

## CLINICAL SIGNS

Diarrhea (often biphasic) which varies in its severity from loose feces to persistent fluid diarrhea. The second phase of diarrhea is initiated by reinvasion from tissue stages.

Source of infection seems to be oocysts produced by sow during periparturient period, piglets being infected initially by coprophagia

## DIAGNOSIS

Difficult unless you have pm materials since clinical signs occur prior to the shedding of Oocysts and are very similar to those caused by other pathogens E.g. rotavirus.

## TREATMENT

Amprolium orally to affected piglets

## PREVENTION

Amprolium in feed to sows during periparturient period. (i.e. a wk prior to farrowing to 3wks post farrowing).

## AVIAN COCCIDIOSIS

*EIMERIA tenella*

*EIMERIA necatrix*

*EIMERIA brunetti*

*EIMERIA maxima*

*EIMERIA acerrulina*

*EIMERIA mitis*

} chicken

*EIMERIA meleagrimitis* turkey

*EIMERIA adenoides* }

*EIMERIA arisens*

*EIMERIA nocens*

*EIMERIA truncata* (kidney)

} Geese

## COCCIDIA OF THE DOMESTIC FOWL

The coccidia of the domestic fowl are responsible for substantial losses to the poultry industry in various countries of the world.

The following spp have been reported from the domestic chicken:

<i>Eimeria acervulina</i>	<i>Eimeria maxima</i>	<i>Eimeria necatrix</i>
<i>Eimeria brunetti</i>	<i>Eimeria mitis</i>	<i>Eimeria praecox</i>
<i>Eimeria hagani</i>	<i>Eimeria mivati</i>	<i>Eimeria tenella</i>

*Eimeria tenella* and *Eimeria necatrix* are the most pathogenic and important spp in domestic poultry.

*Eimeria acervulina*, *E. maxima* and *Eimeria mivati* are common and slightly to moderately pathogenic

*Eimeria brunetti* is not common, although markedly pathogenic when it occurs.

Both *Eimeria mitis* and *Eimeria praecox* are relatively non-pathogenic and common.

*Eimeria hagani* is slightly pathogenic and rare.

## COCCIDIOSIS OF POULTRY

### CAECAL COCCIDIOSIS

*Eimeria tenella* is the spp responsible primarily for this although gametogenous stages of *Eimeria necatrix* also occur in the caecum and at times some stages of *E. brunetti*

Coccidiosis due to *E. tenella* is seen majorly in chicken of 3-7 weeks of age. 1<sup>st</sup> stage schizonts in *E. tenella* develop deep in the glands. The 2<sup>nd</sup> stage schizonts are unusual in that the epithelial cell in which they develop leave the mucosa and migrate into the lamina propria and submucosa. When these schizonts mature and rupture about 72hrs after Oocysts ingestion, there is haemorrhage, the mucosal surface is largely detached and clinical signs are apparent. PPP = 7days and Ovoid oocyst sporulate in 2-3days under normal conditions in poultry houses. Clinical diseases occur when a large number of Oocysts are ingested over a short period and is characterized by the presence of soft faeces often containing blood. Chicks are dull and listless, with drooping feathers. In subclinical infections, there are poor weight gains and food conversion rates. At PM exam of chicken which had blood in their faeces, the caeca are found to be dilated and contain a mixture of clotted and unclotted blood. With longer standing infection the caecal contents become caseous and adhere to the mucosal. As mucosa regenerates the caecal plugs are detached and caseous material is shed in the faeces. Though good immunity to reinfection develops, note that recovered birds often continue to shed a few Oocysts i.e. act as carriers.

### COCCIDIOSIS OF SMALL INTESTINE

Several spp are important in this but *E. necatrix* is the most pathogenic. However, prevalence of disease due to *E. necatrix* and *E. tenella* has declined since many of the anticoccidial drugs in general use were developed specifically to control these 2 pathogenic spp thus enabling others to have assumed greater prevalence.

These include *E. brunetti* which is highly pathogenic but *E. acervulina*, *E. maxima* and *E. mitis* which are moderately pathogenic are commoner but *E. praecox* is only a minor pathogen. PP vary from 4-7 days following ingestion of large no of Oocysts.

Generally older chickens are affected by the spp found in S.I and the clinical signs are similar to those of caecal coccidiosis except that certain spp (*E. necatrix* and *E. brunetti*) cause sufficient damage for blood to appear in faeces.

Subclinical infections are more common than overt disease and may be suspected when pullets have poor growth rate and feed conversion with delay onset of egg laying.

### BASIC DESCRIPTION

	<i>E. tenella</i>	<i>E. necatrix</i>	<i>E. brunetti</i>	<i>E. acervulina</i>	<i>E. maxima</i>	<i>E. mitis</i>
Region	caeca	SI	lower SI	upper SI	mid SI	Lower SI
Intestinal lesion	Haemorrhage white spot	Haemorrhage, thickened wall white spot	Slight haemorrhage cogulative necrosis	Watery exudates White transverse band	Salmon pink exudates thickened walls haemorrhage with heavy infection	No visible lesion
Blood in faeces	++	+	+			
Degree of pathogenicity	++++	++++	+++	++	++	++
Oocyst size(µm)	23x19	20x17	25x19	18x14	30x20	16x15

50% sporulation time (hrs)	21	20	38	12	38	19
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## DIAGNOSIS

- Based on pm examination of affected birds.
- Presence of Oocyst in faecal sample diagnosis may be wrong:
  - (a) Major pathogenic effect usually occur before oocyst production
  - (b) Presence of large nos of oocyst is not necessarily correlated with severe Pathological changes in the gut depending on the spp involved.

NB: At recropsy the location and type of lesion present provide a good guide to the spp which can be confirmed by examination of the Oocyst present in faecal sample and the schizonts and / or oocyst in GIT scrapings.

## TREATMENT

To be introduced as early as possible after diagnosis has been made sulphonamide drugs are the most widely used, it is recommended to be gives at 2 periods of 3days in drinking H<sub>2</sub>O with an interval of 2days between treatment. Sulphaquinoxaline sometime potentiated with diavendine/sulphadinuline are dings of choice where resistance has been developed to sulphonamid a mixture of amprolium with ethopabate will give good results.

## Leucocytozoon

Parasite of domestic and wild birds.

Spp: L. simondi, L. smithi L. caulleryi

1Intermediate host: *Simulium spp.*

- Fusiform spindle shaped protozoa whose presence distort wbc shape. It can be associated with occasional leukocytosis and Diseases.

Causes avaian malaria along with plasmodium and haemoproteus

## LIFE CYCLE

schizogony occurs in hepatocytes and vascular-endothelia cells of various tissue producing merozoites which invade erythroblasts, evythrocytes, lymphocytes and macrophages and there develop to gametocytes. Leucocytozoon gametocytes differ from plasmodium and haemoproteus in not containing pigmented granules and in greatly distorting the host cell.

Some gametocytes are round and push the host cell nucleus to one side so that it forms a cap on the parasite.

### **Ehrlichia** (Genus Rickettsia)

Found in the blood leucocytes as intracytoplasmic inclusions.

Spp: *E. phagocytophila*. (Tick-borne fever in sheep and cattle)

*E. canis* (tropic pancytopenia in Dogs.)

*E. (cytoecetes) phagocytophila*: transmitted by *Ixodes ricinus* incubation period: 7 days.

Clinical signs

A short febrile illness. (fever), leucopenia, dullness, inappetence, death may be seen in young lambs (due to lack of contact with dam).

Rx: rarely necessary, by prophylaxis depends on tick control by dipping

*E. canis* transmitted by *Rhipicephalus* (cause  $K_9$  pancytopenia) and it is found in macrophages

Clinical signs

Leucopenia and thrombocytopenia. Death may occur due to 2<sup>o</sup> infection associated with the leucopenia or due to mucosal and serosal haemorrhages due to platelet deficiencies.

Rx: Tetracyclines

Bovine petechial fever (Ondiri disease) caused by *Cytoecetes ondiri*

Equine ehrlichiosis caused by *E. equi*

Potomac horse fever caused by *E. risticii*

### **Aegyptianella**

:*A. pullorum*: Anaplasma-like parasite affecting chicken, geese and ducks seen in cytoplasm of red cells as anaplasma-like bodies.

Clinical signs

anemia, icterus and diarrhea. Transmitted by soft tick *Argas persicus*.

Rx: Tetracycline compounds

### **Haemobartonella**

:seen as cocci/short rods on erythrocyte surface often completely surrounding the margin of red cell.

H. are tightly attached to the red cell and are rarely free in plasma .

H. felis is the most significant as a cause of haemolytic anaemia in young cats probably depends . transmission is dependent on arthropods/ linstion of during fighting.

Rx Tetracylines

## **Anaplasma**

Small, spherical bodies, red to dark red in color under microscope when stained with Romanowsky stain and intraerythrocytic parasite.

Host: cattle, deer, sheep and goat. 0.2-0.5µm in diameter,with no cytoplasm and occasionally multiple invasion of a cell many occur

Spp

A. Marginale, A. centrale, A. ovis

### Anaplasma marginale

Hosts: cattle ,zebra, deer Sheep and goat may develop inapparent infections . It is transmitted by ticks of various spp and mechanical transmission by blood sucking flies is important in some areas. Mechanical transmission can occur through:

Major and minor operations in cattle: Dehorning, Castration, Vaccination, Blood sample collection etc. PP= 26 days.

### Clinical signs

Anaplasmosis is a disease of the adult animal (Not seen until Cattle are round 18months.) Splenectomised young animals will come down. Fever, infection may be fatal during fever period. Anorexia , severe anaemia, high mortality.

Rx: Tetracycline 6-10mg/kg , imidocarb.