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Ministry of Higher Education
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University of Al-Qadisiyah
College of Veterinary Medicine

Eimeria spp. in poultry

A Graduation Project Submitted to the Department Council of the Internal and Preventive Medicine-College of Veterinary Medicine/ University of Al-Qadisiyah in a partial fulfillment of the requirements for the Degree of Bachelor of Science in Veterinary Medicine and Surgery.

Provided by the student

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فَتَعَٰلَى ٱللَّهُ ٱلْمَلِكُ ٱلْحَقُّ ۗ وَلَا تَعْجَلْ بِٱلْقُرْءَانِ مِن قَبْلِ أَن يُقْضَىٰٓ
إِلَيْكَ وَحْيُهُۥ ۖ وَقُل رَّبِّ زِدْنِى عِلْمًا ﴿١١٤﴾

صَدَقَ اللهُ العَظِيمُ

من سورة طه

Certificate of Supervisor

I certify that the project entitled (----------) was prepared by ---------- under my supervision at the College of Veterinary Medicine / University of Al-Qadisiyah.

Supervisor

Dept. of ----------

Coll. Of Vet.Med./ Univ. of Al-Qadisiyah.

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Certificate of Department

We certify that ---------- has finished his/her Graduation Project entitled (----------) and candidate it for debating.

Instructor

Dr. Muthanna H. Hussain

-- / -- / 2020

Head of Dept. of Int. and Prev. Med.

-- / -- / 2020

Dedication

To the owner of mercy on the face of the earth

To from heaven under her feet ...

to my mother

To whom was the bond and appointed in every step I took.

To my father

To whom my supervisor is proud of

To (Assist lecturer Batool Kadhim Meteab)

To my colleagues and colleagues .....

Accept the soul and fulfill its virtues

You are with the soul, not in the body, a person.

researcher

Summary

Coccidiosis in poultry is still considered as one of the main diseases affecting performance of poultry reared under intensive production system that affects the poultry industry worldwide , having major economic losses in poultry by reducing performance and decreasing productivity .this disease not only hinders the growth of chickens but also facilitates other epidemic diseases. the extensive use of these drugs has resulted in the development of drug resistance by Eimeria spp., Therefore, this study dealt with the main types that cause infection with this disease, the extent of the risk of these types to both chickens and turkeys, the clinical and anatomical symptoms, the definition of the life cycle of these parasites, and how to diagnose these parasites through several main methods, including laboratory diagnosis and differential diagnosis of some diseases with similar symptoms and the use of modern technologies. Such as ELISA, molecular diagnostics, PCR, and the importance of controlling these parasites through good management and vaccines.

List of Contains

List of Shapes

Chapter one

Introduction:

Coccidiosis in poultry is considered as one of main diseases affecting performance of poultry reared under intensive production system. (De Gussem ,2005).

Coccidiosis is caused by protozoa of the phylum Apicomplexa, family Eimeriidae. In poultry , most species belong to the genus *Eimeria* and infect various sites in the intestine. The infectious process is rapid (4-7 days) and is characterized by parasite replication in host cell with extensive damage to the intestinal mucosa.(Kennedy,2001)

Poultry coccidian are generally host-specific and different species parasitize specific parts of the intestine.

In game birds, including quail, the coccidian may parasitize the entire intestinal tract. Coccidia are distributed worldwide in poultry , game birds reared in captivity , and wild birds.(Fanatico,2006).

Chickens are susceptible to at least 11 species of coccidia.

The most common species are *Eimeria tenella* , which cause the cecal or bloody type of coccidiosis , *E. necatrix* , which causes bloody intestinal coccidiosis, and *E. maxima,* which cause chronic intestinal coccidiosis. (Allen,2000).

Aims of study

1-To clarify the effect of coccidiosis on poultry performance.

2-To recognize the E.spp. in chickens and turkey.

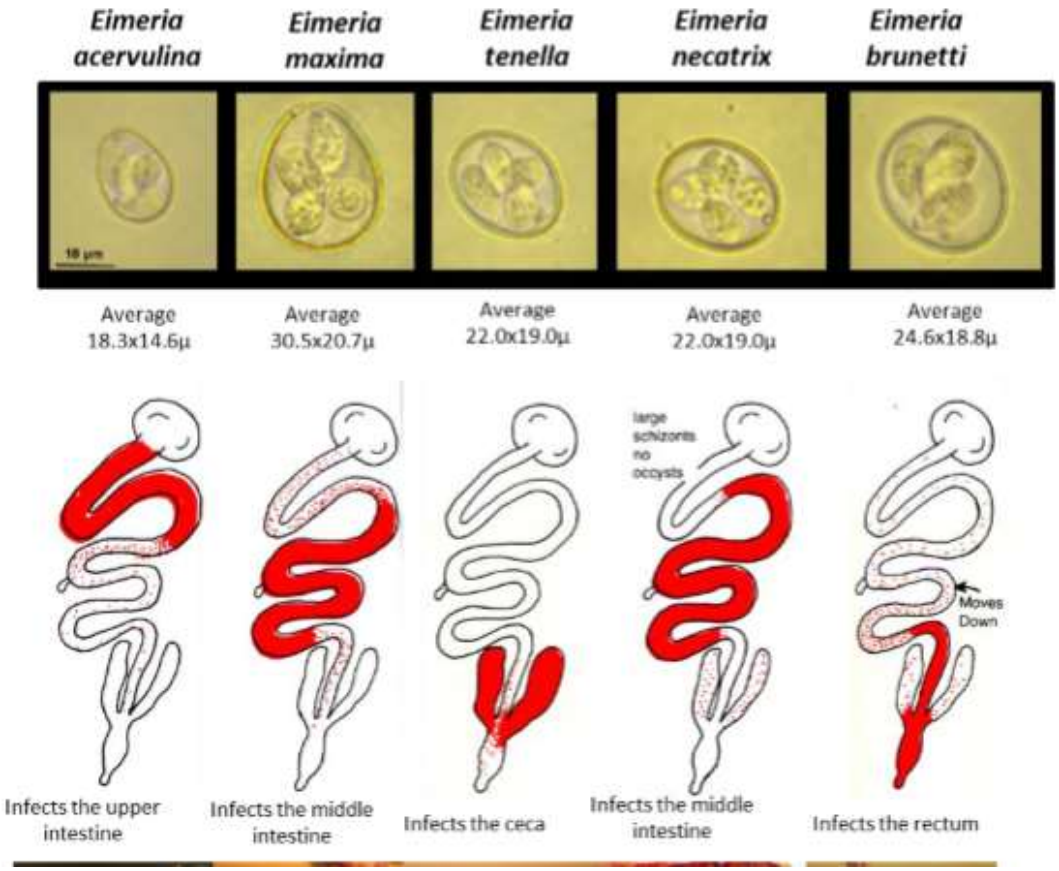
3-To find out the economic importance of the issue of coccidiosis in birds.

Chapter two

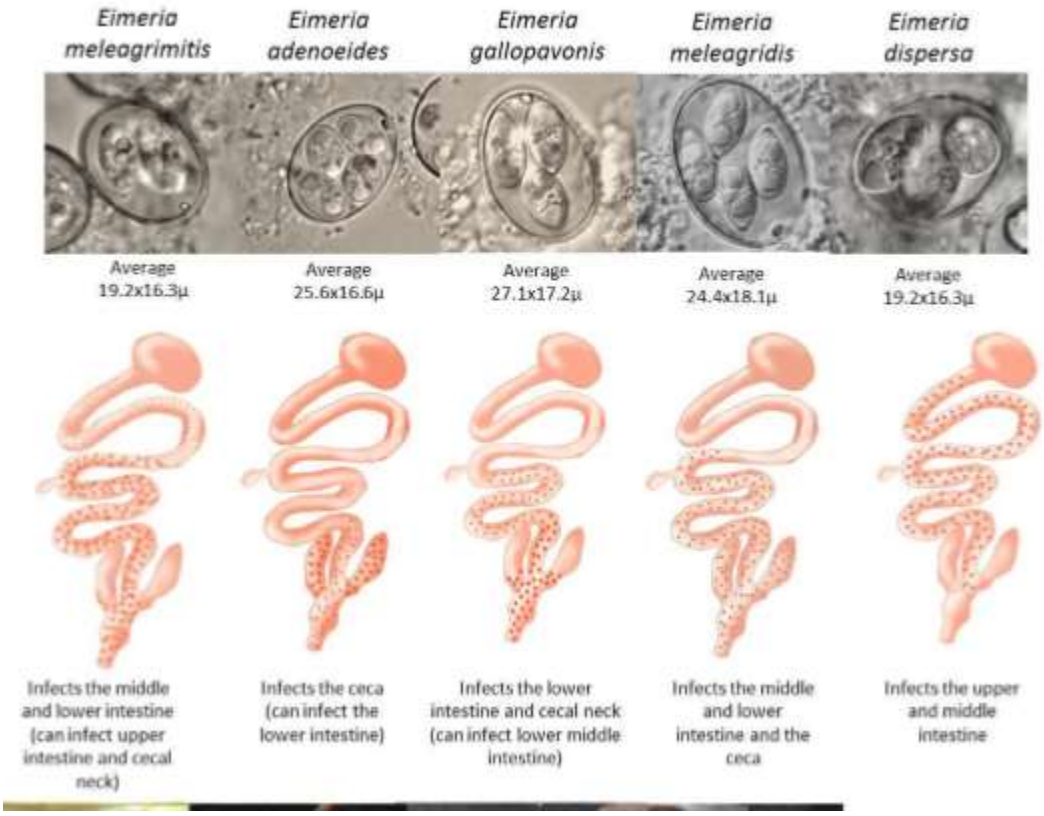
Review and literatures

2.1-Etiology

Most livestock are affected by different types of coccidia. Each type of coccidian infect only one species of livestock – each is species- specific.



(Fig.1)Chicken Eimeria spp.



(Fig.2)Turkey Eimeria spp.

2.2- Scientific classification of parasite

Domain : Eukaryota

Kingdom : Chromalveolata

Superphylum :Alveolata

Phylum : Apicomplexa

Class : Conoidasida

Order : Eucoccidiorida

Family : Eimeriidae

Genus : Eimeria

2.3-Classification :

Protista (unicellular eukaryotes).

Apicomplexa (cell with cluster of organelles known as apicalcomplex).

Coccidia (gamonts small and intercellular , from small resistant spores called oocysts).

Eimeriidae (gametes develop independently without syzygy ; known coccidian parasite). (Allen,2000)

Family :Eimeriidae

These protozoa are known as the entriccoccidia ;monoxenous (one-host) parasite in digestive tract of herbivores or carnivores causing diarrhoeal disease (known as coccidiosis) . Parasite form environmentally – resistant oocysts which undergo faecal – oral transmission between hosts .

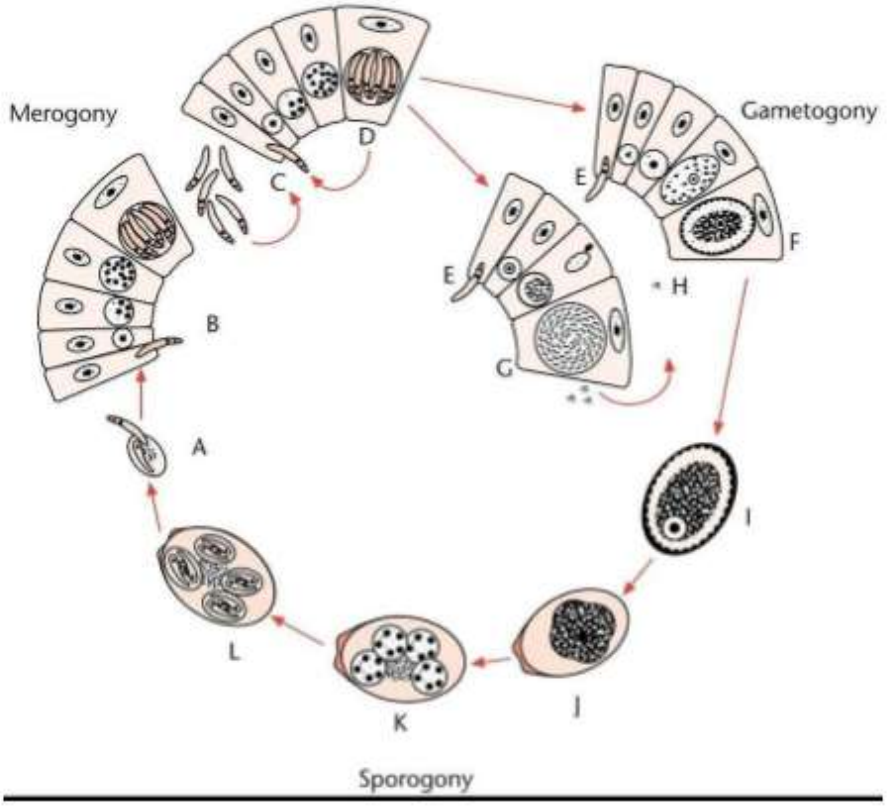
2-4 Life Cycle

The environmentally resistant oocyst contains 4 sporocysts with 2 infective sporozoites per sporocyst, and is transmitted fecal-orally. Sporozoites will then exit the sporocyst to infect a host intestinal epithelial cell and undergo asexual replication for a pre-determined number of cycles. Then sexual replication occurs to produce an uninfective oocyst (MARa,2016) Sporulation occurs outside of the host because only when the oocyst is in the environment at the right temperature, oxygen level and relative humidity does it become infective (FAO, 2014).

Sporulation for most Eimeria species takes about 48 hours to occur (MARa,2016). During the patent period, you will see maximum oocyst shed around 6 to 8 days post inoculation and this will taper out around 10 days .(post inoculation (MARb,2016). The number of Eimeria parasites

ingested will determine the amount of damage to the host. Depending on the Eimeria species, "a few" oocysts would cause an unapparent infection, "thousands" could cause clinical signs and "tens of thousands" could result

in severe coccidiosis and perhaps mortalities in birds that have never been exposed to the disease.



(Fig.3)The life cycle of a typical Eimeria spp.

Sporozoites (A) excyst from sporocysts in the intestinal tract of poultry. Sporozoites infect a host intestinal epithelial cell and initiate asexual replication (B-D). Asexual replication forms motile stages that exit and infect neighbouring host intestinal epithelial cells for a pre-determined number of cycles (C-D). After asexual replication, sexual replication occurs (E-G) where an unsporulated oocyte is formed (H) and is then shed in the feces (I). Sporulation occurs in the environment to produce a sporulated, infective oocyte (I-L) (MARa,2016)

2.5 - Site of infection

Most species undergo endogenous development in the intestinal mucosa(small and/or larg intestines) whereas some species develop in the liver, gall bladder or kidneys. They generally exhibit rigid tissue tropism, infecting host cells in particular locations . (Donaldw,2010).

Eimeria species	**Oocyst size**	**Host species**	**Site of infection**	**Pathogen-necity**
Eimeria acervulina	18x14Mm	chickens	Anterior small intestine	high
E.brunetti	26x22Mm	chickens	Small and large intestine	High
E.maxima	30x20Mm	chickens	Mid small intestine	Moderate
E.Mitis	16x15Mm	chickens	Small and larg intestines	Low
E.necutrix	20x17Mm	chickens	Small intestine, caecum	High
E.Praecox	21x17Mm	chickens	Small intestine	Low
E. tenella	23x19Mm	chickens	Caecum	High
E.adenoides	25x16Mm	Turkeys	Small and large intestines	High
E. dispersa	26x21Mm	Turkeys	Anterior small intestine	Moderate
E.Melegridis	24x18Mm	Turkeys	Caecum	Moderate
E.Melegrimitis	19x16Mm	Turkeys	Anterior small intestine	High

(Fig.4) sit of infection

2.6 - Transmission in the Environment

Chickens get coccidiosis by eating oocysts that have been shed in the dropping of infected chickens. Infected chickens shed oocysts for several days or weeks. Oocysts can also be spread by insects, dust, wild birds, and humans (from shoes and equipment). (Donaldw,2010).

Oocysts can survive many weeks in the outdoors-as long as 600 days. The optimum temperature for sporulation is 72^{o}F. The rate of production is slower if temperatures are much cooler or hotter. Oocysts are killed either by freezing or very high temperature. Sporulation also requires oxygen and moisture (at least 20 percent moisture in the litter for optimal sporulation). Once sporulated, the oocyst remains infected for months if protected from very hot, dry, or freezing conditions. (Fanatico,2006).

In very large poultry houses, oocysts do not last long in the litter because of the action of ammonia released by decomposition of litter and manure and by the action of molds and bacteria. However there are usually so many oocysts that birds continue to pick them up and get sick.(Keith,2013)

2.7 - Clinical Findings

Sings of coccidiosis range from decreased growth rate to a high percentage of visibly sick birds, severe diarrhea or bloody diarrhea, dehydration and high mortality. Feed and water consumption are depressed. Weight loss, development of culls, decreased egg production, and increased mortality may accompany outbreaks. Mild infections of intestinal species, which would otherwise be classed as subclinical, may cause depigmentation and potentially lead to secondary infection, particularly Clostridium spp infection. Survivors of severe infections recover in 10-14 days . (Helm,1999).

The lesions are almost entirely in the intestinal tract and often have a distinctive location and appearance that is useful in diagnosis. (Kennedy ,2001).

2.7.1- Chicken

Coccidiosis caused by ***E. tenella*** first becomes noticeable at about three days after infection. Chickens droop, stop feeding, huddle together, and by the fourth day, blood begins to appear in the droppings. The greatest amount of blood appears by day five or six, and by the eighth or ninth day, the bind is either dead or on the way to recovery. Mortality is highest between the

fourth and sixth days. Death may occur unexpectedly, owing to excessive blood loss. Birds that recover may develop achronic illness as a result of a persistent cecal core. However, the core usually detaches itself by eight to ten days and is shed in the droppings.(Kennedy,2001).

E. necatrix produces major lesions in the anterior and middle portions of the small intestine. Small white sports, usually intermingled with rounded, bright-or dull-red spots of various sizes, can be seen on the serosal surface. This appearance is sometimes described as "salt and pepper". The white spots are diagnostic for *E. necatrix* if clumps of large schizonts can be demonstrated microscopically. In severe cases, the intestinal wall is thickened, and the infected area dilated to 2-2.5 times the normal diameter. The lumen may be filled with blood, mucus, and fluid. Fluid loss may result in marked dehydration. Although the damage is in the small intestine, the sexual phase of the life cycle is completed in the ceca. Oocysts of *E. necatrix* are found only in the ceca. Because of concurrent infections, oocysts of other species may be found in the area of major lesions, misleading the diagnostician. (Kennedy.2001)

E. acervulina is the most common cause of infection. Lesions include numerous whitish, oval or transverse patches in the upper half of the small intestine, which may be easily distinguished on gross examination. The clinical course in a flock is usually protracted and results in poor growth, an increase in culls, and slightly increased mortality. (Merck and Dohme ,2013).

E. brunetti is found in the lower small intestine, rectum, ceca, and cloaca. In moderate infections, the mucosa is pale and disrupted but lacking in discrete foci, and may be thickened. In severe infections, coagulative necrosis and sloghing of the mucosa occurs throughout most of the small intestine. ***E.maxima*** develops in the small intestine, where it causes dilatation and thickening of the wall; petechial hemorrhage; and a reddish, orange, or pink viscous mucous exudate and fluid. The exterior of the midgut often has numerous whitish pinpoint foci, and the area may appear engorged. The oocysts and gametocytes (particularly macrogametocytes),

which are present in the lesions, are distinctly large. (Merck and Dohme,2013).

E. mitis is recognized as pathogenic in the lower small intestine. Lesions are indistinct but may resemble moderate infections of *E. brunette. E. mitis* can be distinguished from *E. brunette* by finding small, round oocysts associated with the lesion. ***E.praecox***, which infects the upper small intestine, does not cause distinct lesions but may decrease rate of growth. The oocysts are larger than those of *E. acervulina* and are numerous in affected areas. The intestinal contents may be watery. *E. praecox* is considered to be of less economic importance than the other species. ***E. hagani and E. mivati*** develop in the anterior part of the small intestine. The lesions of *E. hagani* are indistinct and difficult to characterize. However, *E. mivati* may cause severe lesions similar to those of *E. acervulina*. In severe infections, *E. mivati* may cause reddening of the duodenum because of denuding of the villi. Some consider these species to be of dubious provenance, but work with molecular diagnostics seems to support their validity. (Kennedy.2001)

2.7.2- Turkeys

Only four of the seven species of coccidian in Turkeys are considered pathogenic- *E.adenoeides, E.dispersa, E. gallopavonis, and E. meleagrimitis, E. innocua, E. meleagridis,* and *E.subrotunda* are considered nonpathogenic. Oocysts sporulate within 1-2 days after expulsion from the host; the prepatent period is 4-6 days. (Merck and Dohme,2013).

E. adenoeides and E. gallopavonis infect the lower ileum, ceca, and rectum. These species often cause mortality. The developmental stages are found in the epithelial cells of the villi and crypts. The affected portion of the intestine may be dilated and have a thickened wall. Thick, creamy material or caseous casts in the gut or excreta may contain enormous numers of oocytes. *E. meleagrimitis* chiefly infects the upper and mid small intestine. The lamina propriety or deeper tissues may be parasitized, which may result in necrotic enteritis. *E. disperse* infects the upper small intestine and causes a creamy, mucoid enteritis that involves the entire intestine,

including the ceca. Large numbers of gametocytes and oocyte are associated with the lesions.

Common signs in infected flocks include reduced feed consumption, rapid weight loss, droopiness, ruffed feathers, and severe diarrhea. Wet droppings with mucus are common. Clinical infections are seldom seen in poults >8 wk old. Morbidity and mortality may be high. (Fanatico,2006).

Several factors influence the severity of infection. Some of these include the following:

- The number of oocysts eaten. Generally, an increase in the number of oocysts eaten is accompanied by an increase in the severity of the disease.
- Strain of coccidia. Different strains of a species may vary in pathogenicity.
- Environmental factors affecting the survival of the oocysts.
- Site of development within the host. Coccidia that develop superficially are less pathogenic than those that develop deeper.
- Age of the bird. Young birds are generally more susceptible than older ones.
- Nutritional status of the host. Poorly fed birds more susceptible. (Kennedy.2001).

2.8 - Diagnosis :

Final diagnosis of the disease is based on history, clinical signs, necropsy lesions, fecal flotation to look for the presence of coccidian oocytes (eggs), and microscopic examination of the intestines and ceca to look for coccidian organisms in the tissues. (Helm, 1999).

2.8.1 - Necropsy Finding :

Necropsy done soon after death. It may be possible to identify characteristic lesions or sores in the gut. Coccidiosis causes a thickening of the intestines, which make them feel like a sausage. There may be light-colored spots on the surface of the gut, and inside the gut, hemorrhages and streaks. (Fanatico, 2006).

Except for kidney coccidiosis in geese, all lesions are found in the intestine and ceca of poultry. Lesions can be seen in the upper small intestines to the lower large intestines and ceca, depending on the species of coccidia involved. These lesions can include a red or white speak led appearance of the intestineal wall (coccidian colonies), thickened intestinal wall, intestine and ceca may balloon and be filled with fluid, blood, and tissue debris. (Helm, 1999).

2.8.2- Laboratory Diagnosis :

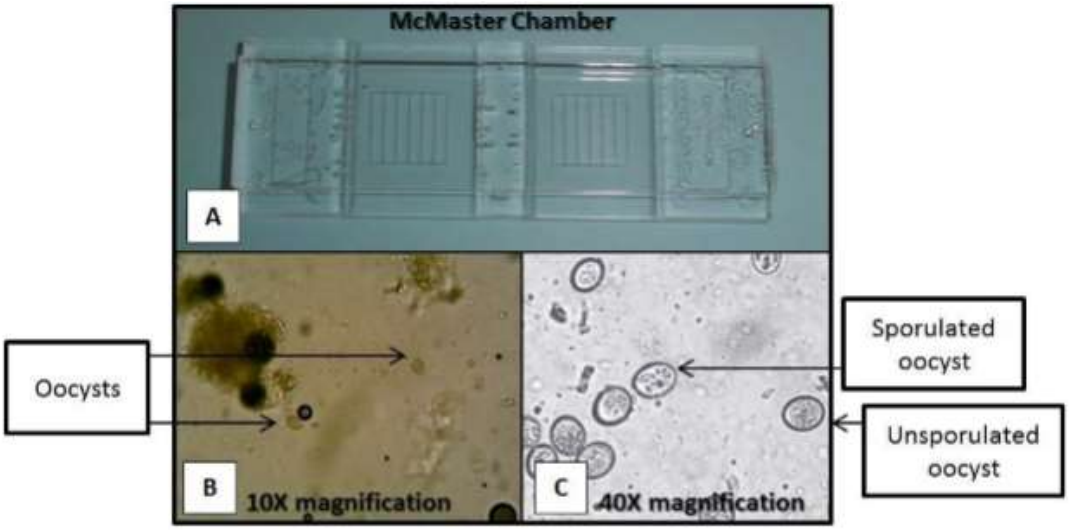
The classical parasitological methods of diagnosis are labor intensive and therefore costly(Mathis,2005). Oocyst per gram (OPG) counts in faeces or litter have a poor relation with the impact of the parasite on the performance of a flock. Identification of different species base on morphology of oocysts is very challenging and requires expertise. Lesion scoring is an interpretation based on macroscopic visible lesions caused by *Eimeria*, usually following a scoring system from zero to four (Allen and Fetterer, 2002). The individual scores for all the species are usually compiled for a certain number of birds (e.g. six) per flock resulting in a Total Mean Lesion Score (TMLS). The method is extremely labor intensive, sometimes subjective and only reliable when performed by skilled people(Williams,1999). The correlation between lesion scores and performance is believed to be stronger than with OPG but still there is a difficult appreciation of the level of lesions towards impact on performance, especially at subclinical level(Mathis,2005). A limitation is for instance the fact that *E. mitis*, although quite pathogenic, does not cause typical lesions and is mostly disregarded when using this method. Lesion scoring still remains the most frequently applied diagnostic method today.(Williams,1999). The seven species of *Eimeria* infecting chickens are considered not equally important. Generally, it is agreed upon that from the

species recognized in broiler chickens, the most pathogenic are *E. acervulina, E. maxima and E. tenella.* The latter is, amongst broiler farmer, the best known. It infects the caeca and because of its deep development in the mucosa and subsequent widespread damage with distinct gross lesions and loss of blood in the faeces, it is easily recognized also by farmers(Conway and Mckenzie,2007). On the other hand, when performing field necropsies on a larger scale, *E. tenella* appears to be the least prevalent of the three species mentioned. Also, the damage is being limited to the caeca, relative less important parts of the gut with regard to digestion and absorption, thus effects on growth and feed conversion rate.(Conway and Mckenzie, 2007). Diagnosis of clinical disease caused by *E. tenella* is quite easy and action can be swift. These facts make its impact on the productivity of the broiler industry is relatively limited compared to the other species, although many broiler farmers associate coccidiosis only with caecal coccidiosis. This is a good example of perception not being in accordance with the facts. *E. acervulina and E. maxima*, both much more prevalent, are less perceived to be related with clinical coccidiosis in the field. *E. acervulina* is causing white lesions in duodenum and in heavier infections also more caudal, interfering even with the ability for *E. maxima* to develop (Mathis, 2005). *E. maxima* causes petechiae in the mid gut. To assess the level of damage caused by these two species, lesion scoring can be performed. An important debate is still ongoing on what levels are to be considered clinical (and requiring treatment) and what levels are subclinical. Some consider lesions higher than 1.5 per species as indicative for clinical disease, and levels below as subclinical, not requiring treatment. *E. praecox and E. mitis* are not scored for and are completely disregarded using the lesion scoring method, although both species are shown to be able to cause

losses through an increased feed coversion rate and in the latter case even morbidity (Fanatico, 2000 ; Williams,1998). Moreover, it has been demonstrated there can be a poor relation between macroscopic and microscopic lesions, emphasizing using macroscopic lesion scoring alone is not suitable to detect all economical relevant coccidiosis infections. It is frequently disregarded that all macroscopic, but also microscopic lesions, in fact any infection of coccidia, requires an invasion and thus destruction of host cells. This is both true when the parasitical life cycle can complete, but even so when an intervention of the immune system occurs. In the latter case not only host cells are destroyed, but also the activation of the immune system requires use of nutrients that cannot be addressed to the conversion of nutrients into meat, the ultimate goal of broiler production. As a consequence it is important to understand that any level of coccidiosis is causing areal, but difficult to quantify, loss in performance. As coccidiosis is a disease that cannot currently be eradicated, the objective of coccidiosis prevention is finding the economical optimal balance between costs of diagnosis, prevention, treatment and development of host immunity while trying to keep the subclinical loss as low as possible. It is clear that producers achieving a better balance will have a competitive advantage over other producers.(Allen and Fetterer,2002).

Coccidial infections in chickens cause significant economic losses because of decreased growth and feed utilization. To detect infection, gross evaluation of the intestinal tract and a microscopic evaluation of a wet smear are used as routine diagnostic methods of diagnosis when we compare qualitative method (Fecal Floatation) with other methods of diagnosis (wet

smear), we find the floatation fastest and most accurate a method for detection of coccidian. (Al Jumaili. 2013).



(Fig.5) McMaster chamber

(A) that can be used for counting oocysts. Oocysts in a fecal sample can be viewed at low (B) and high (C) power magnification with background plant material and air bubbles. At a higher magnification (C) it is easier to view sporulated versus unsporulated oocysts. Photo credits: A, B – (Kobus et.al,1994)

2.8.3- Molecular Diagnosis :

Techniques are a major addition for scientific research and more practical applications such as establishing vaccine quality control, but unfortunately, the lack of a rapid, low-cost and especially quantitative test is preventing their broad scale use. The main application of these techniques for field diagnosticians today is the possibility of defining presence of species currently disregarded such as *E. praecox and E. mitis*. Still, the lack of quantitative aspect of the techniques is preventing an accurate appreciation of different coccidial species certainly with the widespread use of ionospheres that also allow some multiplication of sensitive parasites.

A very innovative technique can be found on a website (Gruber *et al.*, 2007) and is called Coccimorph. This is a computational approach for parasite diagnosis, in this case *Eimeria spp*. From chicken. Images from sporulated oocysts from a confirmed species were assessed on different features: curvature characterization, size and symmetry and internal structure characterization. Users can upload their digital images from unidentified oocysts and have the program identify the species concerned. This is very accessible and the low cost is a major advantage. A disadvantage is only speculated oocytes can be identified, which limits the use of this technique to litter sample identification only.(Naciri *et al.*,2003;Peek and Landman,2003).

Real-time PCR uses species-specific DNA probes to both detect and quantify the species present. The advantage of these types of assays over traditional PCR is that they have exceptional sensitivity and the amount of target DNA can be quantified.

2.8.4 ELISAs

Can detect antibodies in blood (sera) samples and can be designed so that they detect epitope-specific antibodies. Since they detect antibodies rather than the parasites themselves, ELISAs have the advantage that they are able to identify the species to which chickens have developed an immune response even when the parasites are no longer present. They also provide the ability to assess immune responses following vaccination. ELISAs are high through assays that allow rapid screening of large numbers of samples and are common technology in most diagnostic laboratories. (Youn and Noh,2001).

For the serological assays species-specific immune blotting tests were developed that could distinguish between birds infected with *E. tenella or E. necatrix* from birds infected with other species. Attempts to develop a competitive ELSA using monoclonal antibodies specific to gametocyte epitopes were not successful so an indirect ELISA was developed from *E. tenella* merozoite antigens. The indirect ELISA was used to assess sera from vaccinated and unvaccinated birds from commercial farms. (Morgan,2009).

2.8.5 - Species Determination

There are seven valid species of chicken coccidian, *E. acervulina, E. brunette, E. maxima, E. mitis, E. necatrix, E.* praecox, and *E., tenella,* each species developing in a particular location within the chick digestive tract. It is common to find at least six species (e.g., *E. acervulina, E. maxima, E. tenella, E. brunette, E. mitis, and E. praecox*) in litter samples from a single flock during its first 6 weeks. (Youn and Noh.2001). five of the species, *E. acervulina, E. brunette, E. maxima, E. necatrix,* and *E. tenella,* are well known and identifiable with relative ease, because they produce characteristic gross lesions. Their pathogenicities range from moderate to severe. On the other hand, *E. praecox and E. mitis* do not kill chickens or produce path gnomic lesions and often have been considered to be benign. However, experimental infections result in enteritis, diarrhea, and reduced feed efficiencies (Williams and Catchpole, 2000) indicating that two species certainly can cause commercial losses and hence need to be controlled.

Generally, the five most pathogenic species listed above can be differentiated in the host on the basis of clinical signs, characteristic lesions at particular sites of infection in the chicken intestine, and consideration of

prepatent period, size of oocysts, and morphology of intracellular stages. However, the less pathogenic species such as *E. mitis and E. praecox* might be overlooked. Standard diagnostic and testing procedures for *Eimeria* species and includes colored photographs of gross lesions caused by the most commonly found species.(Morgan,2009).

Shirly (Aleen and Fetterer. 2002) was the first to use a molecular biological approach to differentiate species on the basis of isonzyme that rRNA and rDNA probes could be used to identify individual species through characteristic restriction fragment patterns. Procaine et al used a randomly amplified polymorphic DNA assay to differentiate *E.acervulina* and *E. tenella* and detect within-strain differences. Recombinant DNA techniques have been used to discriminate different strains of E. tenella (Shirley and Harvey, 2000) and develop markers for precocious and drug-resistant strains (Shirley and Harvey, 2000) and PCR amplification of internal transcribed spacer region I from genomic DNA has been used to detect and differentiate six Eimeria species (Rothwell, and Kaiser, 2000). Eight species (including *E. hagani)* are claimed to be differentiated using a two-step PCR procedure and six species have been characterized using a PCR-linked restriction fragment length polymorphism approach (Gasser, 2000). These PCR methods should prove very useful for epidemiological surveys of avain coccidiosis.

2.9 - Differential Diagnosis

2.9.1 - Necrotic enteritis:

is an a cut enter toxemia. The clinical illness is usually very short, and often the only signs are a severe depression followed quickly by a sudden increase in flock mortality. The disease primarily affects broiler chickens (2-6 wk old) and turkeys (7-12 wk old) raised on litter but can also affect commercial layer pullets raised in cages. Early mortality is often related to coccidiosis vaccination programs, with *Eimeria* cycling in these flocks. (Immerseel ,2004)

Most often the only sign of necrotic enteritis in a flock is a sudden increase in mortality. However, birds with depression, ruffled feathers, and diarrhea may also be seen. The gross lesions are primarily found in the small intestine. (Jejunum/ileum), with may be ballooned, friable, and contain a foul-smelling, brown fluid. The mucosa is usually covered with a tan to yellow pseudomembrance often referred to as a "Turkish towel" in appearance. This pseudomen brane may extend throughout the small intestine or be localized. The disease, usually persists in flock for 5-10 days and mortality is 2%-50%. (Hargis, 2014).

2.9.2- Ulcerative enteritis

In this disease ,sudden death occurs without signs or weight loss and with up to 100% mortality in just 2-3 days. Acute lesions include hemorrhagic enteritis of the duodenum. In chickens and other game birds, the course of the disease is less sever and is accompanied by anorexia. Signs are similar to those seen in coccidiosis: depressed, listless birds with humped backs, ruffled feathers, diarrhea, and sometimes bloody or watery white droppings especially in quail in the prolonged course. Chickens recover within 2-3 wks., and mortality rarely exceed 10%. (Raul,2013).

In early disease stages, the most common lesions include small, round ulcers surrounded by hemorrhages in the small intestine, ceca, and upper large intestine. The presence of blood in the gut resembles coccidiosis. Characteristic yellow to gray necrotic foci are the predominant lesions in the hepatic parenchyma. Splenomegaly with hemorrhages and nicrotic areas may be present. (Yegani,2008).

2.10 Management

2.10.1 vaccine

Commercial poultry farming is expanding day by day and contributing in the provision of affordable and high quality proteins (Ahmad et al., 2010; Ghafoor *et al.,* 2010). However, this sector is still confronted with many enteric diseases like coccidiosis which are hindering its progress (Saima *et al.,* 2010; Hafez, 2011).

Avian coccidiosis in an intestinal protozoan disease cause by various species belonging to genus *Eimeria*. According to a recent estimate (Chapman, 2009), the United States poultry industry costs about US$127 million annual losses just because of coccidiosis and proportionally similar losses may be faced by the poultry producers in various parts of the world. Thus, in commercial poultry systems, coccidiosis is thought to be the one of the most expensive infectious diseases. Thus far, chemoprophylaxis and anticoccidial feed additives have controlled the disease but situation has been complicated by the emergence of drug resistant strains against commonly used drugs (Abbas et al., 2008; Abbas *et al.,* 2011a).

Vaccination by using live coccidial oocysts has been another effective approach for coccidiosis control (Shirley and Lillehoj, 2012), but, in poorly managed production systems particularly in case of broiler birds, live vaccines may result in the onset of severe reactions ultimately affecting the performance and production of flocks (Chapman, 2000). As a result of this drawback of live vaccines, attenuated vaccines, having reduced pathogenicity, have been developed, but these are expensive to produce. The other drawback of using vaccines is diversity of *Eimeria* strains in different geographical distributions. Therefore, vaccine strain, effective in one geographical area may not be effective in the other area.

Because of development of drug resistance and pathogenicity associated problems with live vaccines, poultry producers all over the world are moving towards alternative control of avain coccidiosis. Cost effective alternative strategies are being sought for more effective and safer control of avian coccidiosis (Abbas *et al*., 2011b, 2011c; Abbas *et al.,* 2012; Arczewska-Wlosek and Swiatkiewicz, 2012; Zaman et al., 2011).

2.10.2 - Management for Control

Management has always been important to coccidiosis control, especially before drugs were available. Management focuses on reducing the number of coccidia to keep infection at a minimum until immunity is established.

2.10.3 - Natural Immunity

A small-scale, low-density production system can allow a low level of exposure to coccidia, which permits the chick to develop immunity without triggering the disease. However, birds may not pick up enough parasites to cause immunity, or they may be overwhelmed by too many. In addition, immunity is only species-specific. Exposure to one type of coccidia will not protect a chicken from the other six types that can infect it. (Onaga *et al*., 2005)

Early detection is a management method to avoid the use of preventative medication. Early detection requires close observation and experience. Watch feed intake in particular—it goes down in the early stages of coccidiosis.

The choice of production system is an importance management decision. High-density, large-scale production almost always requires the use of anticoccidial medication. In contrast, in low-density, small-scale production, the birds tend to stay ahead of the parasites and may not require medication. Many small-scale producers do not use anticoccidial medication; however, as the size of the flocks grow, more problems are encountered and more management is required for natural immunity. Immunity is especially important in turkeys, layers, breeders, and slow-growing broilers that are kept longer than fast-growing broilers marketed at a younger age. (Fanatico,2006).

When chicks are brooded in a separate area before moving them to the go out facility (two-stage production), the brooder stays clean of infective oocytes since fast growing broilers do not remain past three weeks of age. However, chicks are at risk for coccidiosis if they stay in the brooder longer

than three weeks. Pullet chicks for egg laying grow slowly and stay in the brooder longer. If chicks are brooded and grown out in the same facility (one-stage production), they seed the area with coccidia. These birds may require a lower density or, possibly, medication. The following management strategies for good brooding can help. (Williams, 2002).

2.10.4 - Litter Management

The litter must be dry to reduce sporulation of oocytes. Remove any wet or crusted litter. Moisture in the litter is affected by the following:

- Heat source: A propane radiant brooder heats a larger area and dries out litter more than a heat lamp.
- Ventilation: Housing should prevent drafts but not be airtight. Humidity, along with ammonia and other gases, needs to escape.
- Water leaks: Water leaks must be prevented.
- Condensation: Condensation may occur in buildings with insulated roofs and walls and will contribute to litter moisture.
- Feed: Rations with excessive protein or excessive salt can result in wet litter.(Plamondon,2002a).

In the large-scale industry, "new-house coccidiosis syndrome" sometimes occurs when birds are placed on brand-new litter. There is no low-level population of coccidia to establish immunity. So the flock is more susceptible, coccidiosis problems are more likely, and medication may be needed.

Some small flock producers are interested in the built-up or composting litter as an ecosystem of microbes.

Poultry-house litter becomes significantly anti-coccidial after about six months' use, as organisms that eat coccidia start to thrive and knock down the coccidia population…. By never removing more than half the brooder house litter at a time, it can keep its anti-microbial properties indefinitely. (Plamondon, 2002a).

recommend starting with at least six inches of shavings and adding a thin layer of fresh litter on top, which will prevent chicks from eating old litter at first, And keep it from getting packed down and crusted over. If the litter seems too wet, more dry litter. removes litter when it is too deep to manage or when too wet. (Plamondon, 2002b).

Also, although oocysts can be destroyed by microbes in the litter and soil, there may be so many oocysts that the birds become infected. Unfortunately, there is little scientific information available on composting litter.

Chapter three

Conclusion :

1. Coccidiosis disease of poultry is widespread in almost all parts of the world.

2. Coccidiosis disease that causes significant economic losses among poultry flocks.

3. The problems related to coccidiosis change with seasonal change on year .

4. May be used anticoccidial programs to limit cases of subclinical coccidiosis .

5.Life vaccines could have an interesting role to boost the efficacy of anticoccidial program.

Recommendation :

There is important recommendation to prevent infection with coccidiosis:

1- Provision of vaccines needed to curb the spread of the disease in specialized veterinary centers

2- Adherence to preventive measures and very good management is essential for the control and prevention of coccidiosis.

3- Reducing crowding and controlling humidity, especially in the autumn season. keep the litter dry to reduce sporulation of oosystgs. Remove any wet or crusted litter.

4- anticoccidials are given in the feed to prevent disease and economic loss often associated with subacute infection.

Chapter four

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