COCCIDIOSIS

Clinically, "coccidiosis" is applied to the diseases produced by protozoa of t he genera Eimeria and Isospora, in the family Eimeriidae, order Eucoccidiorida.

Many coccidia affect animals and birds. The species and the tissues attacked depend upon the Obligate preferences Of each parasite (Le., preferences biologically essential for their survival). Coccidiosis is particularly common in cattle, sheep, and poultry (see avian coccidiosis), and is a disease of great economic importance.

Important Eimeria and Isospora spp. affecting Animals

ANIMAL Affected	Infective Agent	Main Part Affected
CATTLE	Eimeria bovis	Small Intestine, Caecum, Colon
	Eimeria zuernii	
	Eimeria ellipsoidalis	
SHEEP	Eimeria ovina	Intestines
	Eimeria ovinoidalis	
	Eimeria parva	
GOAT	Eimeria arloingi	Intestines
	Eimeria caprovina	
	Eimeria faurei	
PIG	Eimeria porci ; E. debliecki	Intestines
	Eimeria scabra	
	Isospora suis	
DOGS	Isospora canis	Intestines
	Isospora burrowsi	
	Isospora neorivolta	
CATS	Isospora rivolta	Intestines
	Isospora felis	
HORSES	Isospora leuckarti	Intestines
RABBITS	**Eimeria stiedae**	**Bile Duct / Liver** (Hepatic Coccidiosis)
	Eimeria perforans	Intestines

CHICKEN	**Eimeria tenella**	**Caecum** (Caecal Coccidiosis)
	Eimeria necatrix	Middle portion of Small Intestine
	Eimeria brunetti	Lower portion of Small Intestine
	Eimeria maxima	Middle portion of Small Intestine
	Eimeria acervulina	Upper portion of Duodenum (Small Intestine)
	Eimeria mitis	Duodenum
	Eimeria praecox	Duodenum
TURKEYS	Eimeria meleagriditis	Small Intestine
	Eimeria adenoeides	Small Intestine & Caecum
GEESE	Eimeria anseris	
	Eimeria truncata	**Kidneys**

Coccidia are a protozoa, that are single-celled parasitic organisms and are OBLIGATE INTRACELLULAR PARASITES of Intestinal Epithelium and includes 3 Genera:

(1). Eimeria, (2). Isospora, and (3). Cryptosporidium

Most Coccidia in Poultry and Ruminants belong to the Genus: Eimeria. There are seven important disease producing coccidian species in poultry as are tabulated above.

Coccidiosis is largely a disease of YOUNG BIRDS, because immunity quickly develops after parasitic exposure. It is characterized by bloody diarrhoea and high mortality. Outbreaks are common between 3-6 weeks of age. There is No Cross-Immunity (cross protection) between different species of Eimeria & later outbreaks may result from different species.

Coccidiosis mainly occurs under conditions of Overcrowding that favours build-up of the parasite in disease producing numbers. Coccidiosis is particularly important under INTENSIVE POULTRY Operations and inflicts heavy mortality each year mainly in Broilers & also Growers raised on deep litter.

Life Cycle:

It is divided into 3 Phases: (I) SPORULATION, (II) Infection & SCHIZOGONY (Asexual Reproduction), and (III) GAMETOGONY & OOCYST formation (Sexual Reproduction)

I. SPORULATION

Unsporulated Oocyst (nucleated mass of protoplasm in resistant wall) is passed in faeces

On Soil; with Suitable oxygenation, high humidity; temp 27*C

Nucleus of each **Unsporulated Oocyst** divides Twice – to form 4 Conical Bodies i.e. **4 SPOROBLASTS**

Each of 4 Sporoblast, secretes a Refractile Wall & transform to form **4 SPOROCYSTS**

Protoplasm inside each Sporocyst further divides into Two Banana Shaped SPOROZOITES each

Now OOCYST encloses 4 Sporocysts, each containing 2 Sporozoites each; and this is referred as SPORULATED OOCYST

Unsporulated Oocysts ⇒ Sporoblasts (4) ⇒ Sporocysts (4) ⇒ forms Sporozoite (2 inside each sporocyst; *so 1 unsporulated oocyst leads to 4 sporocysts or 8 sporozoites in them*)

Sporulated Oocyst (*each Sporocysts with 2 Sporozoites each*) are the infective stages of Coccidia

II. INFECTION & SCHIZOGONY (Stage of Asexual Reproduction)

<table>
<tr><td colspan="2">HOST infected by ingesting Sporulated Oocyst</td></tr>
<tr><td colspan="2">⇓</td></tr>
<tr><td colspan="2">Reach in Intestine – Mechanically of by Co2 tension – the Sporocysts are Liberated</td></tr>
<tr><td colspan="2">⇓</td></tr>
<tr><td colspan="2">SPOROZOITES inside Sporocyst are activated by Trypsin & Bile;
& so the Sporozoites leaves out of sporocyst</td></tr>
<tr><td colspan="2">⇓</td></tr>
<tr><td colspan="2">SPOROZOITES enter Epithelial cells of Intestine – and transform into TROPHOZOITES</td></tr>
<tr><td colspan="2">↔</td></tr>
<tr><td>In most coccidian species –
Each Sporozoite enters INTESTINAL Epithelial Cells – Grows – Rounds Up
and forms whats k.a. TROPHOZOITE</td><td>In few other species, like E. tenella, the Sporozoite penetrates Epithelium – then are taken-up by Macrophages deeper into the Lamina propria of Villi - and transported deep into epithelial mucosa – where they leave macrophages and enter Epithelial Cells to form TROPHOZOITES</td></tr>
</table>

In few days, TROPHOZOITE, divides by Multiple fission to form Schizont.

SCHIZONT: is a structure consisting large number of elongated nucleated structures that are known as MEROZOITES. When the divisions are complete, schizont is fully matures

and it is then that HOST CELLS and the SCHIZONT ruptures and MEROZOITES are released, escape to enter/infect neighbouring cells. Schizogony may be repeated and number of Schizont generations vary depending on species.

The sporozoite gradually increases in size, becomes first a trophozoite and finally a schizont. The schizont completely fills the cytoplasm of the epithelial cell, displacing the nucleus to one side. Each mature schizont contains many elongated structures (spores), which are similar morphologically to sporozoites, but are known as merozoites. The schizont then ruptures its own wall and also that of the epithelial cell's, liberating the merozoites. The merozoites infect other epithelial cells and continue this asexual life cycle.

At certain stage, some of the merozoites enter into the sexual phase of the cycle, known as gametogenesis or gametogony. Each of these merozoites develops within an individual epithelial cell into a female form, a macrogamete, or into a male counterpart, a microgametocyte. The microgametocyte finally ruptures to release a **large number of tiny motile microgametes**. One of the microgametes unites with a **single macrogamete.** Once thus fertilized, the macrogamete soon becomes an oocyst.

III. GAMETOGONY & OOCYST Formation (Stage of Sexual Reproduction)

Schizogony (Asexual) terminates when Merozoites – give rise to – Male & Female Gametocytes. The factors responsible are not fully known.

MACROGAMETOCYTES are Females. It is Unicellular but increases in size to fill parasiticized cells.*These differ from Trophozoites / developing Schizonts by Single-Large Nucleus.

MICROGAMETOCYTES are males. It undergoes Repeated Divisions to Form Large number of Flagellated Uninucleate Organisms k.a **MICROGAMETES**. Only during this brief period, the Coccidia have Organs of Locomotion.

MICROGAMETES ⇒ freed by rupture of Host Cells ⇒ penetrates the **MACROGAMETE** ↵

Fusion of Nucleus takes place with formation of Cyst Wall around resulting in **ZYGOTE**. Its k.a **OOCYST**. These Oocysts are liberated in the faeces

TRANSMISSION:

The coccidian infection may spread by:

- ✓ Ingestion of infective stage of coccidian (Sporulated Oocyst)
- ✓ Mechanically through Fomites, movement of People,
- ✓ Cooler & wetter humid Weather is most favourable for infections

PATHOGENESIS:

Coccidia of domestic animals pass all the stages of their cycle in the alimentary mucosa and do not invade other organs, although schizonts have been found in the mesenteric lymph nodes in some cases. The different species of coccidia show a tendency to localize in different parts of the intestine.

The coccidial life cycle is self-limiting. As described under life cycle, sporozoites are released from the ingested oocysts. Sporozoites invade the intestinal epithelium, and develop into asexual schizonts. After the schizont matures, merozoites are released by rupture of the epithelial cell. New epithelial cells are again invaded and second generation schizogony occurs in the large intestine. This is followed by the release of second generation of merozoites which invade epithelial cells and produce the sexual stages, the macrogametocyte and microgametocyte. The second generation schizogony and fertilization of the macrogametocyte by the microgametocyte (gametogony) are the stages of the life cycle which cause functional and structural lesions of the large intestine.

As the second generation Schizonts (gamonts) mature, the cells containing them slough from the basement membrane, and cause haemorrhage and destruction of the caecum and colon. This period is termed **Prepatent period** - which is the period between the time of introduction of parasitic organisms into the body and their appearance in the blood, tissues, or faeces.

SIGNS & LESIONS:

In domestic Poultry, coccidiosis is divided in two forms:

1. Caecal Coccidiosis 2. Intestinal Coccidiosis

1. CAECAL COCCIDIOSIS:

Eimeria tenella is primarily responsible for caecal coccidiosis, although Gametogenous stages of *Eimeria necatrix* also takes place in Caecum.

It occurs principally in Chicken of 3-7 week age. **Prepatent period is 7 Days** & ovoid oocyst Sporulate in 2-3 days under normal conditions in poultry houses.

It is a severe disease associated with Bloody Droppings, high mortality, Reduced weight gain & Emaciation. Most mortality occur within 5-6 Days of infection with sudden onset of bloody diarrhoea.

The caeca may be greatly enlarged, dilated and distended with mixture of clotted & unclotted blood. In long standing cases, caecal contents become Caseous & adherent to mucosa.

As regeneration of mucosa occurs, these caecal plugs are detached and Caseous material is shed in the faeces.

2. INTESTINAL COCCIDIOSIS

Eimeria necatrix, E brunette, and others (except E. tenella) are main causative agents of Intestinal Coccidiosis.

Coccidia are Obligate Intracellularly Parasites and total effect on birds depend on Initial infecting Dose of Eimeria sporulated Oocysts.

The transmission is often via faecal-oral route. Interestingly each Eimeria specie is unique both Immunologically (i.e. exposure of one coccidian sp. do not afford protection to other specie of coccidia) AND also in its ability to parasitizes a Specific portion of GI tract of Chickens.

Therefore the Clinical signs and Pathological changes may also vary. The **Prepatent Period vary from 4-7 Days**. The Clinical disease occurs 3-4 days after / following the ingestion of large number of Oocysts. When many cells of Intestinal Epithelium are attacked, the denuded mucosa bleeds freely, showing frank hemorrhages. An intense inflammation (**ENTERITIS**) ensues that also involves the lamina propria & sometimes Submucosa. Continued epithelial destruction & regeneration of rest epithelium to replace the loss, leads to HYPERPLASIA of Intestinal Epithelium, which is cast into Long Papillary Folds (Fronds).

Various stages of Schizogony are seen in Erosive / Hemorrhages stage of lesion while in contrast stages of GAMETOGENESIS are present in large numbers in lesions showing Hyperplasia.

The most pathogenic coccidian leading to Intestinal Coccidiosis are ***E. nacatrix***. It affects middle portion of Small Intestine which when infected is usually **distended Twice** its normal size (Balooning) and the Lumen may be filled with Blood. From serosal surface, infection appears as Small White Plaques and Tiny red hemorrhages.

In ***E. brunette*** - the small intestine is covered with Tiny hemorrhages and Clotted blood and mucosa are seen in droppings.

In ***E. acervulina*** – Duodenum is involved & mucosa may be covered with **White plaques, which tend to arrange in Transverse fashion** giving Ladder-like appearance because of Striations.

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In TURKEYS, Coccidiosis is due to two species:

(i) Eimeria meleagrimitis
(ii) E. adenoides

E. meleagrimitis infection occurs only in Small Intestine and it has Three (3) generation of Schizogony & disease occur with rupture of 3rd stages Schizonts, approx 4 days after infection.

E. adenoids – occurs in Small Intestine and Caecum, and it has Two generation of Schizonts and Clinical Sign appear 4 days after infection with second stage Schizonts.

The **Prepatent period for both is FIVE Days**.

DIAGNOSIS:

1. Clinical Signs and Pathological Lesions
2. Demonstration of Eimeria stages in Epithelial Scrappings and Oocysts in Faeces. Direct smears can be prepared on slides and observed under a microscope

COCCIDIOSIS IN CATTLE

The two species of coccidia which are considered most pathogenic to cattle are *E. bovis* and *E. zuernii.* Their life cycles are similar. The gametocytes are the pathogenic stages and cause rupture of the cells they invade. This results in exfoliation of the epithelial lining of the intestine. The oocyst count is usually low when the disease is at its peak. This is because the oocysts have not yet formed. Exfoliation of the mucosa causes diarrhoea. In severe cases, haemorrhage into the intestinal lumen and the resulting haemorrhagic anaemia, may be fatal. If the animal survives this stage, the life cycle of the coccidian terminates without further damage. The intestinal mucosa regenerates and returns to normal.

Coccidiosis in cattle is immunosuppressive which increases their susceptibility to other common infections. Neutrophil function may be inhibited.

CLINICAL SIGNS

Coccidiosis affects the host in many ways. This depends on the tissue affinity of the particular coccidia, and the number of oocysts present in the initial infection. Most coccidia attack the mucosa of the intestinal tract. Therefore, symptoms are mainly enteric. Clinical signs include sudden onset of bloody diarrhoea, with fever, followed by dehydration. emaciation, and sometimes death.

LESIONS

Coccidia are obligate intracellular parasites. (An obligate parasite is one that can exist only at the expense of another organism. Biologically this is essential for its survival). Development of coccidia within the cytoplasm 0 f epithelial cells results in the death of each cell which is parasitized. The total effect on the animal depends on:

1. The initial infecting dose of oocysts. This determines the number of cells invaded at the outset by sporozoites, and

2. Spread of infection during schizogony. This is affected to a great extent by immunity acquired by the host.

As increasing numbers of organisms enter the sexual phase (gametogenesis), infection of new cells by merozoites decreases and the disease gradually becomes less intense.

When many cells of the intestinal epithelium are attacked at one time, the denuded mucosa bleeds freely. An intense inflammation involves the lamina propria and sometimes the submucosa. As large numbers of epithelial cells are destroyed, the remaining epithelium regenerates to replace that which was lost. This leads to hyperplasia of the intestinal epithelium, which is cast into long papillary folds (fronds). This is because replacement of epithelial cells exceeds their loss. In lesions showing hyperplasia, coccidia in various stages of gametogenesis are present in large numbers. This is in contrast to the erosive, haemorrhagic stages, in which coccidia in various stages of schizogony are most common.

Certain coccidia attack cells other than those of the intestinal tract. The most important of these is hepatic coccidiosis **in rabbits** due to ***E. stiedae***, which affects the intrahepatic biliary epithelium. In the early lesions, there is destruction of the biliary epithelium, but when course of the disease, is somewhat longer, proliferation of this epithelium is the main feature. The bile ducts become greatly enlarged by proliferation of epithelium, which is thrown up into papillary folds resembling adenomatous hyperplasia. These greatly enlarged portions of the bile ducts displace the adjoining liver parenchyma and appear grossly as irregular greyish areas, seen as depressions in the surface of the capsule.

Gross lesions include intensely congested, eroded, and bleeding areas of certain portions of the small intestine. Sometimes these alternate with, or are replaced by, areas in which the mucosa is thickened.

DIAGNOSIS

- ✓ The clinical diagnosis is usually based on the presence of **oocysts in faecal samples**, associated with **sudden onset of typical bloody diarrhoea**.
- ✓ The microscopic lesions are characteristic. These are confirmed by demonstration of the organisms in tissue sections.