ADENOVIRUS INFECTIONS

by

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ADENOVIRUS INFECTIONS

AVI are group of infections coursed by a members of avian adenovirus group in many species of poultry ch. By respiratory , enteric , hepatitis , splenomegally , hedropericarditis , anemia , mortality , drop in egg production and immunosuppresion.

Member of this family coursed disease condition

Or charring others in inducing syndrome or act as helper virus for tumor viruses.

Familiy Adenoviridae

- Genus Aviadenovirus: **Group I Avian Adenoviruses**
  - **Fowl Adenovirus** - 12 Serotypes / 5 Species
    - Inclusion Body Hepatitis (IBH)
    - Hydropericardium Syndrome (Angara Disease)
    - Gizzard Erosion and Proventiculitis
- Genus Siadenovirus: **Group II Avian Adenoviruses**
  - Hemorraghic Enterititis (turkeys), Marble Spleen Disease (pheasants), Splenomegaly (chickens)
- Genus Atadenovirus: **Group III Avian Adenoviruses**
  - Egg Drop Syndrome (EDS)

Virus

Adenoviruses are double stranded DNA , enveloped , grow in cell nucleus with intranuclar inclusion. AVs not agglutinate generally chicken rbcs ; Except EDS and CELO who agglutinate rat and human O type .

VIRUS GROUPS:

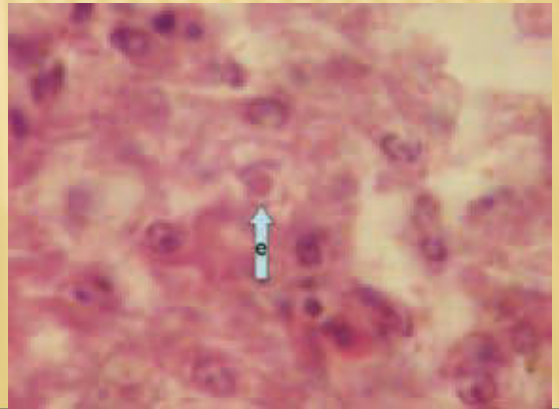
1.Group 1: Include Quail bronchitis (CELO/Phelps) and IBH.

2.Group 2: „ THE , MSD and AV. splenomegaly.

3. „ 3 : „ EDS and similar viruses from ducks.

Lab.Host:

E.C.E.: AVs grow on all sac → Serial passage → curling & Dwarfing , death ,stunted hepatitis , splenomagaly , congtion and hemorrhages in all parts . Hepatocytes have Basophilic or eosinophilic inclusions .



TC : It is useful to use homologous cell to examine host sample.

Transmission and epidemiology:

- AVs transmitted through both vertical and horizontal routes.
- Infection can remain latent for at least one generation in SPF.
- Virus present in feces ,tracheal ,nasal and kidney mucosa i.e. all secretions .
- Newly hatched chicks induce higher virus secretion.
- Broiler: 1st wave of excretion between 4-6 ws. Layer: 1st 5-6 ws till 15 ws & 2nd wave of virus excretion around peak of egg production due to stress of production and SEX hormones.

Infection with group -1

a) Quail bronchitis:

Acute highly contagious disease of young quail with rabid onset ,high mortality and morbidity.

Infection:

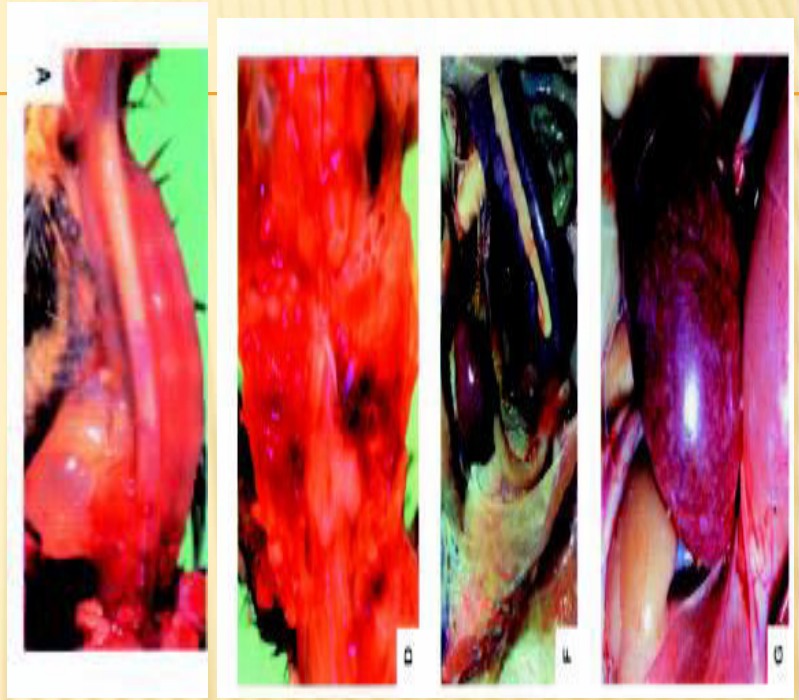
Through contact aerosol and fecal oral route.

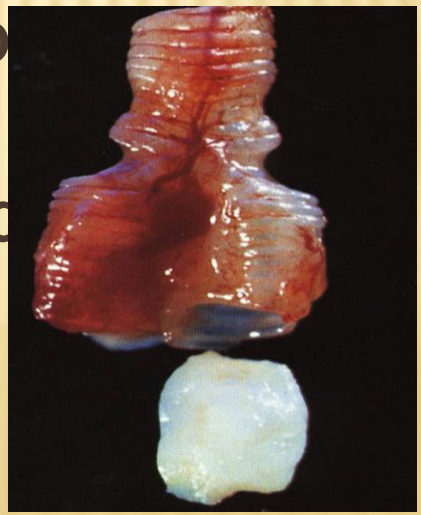
Signs:

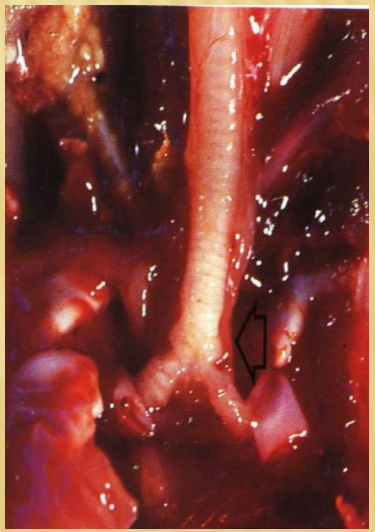
Occur within 2 to 3-7 dpi in birds less than 3 ws show respiratory signs including :open mouth breathing , nasal and ocular discharge ,sudden increased mortality and morbidity up to 50 % . General signs of huddling under brooder , riffled feather and off food.

LESIONS:

- Nasal and ocular discharge.
- TracheaL opacity and filling of with moist necrotic exudates , thick tracheal mucosa .
- Necrotic exudates in anterior air sacs.
- Lung: red consolidated area around bronchi.
- Liver : mottled pale pin -point to mm necrotic foci .
- Spleen mottled slightly enlarged







Hydropericardium Syndrome

- In broiler 3-7 weeks, there is an accumulation of clear straw-colored fluid in the pericardial sac, pulmonary edema, swollen and discolored liver, and enlarged kidneys with distended tubules showing degenerative changes. Mortality can reach 70%.
- The disease can be seen in layer and breeders.
- Multiple areas of focal necrosis exist with mononuclear infiltration in the heart and liver.
- Basophilic inclusions are present in the hepatocytes

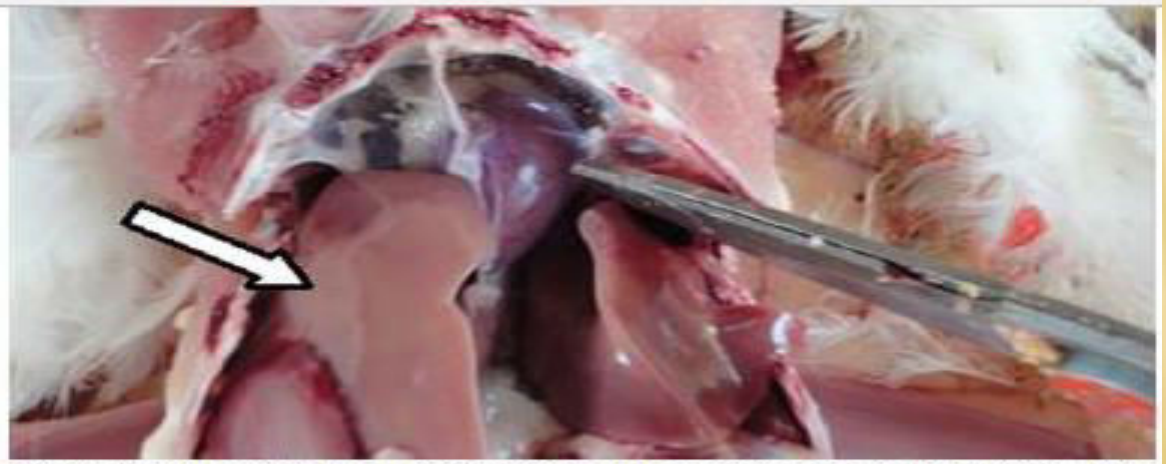
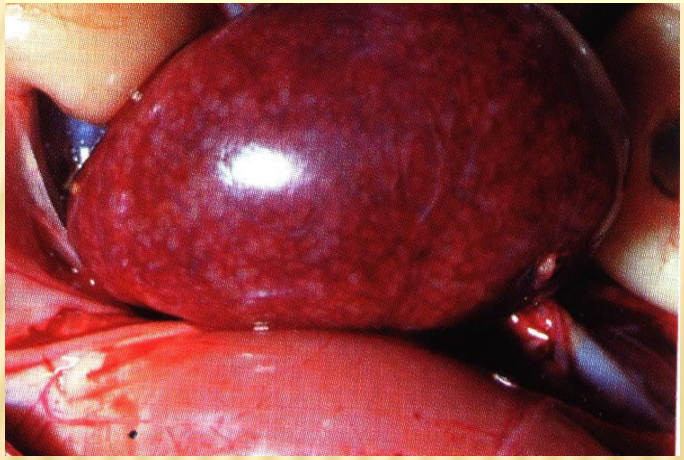


Fig.1. Broiler chicken with hydropericardium and hepatitis. The liver is pale, enlarged and discolored.

Avian Adeno Spleenomegaly

Enlarged and mottled Spleen.



TURKEY HEMORHAGIC ENTERITIS

Acute infection of young turkey ch. by: sudden onset , short course , depression , bloody dropping ,immunosuppresion and high mortality.

VIRUS:

THE & MSD virus replicates in nucleus of reticuloendothelial cells. Virus grows in turkey embryos.

HOSTS;

Young turkey aged 6-12 ws , birds under 6 ws are refractory. Pheasants and chickens are also infected.

SIGNS:

Rabid progress in signs within 24 hs , depression ,bloody dropping and death. Feces with frank blood on skin and feathers of vent. Bloody feces can be forced from vent. Well fleshy birds with diarrhea die. Mortality up to 60% in average 10 days.

LESION

Dead birds are well flesh with pale skin and massive hemorrhages. Hemorrhages on heart ,muscles ,liver and proventriculus. Intestine filed with blood. Duodenum covered with yellow fibrinous exudates

Spleen enlarged mottled .Lung congested. “Spleen of dead birds is small”.

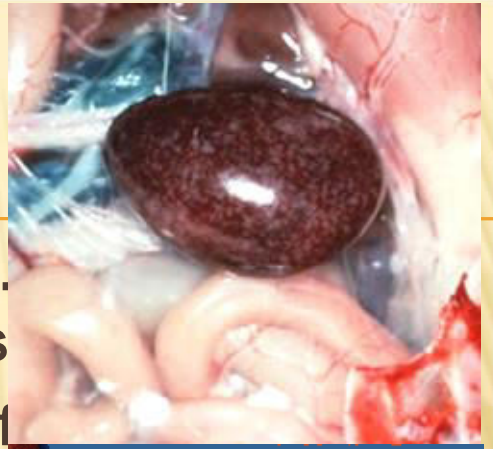
IN IBs can be seen in liver, spleen, bone marrow, pancreas, lug intestine and blood lymphocytes.

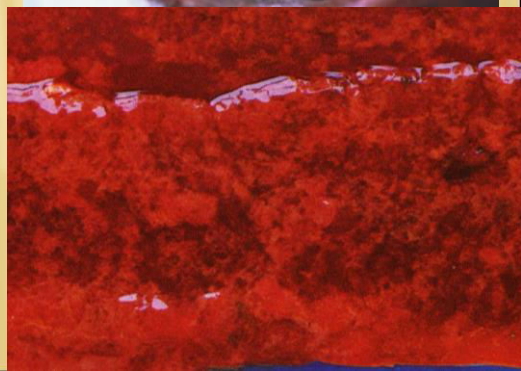
TREATMENT:

.At first signs injection of 0.5 ml immune serum.
.Vitamin K to stop hemorrhages.

.Antibiotic for 2nd.envedors.

.Electrolytes to prevent dehydration.





INCLUSION BODY HEPATITIS

Chicken of young age show sudden onset, sharp increase in mortality, short course, anemia, hepatitis with IN IBs and immunosuppresion.

virus

19 virus serotypes are isolated from chicken, turkey duck and geese.

Virus induce severe signs in presence of IBD an CA.

SIGNS:

Sudden mortality in 3-8 ws old meat type chickens, peaking 3-4 days to reach 10 – 30%. Affected bird with signs died within few hours or recovered. Pale comp and wattles and facial skin, general signs.

LESIONS:

SKIN: Pale, enteric.

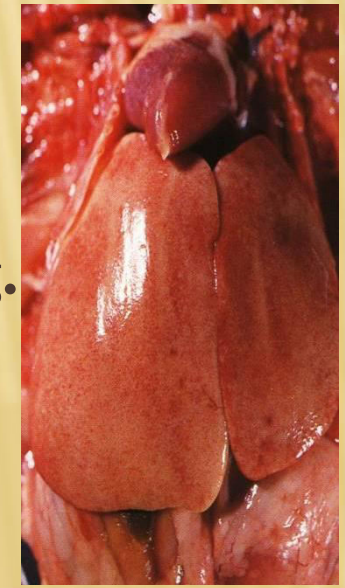
Hemorrhage in muscles and serous mm ..

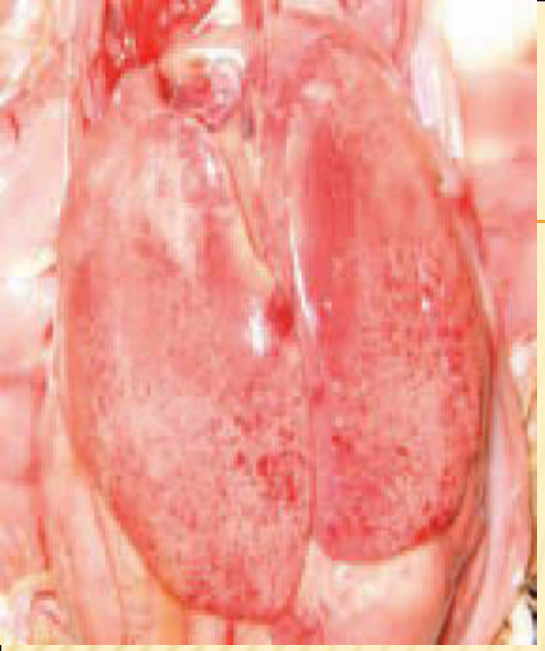
Liver: swollen ,pale, friable with hemorrhage→ mottling.

Kidneys: ,, , ,, with cortical hemorrhage..

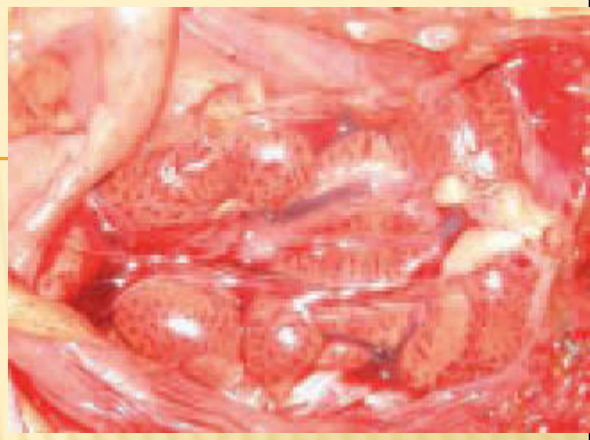
Bone marrow: Pale. Blood; watery thin.

SPLEEN & BURSA-→ small.

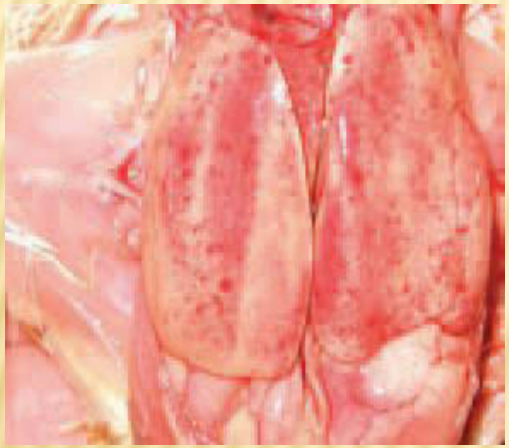




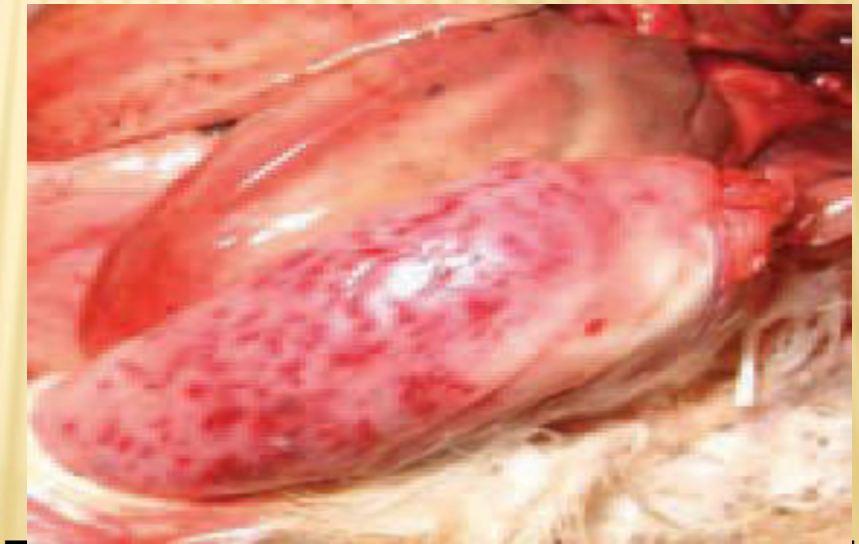
pericardial fluid is increased (hydropericardium).



kidneys are enlarged, pale and mottled with multiple hemorrhages.



massive mottled or striated hemorrhages with necrotic foci of the liver.



Ecchymoses and striated hemorrhages in skeletal muscles **(legs and breast.)** .

EGG DROP SYNDROME

Infection of laying chicken ch. by :mild respiratory signs and drop in egg production with change in shell quality at beak of production.

VIRUS:

AV-3 agglutinate chicken ,duck and turkey ,but not rabbit and hours rbcs. HA character is heat resistant.

The virus is of one serotype and 2 isolates: chicken (EDS76) and duck (BC14) isolates.

LAB.HOST:

Virus grow firstly on all sac of duck and geese embryo, then on chicken embryo.

TC. From duck kidney and duck embryo liver-→IN IB.

NATURAL HOST:

* Disease occur in laying hens, ducks and geese at all ages.
* Naturally broiler breeder and heavy breads producing brown eggs are more severely affected.

PATHOGENESIS

1.Infection of adult with limited inflammation in nasal mucosa→ mild respiratory signs and viremia in 3-4 days.

2. Virus reach and replicates in lymphoid tissues 7-20 days.

3.Virus reach shell gland pouch inducing inflammation.

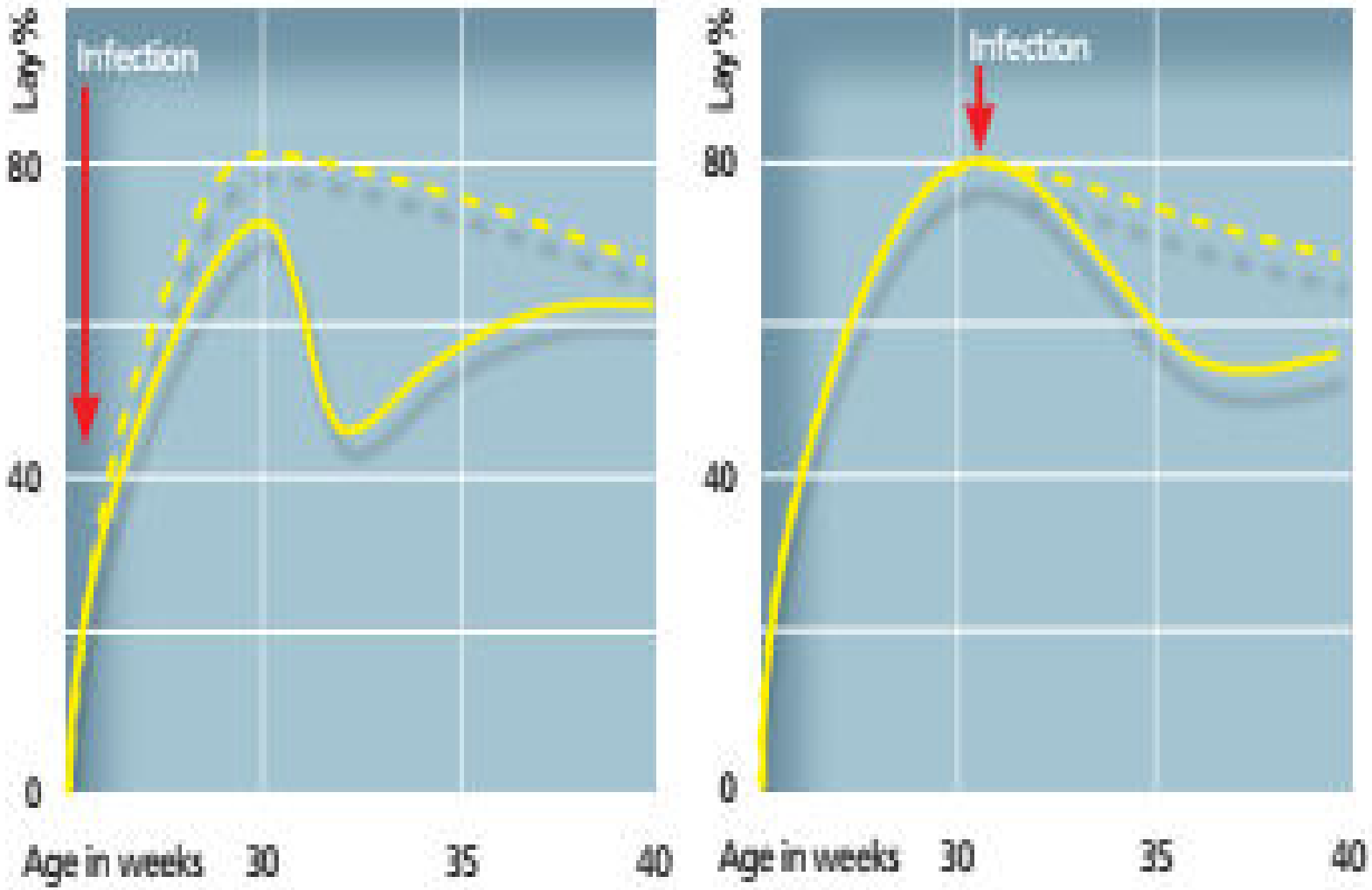
4. Abnormal shell appear.

NB. EDSV NOT REPLICATE IN INTESTINAL MUCOSA AND ITS PRESENCES IN FECES IS DUE TO CONTAMINATION FROM OVIDUCT SECRATION.

TRANSMISSION:

EDS transmitted via both *vertical and horizontal* routs, outbreaks can be classified according mode of transmission into:

1. *Classical* :breeder flock is infected and gives vertical infection to baby chicks those → shed virus and form antibodies at 50% egg production
2. *Edemic* : Virus execrate in (1) to contaminate farm.
3. *Drinking water*: Wild and domestic ducks contaminate water that can reach chickens.

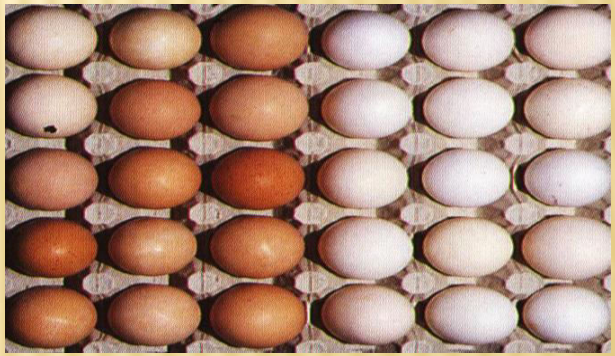


Non-peaking effect of Egg drop by early EDS infection and drop by infection during lay

SIGNS:

Mild transient respiratory signs, loss of egg color in pigmented eggs followed by soft shell ,then ,spotted or shell less egg. Rough one end of egg "SAND PAPER LIKE". No change in fertility, hatchability or in outer egg character. Drop in production up to 40% for 4-10 weeks "HEN LOST 10-16 EGGS".

NB:DROP DUE TO ACTIVATION OF LETANT INFECTION USUALLY OCCUR WHEN FLOCK REACH 50% PRODUCTION.



LESIONS:

Active ovary, atrophied oviduct and uterine edema. Exudates in shell gland pouch. Mild splenomegally. Ova of different size in abdominal cavity. IN-IB. can be detected in shell gland cells.

DIAGNOSIS

.Change in shell without mortalities…

Samples from BC at virimia or shell gland ,oviduct secretion egg albumin..

Isolation in duck embryo or Tc .. Identification of HA Virus identification with HI test..

Detection of antibodies is serum by ELISDA , HI, AGP,

DIFFERENTIAL DIAGNOSIS

EDS must differentiated from; ND , IB , SHS , CELO non infectious courses of shell changes.

PREVENTION:

1. Hygienic measures.
2. prevent contact ducks.
2. Avoid use of contaminated water.
4. Egg sanitation.
5. Testing of breeder flocks.

VACCINATION:

There is an inactivated vaccine given by injection at 14-16 weeks of age (4-6 ws before egg) .