrhea in mammals, diarrhea and respiratory illness in birds and gastroenteritis in reptiles and fish [5].

Effects of infection vary with the species of *Cryptosporidium.* Some species of *Cryptosporidium* infect many host species, whereas others appear restricted to groups such as rodents or ruminants and still others are known to infect only one host species. Some species primarily infect the stomach, whereas others primarily infect the intestine. Some species are pathogenic, whereas the presence of others has not been shown to be related to any disease manifestations [1].

Currently, there are 16 valid species (Table 1) and over 40 genotypes, some of which eventually might represent different species.Of the valid species, which can infect humans, *Cryptosporidiumparvum* and *C. hominis* are the most frequently identified species [5]. *Cryptosporidium parvumis* the species normally, but not exclusively, associated with zoonotic transmission. *Cryptosporidium parvum* and *C. andersoni* have been associated with morbidity in livestock, whereas *C. baileyi, C. galli* and *C. meleagridis* have been associated with morbidity in domesticated fowl [1].

Life Cycle of *Cryptosporidium*: *Cryptosporidium* has a complex, homoxenous life cycle [9] with a capability of completing all stages of its development (asexual and sexual) within a single host [10]. The life cycle of most *Cryptosporidium* spp. is completed within the GIT (small intestine) of the host, with developmental stages being associated with the luminal surface of the mucosal epithelial cells [11]. The cycle has an endogenous and exogenous stage. The exogenous stage is represented by sporulatedoocysts, which is excreted in the environment with the feces of infected hosts [10]. These oocysts may remain in the environment for very long period without losing its infectivity; due to a very robust oocyst wall that protects the four sporozoites against physical and chemical damage. The endogenous phase begins with the ingestion of oocysts by a suitable host [12]. When a new host ingests the oocyst, the suture in the oocyst wall opens (excystation), triggered by the body temperature and the interaction with stomach acid and bile salts. Four sporozoites are released in to the small intestine of the host and they infect the epithelial cells of the small intestine, mainly jejunum and ileum [13]. The invasion process is likely to involve molecules discharged from parasite organelles found in the apical end of the sporozoits. The sporozoites affix to the luminal surface of epithelial cells and differentiate asexually into trophozoites which to produce two different types of meront (type I and II) by merogony. Type I meronts form eight merozoites, which then rupture out of the host cell, infect other neighbouringhost cells and either develop into type II meront or complete another cycle of type I meronts. Type II meronts produce four merozoites which become microgamonts or macrogamonts [7]. The differentiated gamates, undergo sexual reproduction within the same host. Fertilization between macrogamonts and microgametes results in formation of a zygote which develops into oocyst containing four sporozoites [14].

Table 1: Major and minor species of *Cryptosporidium* infecting humans and selected domesticated animals and wildlife

Host	Major Species	Minor Species
Camel	*C. andersoni, C. parvum?*	
Cat	*C. felis*	
Cattle	*C. parvum, C. bovis, C. andersoni*deer like genotype	*C. suis*
Chicken	*C. baileyi*	*C. meleagridis, C. galli*
Deer	*C. parvum*	
Dog	*C. canis*	
Fish	*C. scophthalmi, C. molnari*	
Fox	*C. canis*fox genotype	*C. canis*dog genotype, fox genotype II
Goat	*C. parvum*	
Guinea pig	*C. wrairi*	
Horse	Horse genotype	*C. meleagridis, C. felis, C. canis, C.suis, C. baileyi*, cervine genotype
Human	*C. hominis, C. parvum*	
Lizard	*C. serpentis, C. varanii*	Lizard genotype
Mouse	*C. muris*, mouse genotype	
Pig	*C. suis*	Pig genotype II
Sheep	Cervine genotypes 1–3, bovine genotype	*C. parvum*, sheep novel genotypes
Snake	*C. serpentis*	*C. varanii*, snake genotype
Squirrel	*C. muris*, squirrel genotype	
Turkey	*C. meleagridis, C. baileyi*	

*Source: [1]

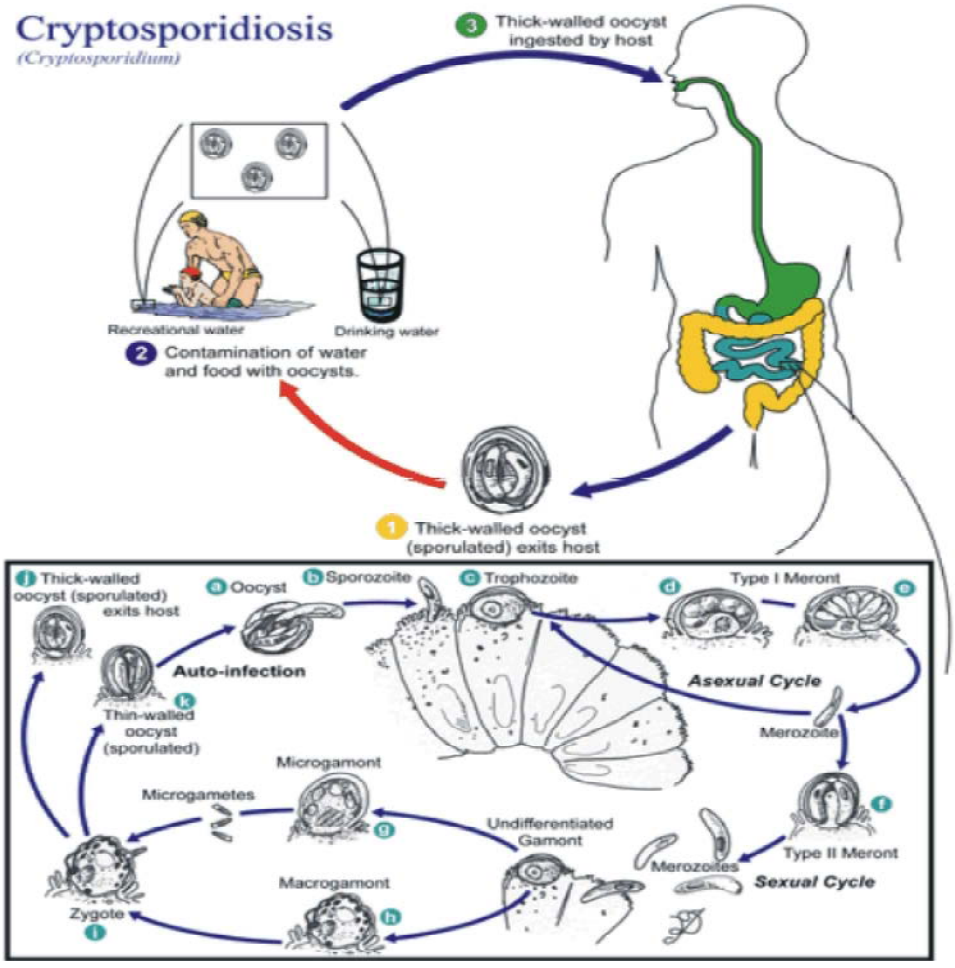

Fig. 1: Life cycle of *Cryptosporidium* spp.* Source:[11].

Two different types of oocysts are produced: the thick-walled, which is commonly excreted by the host, after sporogony and the thin-walledoocyst, which is primarily involved in autoinfection. The thin- walled oocysts may exist within the same host and start a new life cycle (autoinfection). This may lead to a heavily infected epithelium of the small intestine, resulting in malabsorpitive or secretory diarrhea. The thick-walled oocyst is excreted with the faeces and is environmentally robust. The oocysts are the result of the sexual reproduction cycle [15].

Epidemiology

Geographical Distribution: Cryptosporidiosis has worldwide distribution [3]. Since the first of its presence in cattle; the disease has been the object of many prevalence studies worldwideand has been documented inanimals and human beings. Most infections have been attributed to *C. parvum* associated with clinical disease. Most of the published studies were from industrialized countries and little is known on the prevalence of the disease in African countries [16].

Cryptosporidiosis in cattle has beenrecognized as an emerging threat in different parts of the world.Reports of cryptosporidiosis have been made from many countries including United States, Canada, South America, United Kingdom, Norway, Iran, Africa, Pakistan, Thailand, Australia, Japan, Germany and Hungary [17]. In India the *Cryptosporidium* oocysts were firstdemonstrated in the faeces of buffaloes and zebu cattle and organized dairy farms in and around Bangalore, South India [18].

In developing countries, *Cryptosporidium* infections occur mostly in children younger than 5 years, with peak occurrence of infection and diarrhea in children younger than 2 years. In industrialized countries, epidemic cryptosporidiosis can occur in adults by the food-borne or water-borne route [19].

In addition to coprological data, several immune-serological surveys have been conducted. Sero-prevalence ranges from 25-30% in industrialized countries and 50-60% in developing countries. These data strongly suggest that cryptosporidiosis highly prevalent in humans and animals, both in developed and developing countries [19].

Source of Infection and Modes of Transmission: The source of infection is feces which contain oocysts that are fully sporulated and infective when excreted. Large numbers are excreted during the patent period in calves resulting in heavy environmental contamination [20].

*Cryptosporidium*oocysts are released in large quantities from clinically infected humans and animals (acuteor chronic infections). Human and dairy effluents are probably the most importantsources of environment and surface water contamination.*C. hominis* and *C. parvum* account for the vast majority of human infections, the sources of these species are the predominant reservoir of human cryptosporidiosis. Humans are the only significant source of *C. hominis* and humans and ruminants are the predominant sources of *C. parvum* [5]. Cattle have been implicated as a major source of *C. parvum*in pasture run off, which is responsible for environmental contamination that cause infection either by direct or indirect contact through fecal contamination of feed or water for animals [21].

The role of cows as a possible infection source for calves has been addressed.Such transmission could be facilitated by a per parturient rise in oocystshedding in infected cows. Per parturient rises have been shown for*C.parvum*and for *C. andersoni*oocysts [22].

Transmission from one host to another is achieved by ingestion of an encysted, sporulatedoocyst for *Cryptosporidium* [23]. The routes through which oocysts are transmitted from feces to the mouth are diverse and reflect the main transmission routes for many intestinal pathogens [1]. Transmission can be direct from host to host or indirectly by ingestion of fecal contaminated feed or water. Transmission is likely to be direct between infected animals since environmental contamination on farms with oocystswould be insufficient to account for the high levels of infection seen in cattle [23].

Contact with any acquaintances or household member with a similar illness attendance or work of a child care facility by case or a household member; scourers of drinking water, intending water of homes and work, as well as streams, lakes and other untreated sources, high risk foods (e.g. row milk or raw milk products) are the common sources of infection for humans [24].

Risk Factors

Age: There is a significant association between age and risk of infection with *Cryptosporidium*. Cryptosporidiosis due to *C. parvum* is predominantly a problem of neonate animals with maximum rate of excretion of oocysts between the age of 4 and 21 days .Although exceptions occur, older animals generally develop poor infections, even when unexposed previously to this parasite [5]. Age-related resistance, unrelated to prior exposure, has been observed in lambsbut not calves [20]. The common

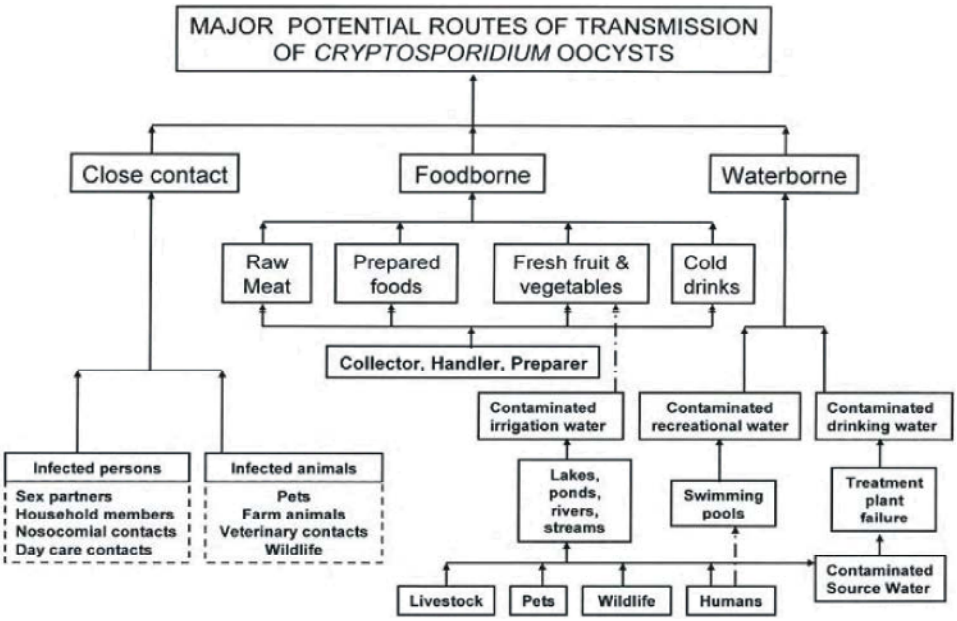

Fig. 2: A "Fault Tree" depicting sources of oocysts and routes of transmission. Source: [1]

occurrence of cryptosporidiosis in young animals reflects their susceptibility to infection with a low number of oocysts and common exposure to oocysts [1].

Cryptosporidiosis is a major cause of diarrhea in children with and without HIV infection in developing countries [25]. In these countries, *Cryptosporidium* infection in early childhood has been reported to be associated with subsequent impairment in growth, physical fitness and cognitive function. Incidence is higher in child day care centers that serve food [3].

Immune Status: Undeveloped immune systems are usually seen in young livestock and human infants. Weakened immune systems may be seen in animals suffering from other diseases, elderly humans and malnourished persons; individual's receiving chemotherapy or corticosteroid therapy and HIV positive individuals [4]. These individuals experiences increased mortality, decreased weight gain or weight loss and generally poorer performance overall when compared to healthy animals. Additionally, infected animals represent a potentially large source of oocysts that can contaminate water and foods used for human consumption. A single infected calf can excrete up to 10 billion oocysts during a 2 weeks infection [14].

Innate immunity serves as an early sensor of infection and also activates antimicrobial killing mechanisms that might curb the reproduction of invading microorganisms until the adaptive immune response becomes functional. The innate immune system includes the activities of inflammatory immune cells such as natural killer (NK) cells that produce cytokines and that might be cytotoxic to infected cells; macrophages and neutrophils that can engulf extracellular microorganisms and eosinophils. Non immune cells, including epithelial cells, also have immunological functions such as the ability to produce cytokines and antimicrobial peptides [26]. Epithelial cells are now known to play a key role in establishing mucosal immune responses to infections. They produce main inflammatory molecules such as chemokines involved in establishing inflammation. They also mediate mechanisms of microbial inactivation that can be activated directly by infection or via inflammation. Interaction and synergy between the components of innate and acquired immunity will promote immunity against *Cryptosporidium* [27].

The adaptive immune response induced by specific antigens recognized by T- and B-cells is generally required to eliminate rapidly proliferating or virulent microbial pathogens and has the added advantage over innate immunity in having immunological memory, which allows prompt reactivation of memory T- and B-cells if re-infection occurs [28, 29]. T-cells activate B-cells to proliferate and differentiate into plasma cells that produce antibodies. Cell mediated immunity (CMI) is thought to be the cornerstone of the immune response to *Cryptosporidium* infection and appears to be vital in both protection against and recovery from infection [30]. Both CD4+ and CD8+ T cells contribute to resistance and clearance of acute *Cryptosporidium* infection [31, 32].

Management and Hygienic Condition: The risk of *Cryptosporidium* infection increases when animals are commonly housed and overcrowded and sanitary conditions found on much ruminant exploitation also contribute to the important presence of the parasite on such farms [33]. On most exploitation typically concentrates over a short period of time, in which crowding of the animals is common there by facilitating contact between diseased and healthy individuals. If the hygiene and sanitary conditions are moreover deficient, the conditions for a diarrhea outbreak are favorable. Contamination by water-born infectious disease is closely linked to urban slums conditions such as overcrowding and high level of fecal pollution by animal and human excreta. These conditions will increase the risk of disease in animals and human [14].

Pathogenesis: Host parasite interactions occur at a number of stages during the process of infection with *Cryptosporidium*. The initial host-parasite interaction of attachment and invasion are crucial primary steps in the pathogenesis of Cryptosporidiosis [4].

Followingingestion of the oocyst there is excystationorrelease of infective sporozoites [20]. The parasite infects the epithelial cell lining of the intestinal tract, causing blunting of the small intestinal villa, proliferation of the cells and infiltration of the underlying tissue with inflammatory cells [24]. The attachment of the sporozoites, which is ligand–receptor mediated, triggers rearrangement of the host actin cytoskeleton to form a parasitophorous vacuole within which the parasite develops [34] and exerts its effects on the host and surrounding cells. The parasitophorous envelope of the trophozoites and schizonts are derived from the microvilli and the intracellular location of the organism is confined to fusion of the organism, with the apical cytoplasm of the epithelial cells and their enclosure by host membranes. Thus the organism is intracellular but extracytoplasmic. Microscopic analysis of *C. parvum* infected intestinal tissue reveals heterogeneity in the immune response mounted against the parasite, ranging from acute inflammation with numerous polymorphonuclearneutrophils infiltrating the mucosa to chronic inflammation composed of lymphocytes, histiocytes and eosinophils [24]. The pathogenesis of the diarrhea is unknown, but the varying degrees of villous atrophy suggest that digestion and absorption may be impaired, resulting in diarrhea [20].

Clinical Features and Necropsy Findings: Depression and anorexia are followed by a profuse yellow watery diarrhea associated with dehydration. Tenesmus may be present. After a few days the diarrhea become intermittent and the feces pasty for up to 10 days. The feces may conation flecks of blood and mucus plugs and the diarrhea may persist for several days until death. Diarrhea also accompanied by other symptoms of gastrointestinal distress, including nausea, vomiting, abdominal cramps and discomfortt [26] specially in man, dog and cat [7].

Immune-compromised hosts experience protracted, sometimes life – threatening diarrheal illness. (E.g. up to 20 liters of stool output) in AIDS patients, it is now apparent that, even in AIDS patients (with CD4 count less than $100/mm^3$,) the disease has a variable presentation. Approximately 50% of AIDS patients shows persistent or relapsing diarrhea and $1/3^{rd}$ will experience dehydrating diarrhea requiring intravenous rehydration (i.e. 'cholera'-like); and 15% will experience self-resolving diarrhea [30].

Generally, the extent of the mucosal injury in the gastrointestinal tract and the severity of the disease are directly correlated. Enteric lesions increase in severity towards the terminal portion of the ileum, the cecum and colon may also be seen. The mucosa of the affected parts of the intestine is often intensely hyperemic. The carcass may be emaciated and dehydrated. Adjacent intestinal microvilli are blunt and atrophy in affected parts of the intestine and focal necrosis of epithelial cells [24].

Diagnosis: Numerous techniques, including histology and ultrastructural examination of biopsy material for lifecyclestages, examination of feces for the presence of oocysts and detection of*Cryptosporidium*antigens and DNA, have been used to diagnose infection in humans and animals. Molecularbasedtechniques are required for species identification[1].

Histologicaly, the diagnosis of cryptosporidiosis rests on the identification of spherical oocysts in stool or the intracellular stages with in biopsy specimens of human GIT mucosa. In tissue sections, a simple haematoxylin and eosin should suffice to identify the morphology of the intracellular life stage of the parasite in its unique apical location within the intestinal epithelial cell. However, this method of testing can give false negatives due to "patchy" nature of the intestinal parasitic infection [4].

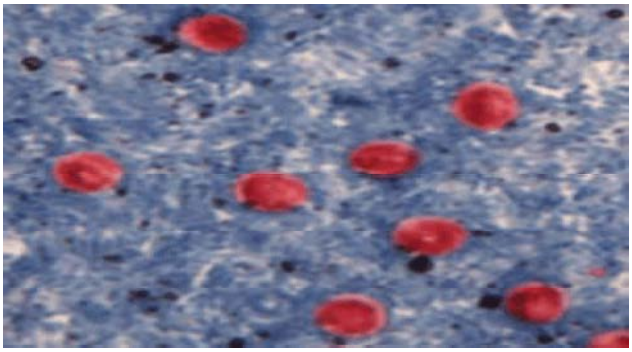

Fig. 3: Ziehl-Neelson staining of *Cryptosporidium*oocysts.* Source: [36]

A wet mount using saline and/or iodine is the basis of all microscopic technique. Routine diagnosis of cryptosporidiosis in most countries has been based on microscopic detection of oocysts after staining of fecal smears [7]. In specimens containing small numbers of oocysts, increased sensitivity can be achieved by employing a concentration method [1]. Concentration methods using the principals of flotation and sedimentation have been widely used, with solutions such as sucrose, salt and zinc sulphate [7].

Detection of Oocysts: Stool samples from most clinically ill cases will contain large numbers of thick-walled oocysts and sufficient *Cryptosporidium* antigen; therefore, the use of standard staining and immunological techniques should result in a positive diagnosis. Staining methods were then developed to detect and identify the oocysts directly from stool samples [4].

The modified acid-fast (Ziehl-Neelson) stain is used most reliably and specifically to detect the presence of cryptosporidialoocysts.Theoocysts will appear as pink stained, round to oval structures of about 3 to 6 ìm in diameter, containing distinct internal structures. Acid-fast stains can also be performed by using either the hot staining method or the cold method. *Cryptosporidium* oocysts are acid-fast and readily identified microscopically with oil immersion at a magnification of 400 X. This method is low cost, good for screening large number of samples, permanent stain, making it possible to send doubtful or scanty positive slides to a reference laboratory for confirmation. However, its time-consuming procedure (about 30 to 45 minutes), requires intensive training and experience to interpret the results [35].

Immunoassay: Immunologically, the ELISA or IFA can detect anti-cryptosporidium antibodies, but neither of these assays can provide a direct diagnosis of cryptosporidiosis. Several IFA kits are commercially available for the detection of *Cryptosporidium* in stool and environmental samples [11]. Although there are several sensitive and specific tests, they require technical skills and sophisticated equipments. Therefore, these methods are not used routinely in the majority of laboratories [37].

Faecal enzyme immunoassays have been reported utilizing oocyst reactive monoclonal antibodies. The monoclonal antibodies were adapted for antigen detection in an antigen-capture enzyme immunoassay. Enzyme immunoassay is less sensitive than the modified Ziehl-Nielsen stain or immunofluorescence assay especially when oocyst numbers were small. An indirect double antibody enzyme immunoassay using polyclonal antisera has also been developed, but the test was not as sensitive as the immunofluorescent [38].

In ELISA the specimens should not be concentrated prior to testing. This is highly sensitive and specific technique and is useful for screening large numbers of specimens in a short time period. Also, it does not rely on skills in microscopy [37].

Polymerase Chain Reaction: Recently, new genetic methods of detecting *Cryptosporidium* have been developed, using PCR or other DNA-based detection methods. PCR-based detection of microbes in clinical samples is attractive due to its extreme sensitivity and specificity. Additionally, the genetic information obtained from the sample may permit non human pathogens to be distinguished from human pathogens. Some investigators have found high sensitivity for PCR-based assays and suggest that these assays are more sensitive than microscopic analysis of acid-fast smears but the sensitivity of the PCR assays was inhibited by "uncharacterized components in the samples" [5].

Treatment: There is no effective drug therapy available for cryptosporidiosis, due to antimicrobial resistance. However, spiramycin, halofuginone and paromomycin may be of some valueinreducingoocyst output and the severity of diarrhea and/or mortality in infected animals [1, 20]. One possible explanation for the antimicrobial resistance is that *Cryptosporidium* establishes a compartment within the host cell, which is morphologically different from the setting used by the related parasites. This unique vacuole may shelter the parasite from antimicrobial. The other explanation is that the parasite has two morpho-functional type of oocysts, thick-walled and thin-walled, with the latter responsible for the initiation of the auto-infective cycle in the infected host; which lacks morphological structure such as sporocyst, micropyle and polar granules and finally the insensitive of *Cryptosporidium* to all anti-coccidian agents tested so far .Effective treatment of animals suffering from cryptosporidiosis may require oral or parenteral rehydration with fluids and electrolytes, in addition to antidiarrheals and attempted chemotherapy with putative anticryptosporidial drugs. Rehydration is particularly important in young animals and those immunocompromised or suffering from intercurrent disease. The latter should receive appropriate antibiotic therapy if bacterial copathogens are involved [1].

In calves treatments of cryptosporidiosis consists of supportive therapy. Keep affected animals warm, dry, well fed and at a constant ambient temperature, to minimize their energy requirements during the course of clinical disease. If managed in this fashion, most calves with uncomplicated infection will recover in 5 to 10 days. Calves that become dehydrated should be given appropriate IV fluids or orally. Early electrolyte treatment protocols addressed electrolyte and partial energy balance. The last treatment protocols addressed electrolyte balance, compensate for metabolic acidosis resulting from long term fluid losses, addressed energy balance and partially addressed protein balance by incorporating milk or milk replacer feeding between electrolyte treatments [39].

Drug approved by the US Food and Drug Administration for treatment of cryptosporidiosis in children and immunocompetent adults is the anti-protozoal agent nitazoxanide. However, nitazoxanide is not effective without an appropriate immune response and is therefore ineffective against immunocompromised individuals. Recovery and survival rates has been dramatically improved with the use of highly active antiretroviral therapy, which cause increased CD4+ T-lymphocyte counts in immunocompromised individuals [7].

Controlandprevention: Prevention of cryptosporidiosis transmission is clearly dependent on good hygienic measures in setting. The logical approach to prevention is to minimize fecal-oral transmission between young animals, especially calves. Healthy calves should be confined separately from sick calves. Use all-in, all-out management, with thorough cleaning and several weeks of drying between batches of calves. Pens and utensls should be cleaned with an ammonia solution and dried for several days before introduction of a new group of calves. Rats and mice should be controlled, because they probably are a reservoir for *C. parvum* infection of calves [5].

Hands must be washed after handling household pets, laboratory and farm animals or after working in soil. Washing or cooking of food. Food that will be eaten uncooked should be washed with purified (boiled) water before serving and avoid drinking or eating any of the following items unless they are pasteurized: milk, dary products and juice).*Cryptosporidium* species oocysts are resistance to most chemical disinfectants. Emphasis should be on cleaning and removal of fecal material rather than disinfecting. Moist heat, freezing, or drying is probably the most effective practical means for killing oocyts. Moreover, avoid drinking of water from rivers, lakes, streams and ocean, swimming pools or hot tubs [40, 5].

Public Health and Econonic Importances: There is evidence of zoonotic cryptosporidiosis associated with farms and exposure to infected livestock, particularly young cattle, animal manure and contaminated water. *Cryptosporidium*have been the cause of diarrhea in man and one of the more common opportunistic pathogen affecting human patient with AIDS. Infection in domestic animals and pets may be a reservoir for infection of susceptible humans. In humans, *Cryptosporidium* is considered to be a relatively common non-viral cause of self- limiting diarrhea in immune competent persons, particularly in children. Other manifestations include nausea, vomiting, abdominal cramps, weight loss and fever. In contrast, in immune-compromised patients cryptosporidiosis may be debilitating chronic diarrhea

associated with malabsorpition and significant weight loss. Although *C. parvum* is the most common zoonotic species, other cryptosporidium species have also been identified including *C. canis, C. felis and C. muris*.[26].

The economic impact of cryptosporidiosis, stemming from lost productivity, is enormous. *Cryptosporidium* infection in livestock may cause important economic impact to farmers because of its high morbidity and sometimes mortality rates among farm animals. Cryptosporidiosis, especially in young animals, can cause severe illness or death, resulting in decreased performance and production loss and results in financial loss to the producers from both extra care and supportive therapy needed and the death of production animals [5].

CONCLUSION AND RECOMMENDATIONS

Cryptosporidiosis is a ubiquitous parasitic disease that infects wide variety of animals and humans. Infected humans and animals shed oocysts in very high numbers. Transmission occurs through direct or indirect contact with feces of these shedders. Outbreaks illustrate the different routes, person-to-person spread in institutions, animal contact during farm visits and contact with recreational waters, swimming pool visits, municipal drinking water and food. Fatal diarrhea with mal-absorption and dehydration occurs in young and immune compromised hosts. Currently there is no effective therapeutic agent for *Cryptosporidium* infection. Boiling in addition to filtration is useful in destruction of resistance oocysts. Following adequate management and hygienic practices are paramount importance in the control and prevention of the disease.

Based on the above conclusion, the following recommendations are made:

- Implementation of better management practices like stream-bank fencing, use all-in, all-out management system, isolate infected animals, cleaning and disinfection activities to limit environmental contamination with fecal matter and use protective materials when handling animals
- There should be extension service including healthy education to the farm owners and cattle attendants.
- Avoid swimming and direct drinking from lakes, streams, rivers and pasteurization or boiling of water before consumption.
- Further study is needed to enhance the understanding and asses the failure of most therapeutic agents against the disease.
- Improved diagnostic techniques should be implemented to identify the *Cryptosporidium* species in humans and animals.

REFERENCES

1. Fayer, R. and L. Xiao, 2008. *Cryptosporidium* and Cryptosporidiosis. 2nd edition. Boca Raton, London, New York, CRC Press, pp: 1-450.
2. World Health Organization, 2001. Guidelines on standard operating procedures for laboratory diagnosis of HIV opportunistic Infections. Bloodsafty and clinical technology, WHO, Seara.
3. Center for Disease Control, 2007. Preventing Cryptosporidiosis: A Guide for persons with compromised immune systems. *Cryptosporidium* systematic and implications for public health.Available athttp://www.cdc.gov/ cryptosporidiosis. Accessed on March 22/2015.
4. Suleiman, I., A. Lal and L. Xiao, 2001. A population genetic study of the *Cryptosporidium parvum* human genotype parasites. J. Eukaryot. Microbial., 57: 245-275.
5. Xiao, L., R. Fayer, U. Ryan and S. Upton, 2004. *Cryptosporidium* Taxonomy: Recent Adivances and Implications for Public Health. Clinical. Microbial. Reviews., 17: 72-97.
6. Inungn, J., A. Morse and C. Gordon, 2000. Risk factors, seasonality and trends of cryptosporidiosis among patients infected with human immunodeficiency virus. Am. J. Trop. Med. Hyg., 62: 384-387.
7. Furul, F. and L. Baha, 2013. Cryptosporidiosis as Treating Health Problem: Asi. Pac. J. Trop. Biom., 3(11): 916-924.
8. Carr, A., D. Marriott, A. Field, E. Vasak and D. Cooper, 1998. Treatment of HIV 1- associated micro-sporidiosis and cryptosporidiosis with combination antiretroviral therapy. Lancet, 351: 256-261.
9. Widmer, G., 1998. Genetic hetrogeneity and PCR detection of *Cryptosporidumparvum*. Adv. Parasitol., 40: 223-239.
10. Xiao, L., U. Morgan, R. Fayer, R. Thompson and A. Lal, 2000. *Cryptosporidium* systematic and implications for public health. Parasitol, Today, 16: 287-292.

11. Center for Disease Control, 2000. Summery of notifyable disease. United States, Morb. Mortal. Wkly. Rep., 49: 101-102.
12. Sonia, A., 2011. Cryptosporidiosis from Epidemiology to Treatment, Microb, Virus and Parasit. AIDS Pro., 14: 292-293.
13. World Health Organization, 2006. Guidelines for Drinking Water Quality.
14. Douglus, P., 1999. New insights into human cryptosporidiosis. Cli. Microbial. Rev., 12: 554-563.
15. Tzipori, S. and J. Griffiths, 1998. Natural history and biology of *Cryptosporidium parvum*. Adv. in Parasit., 40: 5-35.
16. Donoghue, P.J., 1995. *Cryptosporidium* and cryptosporidiosis in man and animals. Inter. J. Parastol., 25: 139-195.
17. Fayer, R., M. Santin and L. Xiao, 2005. *Cryptosporidium bovis*(Apicomplexa: Cryptosporidiidae) in cattle (*Bos Taurus*). J. Parasitol., 91: 624-629.
18. Mallinath, R., A. Chikkachawdappa, G. Gowda and E. Dsouza, 2009. Studeis on the prevalence of cryptosporidiosis in bovines in organized dairy farms in and around Bangalore, South India. Vet. Arhiv., 79: 461-470.
19. FAO, 2004. HIV Infections and Zoonosis.Animal production and health paper.Food and Agricultural Organization of the United Nations, Rome, Italy.
20. Radostits, O.M., C.C. Gay, K.W. Hinchcliff and P.D. Constable, 2006. Diseases associated with protozoa. 10th edition, In: Veterinary Medicine: A Textbook of Diseases of cattle, horses, sheep, pigs and goats. Saunders Elsevier, pp: 1483-1540.
21. Ayinmode, A., B. Adekunle and O. Benjamin, 2010. Prevalence of *Cryptosporidium* infection in cattle from South Western Nigeria. Vet. Arhiv., 80: 723-731.
22. Ralston, B.J., T.A. McAllister and M.E. Olson, 2003. Prevalence and infection pattern of naturally acquired giardiasis and cryptosporidiosis in range beef calves and their dams. Vet. Parasitol., 114(2): 113-122.
23. Merle, E., 2004. Zoonotic Protozoan Parasites in Cattle.http://www.ivis.org.Accessedon April 18/2015.
24. Okyuhsen, P., S. Rich, C. Chappell, K. Grimes, G. Widmer, X. Feng, H.paul and N. Gordon, 2002. Epidemiology and clinical feauture of *Cryptosporidium* infection in immunocompromisedpatients. Clin. Microbial. Rev., 15: 145-154.
25. Javier, E., C. Avila, S. Ignico, L. Tanaco-koido, O. Valijo and R. Sterling, 1997. *Cryptosporidium* infection in Mexican children: Clinical, nutrional, enteropathogenic and diagnostic evaluation. Am. J. Trop. Med. Hyg., 56: 254-257.
26. Chen, X.M., J.S. Keithly, C.V. Paya and N.F. LaRusso, 2002. Cryptosporidiosis. New Eng. J. Med., 346: 1723-1731.
27. McDonald, A., W. Mac Kenzie, D. Addiss, M. Gradus, G. Linke, E. Zembrowski, M. Hurd, M. Arrowood, P. Lammie and J. Priest, 2001. *Cryptosporidium parvum*-specific antibody responses among children residing in Milwaukee during the 1993 Waterborne Outbreak, 183: 1373-1379.
28. Seder, R. and R. Ahmed, 2003. Similarities and differences in CD4+ and CD8+ effector and memory T cell generation. Nature Immuno., 4: 835-842.
29. Kalia, V., S. Sarkar, S. Gourley, T. Rouse and R. Ahmed, 2006. Differentiation of memory B and T cells. Current Opinion in Immuno., 18: 255-264.
30. Manabe, Y.C., D.P. Clark and R.D. Moore, 1998. Cryptosporidiosis in patients with AIDS: Correlates of disease and survival. Clin. Infect. Dis., 27: 536-542.
31. Panterburg, B., S. Dann, H. Wang, P. Robinson, A. Castellanos-Gonzalez, D. Lewis and A. White, 2008. Immune responses to human *Cryptosporidium* sppinfection. Infec. and Immun., 1: 23-29.
32. Borad, A. and H. Ward, 2011. Human immune responses in Cryptosporidiosis. Futu. Microbio., 5(3): 507-519.
33. Mallon, M., A. Macleod, J. Wastling, H. Smith, B. Reily and A. Tait, 2003. Population stractures and the role of genetic exchange in the zoonotic pathogen *Cryptosporidumparnum*. J. Mol. Evo., 56: 407-417.
34. Chen, X.M. and N.F. La Russo, 2000. Mechanisms of effacement and internalization of *Cryptosporidiumpartum* to Villiary and intestinal epithelial calls. Gastroentimology, 118: 368-379.
35. Prakriti, V., S. Madhu and C. Uma, 2012. A comprehensive review of diagnostic techniques for detection of *Cryptosporidium parvum*inoocyst samples. J. Pharma., 2: 15-26.
36. Mor, S., J. Tumwine, G. Ndeezi, M. Srinivasan, D. Kaddu-Mulindwa, S. Tzipori and J. Griffiths, 2010. Respiratory cryptosporidiosis in HIV-seronegative children in Uganda: potential for respiratory transmission. Clin. Infec. Dise., 50(10): 1366-1372.
37. Mehta, P., 200. Laboratory diagnosis of cryptosporidosis. J. Postgrad. Med., 48: 217.

38. Ajjampur, S., R. Sarkar, G. Allison, K. Banda, A. Kane, J. Muliyil, E. Naumova, H. War and G. Kang, 2011. Serum IgG Response to *Cryptosporidium* Immunodominant Antigen gp15 and Polymorphic Antigen gp40 in Children with Cryptosporidiosis in South India. Clin. Vac. Immuno., 18: 633-639.
39. Blasdall, S.A., J.E. Ongerth and N.J. Ashbolt, 2001. Differentiation of *Cryptosporidium parvum* subtypes in calves of four dairy herds by a novel microsatellite telomere PCR with proceedings of *Cryptosporidium* from molecules to Disease.
40. Office International Des Epizootics, 2004. Cryptosporidiosis in manual of standards for laboratory tests and vaccines. 5th edtion, Paris. avalable at http://www.oie.int/eng/norms/ enmanual. Accessed on March 28/2015.