# Chicken Infectious Anaemia

**Def:**

Infectious disease affecting either **meat and egg type chickens**

**CIA is characterized by :**

* **Aplastic anaemia .**
* **Generalized lymphoid atrophy .**
* **Retardation of growth .**
* **Immunosuppression .**
* **Increased mortality (10 – 60 %) .**
* **Impaired immune response to vaccines**
* **First described in 1979 in Japan .**
* **Worldwide in distribution.**

*Frequently complicated by secondary infection.

* CIA and closely associated syndromes have commonly been termed :

* Haemorrhagic syndrome .
* Anaemia-dermatitis .
* Blue wing disease .

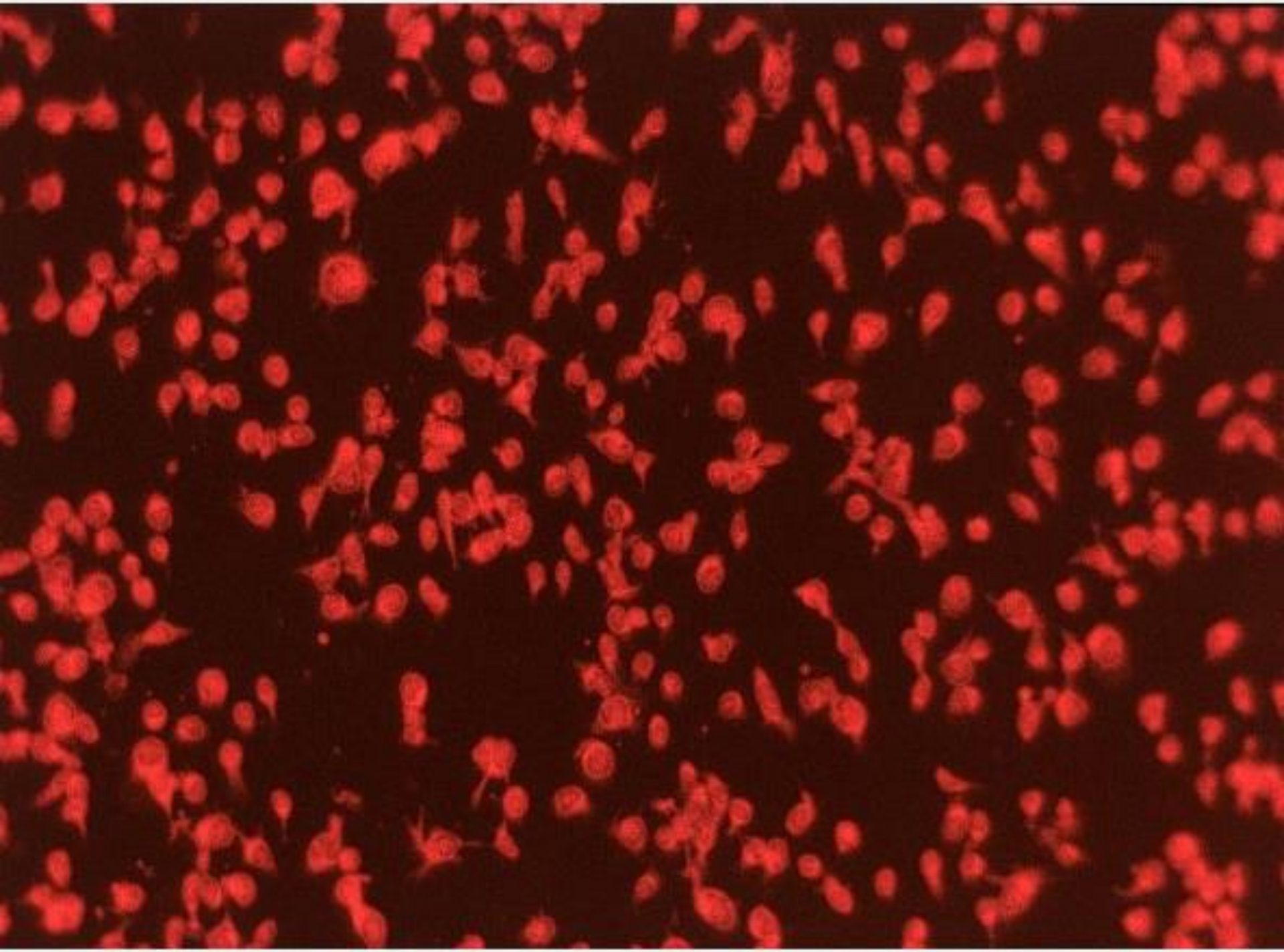
# AETIOLOGY

* Small non-enveloped virus ( 25 nm ) .
* Family Circoviridae .
- The virus replicates in haematopoietic precurser cells (bone marrow) and in thymic precurser cells (thymus cortex).
* All strains belong to one serotype.
- * The virus is very resistant to a wide range of physical and chemical factors

# CAV

- ***Resistance to chemical and physical agents***

- resistant to ethylether and chloroform
- stable at pH3
- resistant to 70$^{o}$C for 1 hour
- resistant to 80$^{o}$C for 15 minutes
- inactivated by 50% phenol (5 minutes)
- resistant to lipid solvents
- resistant to 5% quat. ammonium compound



# HOST

* **Chickens** is the only known host .

* All ages are susceptible to Infection.

* Maternal immunity protect chicks during the first 1-3 weeks .

* In immunosuppressed chicks (IBDV) maternal immunity to CIAV could be overcome .

* Outbreaks in chicks correlate with absence of maternal immunity .

# TRANSMISSION

## Vertical :

Through hatching eggs ( for 3-6 weeks ) .
Cause CLINICAL DISEASE in progoney during the 2nd – 4th week of age .
N.B.cocks may transmit infection .

## Horizontal :

-Mainly via oral route ( by 2-3 weeks of age ).
-Respiratory route is possible .
-Mostly cause SUBCLINICAL INFECTION in progony

# INCUBATION PERIOD :

**In congenitally infected chicks :**

**10 -12 days**

**In horizontally infected chicks :**

**30 – 34 days**

# PATHOGENESIS

Infection of breeders with CIAV **(Clinically inapparent )**

↓

Vertical transimission to their progoney

( damage to **haemato-and lymhopoietic tissue**)

(depression of macrophage functions , interleukin and interferon production )

↓

Anaemia – mortality – low performance – immunosuppression and susceptibility to Secondary infection

# CLINICAL SIGNS

Apparent form :

( follow vertical trasmission )

* **Deppression .**
* **Paleness ( sometime)**
* **Anaemia ( haematocrit less than 27 )**
* **Increased mortality ( up to 60 %)**
* **Reduced weight gain .**
* **Survivors recover 20 -28 days P.I.**

## Subclinical form :

**(follow horizontal infection)**

* **May go unobserved .**
* **May cause :**
  - **Slight increased mortality ,**
  - **Transient poor performance .**
    **(Av.B.W, Feed Conv.Ratio )**

* **Detection of antibodies in clinically normal chicks .**

# FACTORS INFLUENCING MORBIDITY AND MORTALITY

* *Virulence* of CIAV .
* *Dosage* .
* *Route* of infection .
* *Environmental* factors .
* *Age* of the birds .
* *Concurrent* infection .
* Other *immunosuppressive* factors
* *Genetic* constitution .

# IMMUNOSUPPRESSION

**-CIAV is highly immunosuppressive in susceptible young result from :**

1- **Damage** to haematopoietic and lymphopoietic tlymphoid depletion .

*** 2-Transient decrease in :**

Macrophage functions and Cytokine production

# Immunosuppression

**CIAV infect.up to 14 days of age depresses :**

* **Immune response to Marek's disease and inactivated ND vaccines**

**Imunosuppression in anaemic bird is indicated by :**

* **Increased suscept.to other infections.**
* **Enhanced pathogenicity of :**
  - **Adenovirus**
  - **Reovirus**
  - **Live attenuated NDV .**

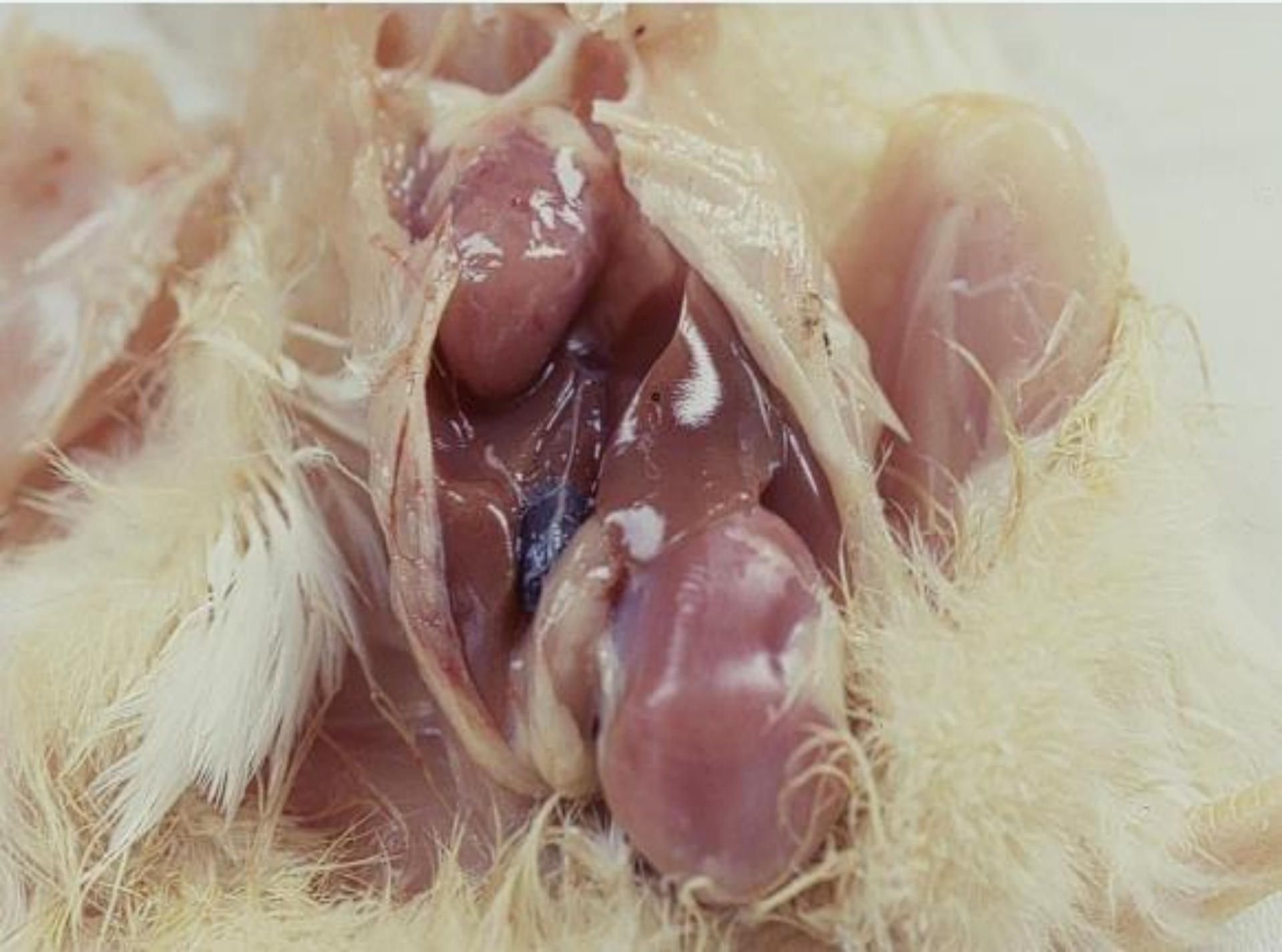
# Enhancement factors

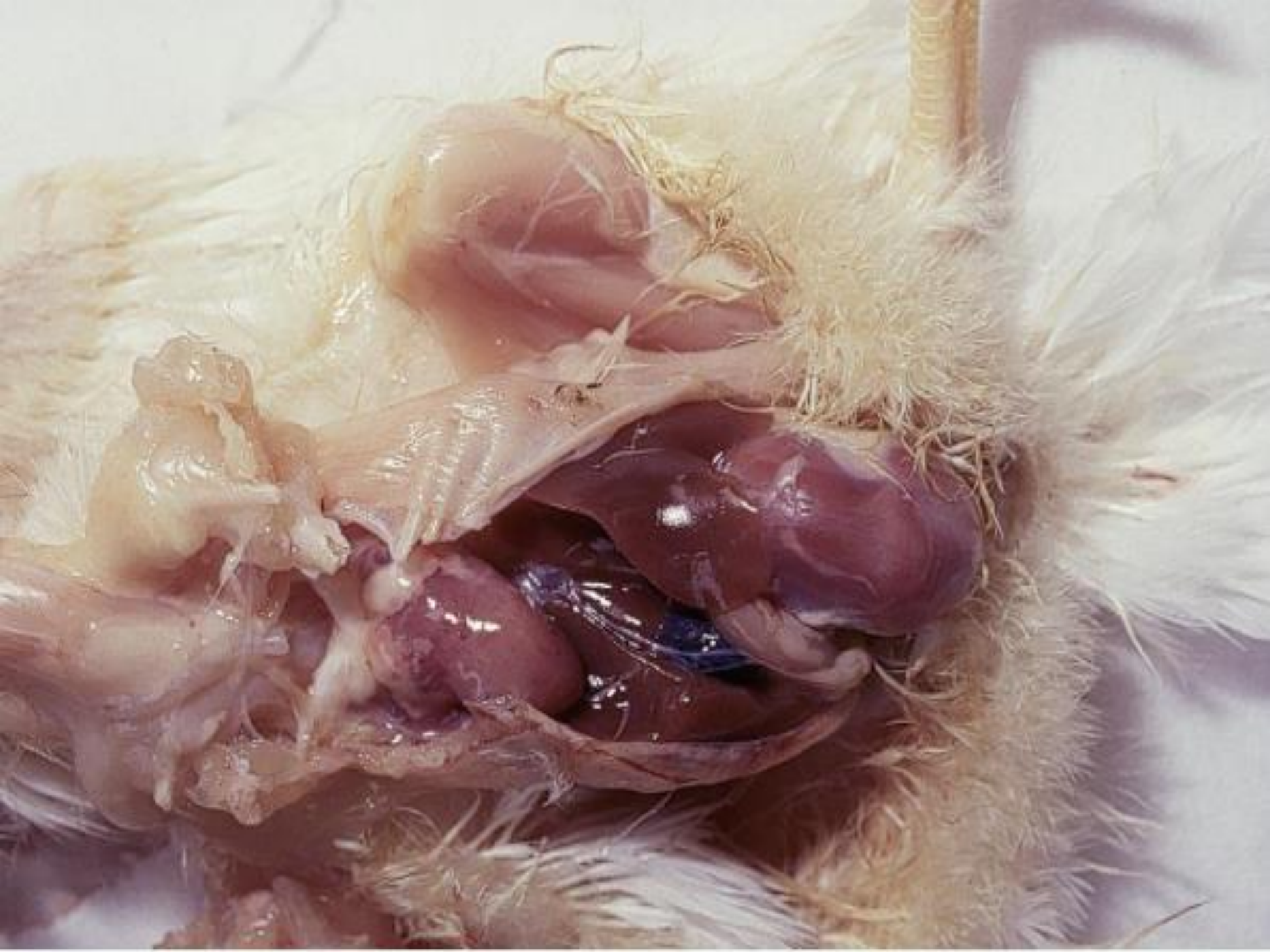
- The infection due to CAV can be enhanced by other immunosuppressive factors e.g. IBDV, MDV, REO

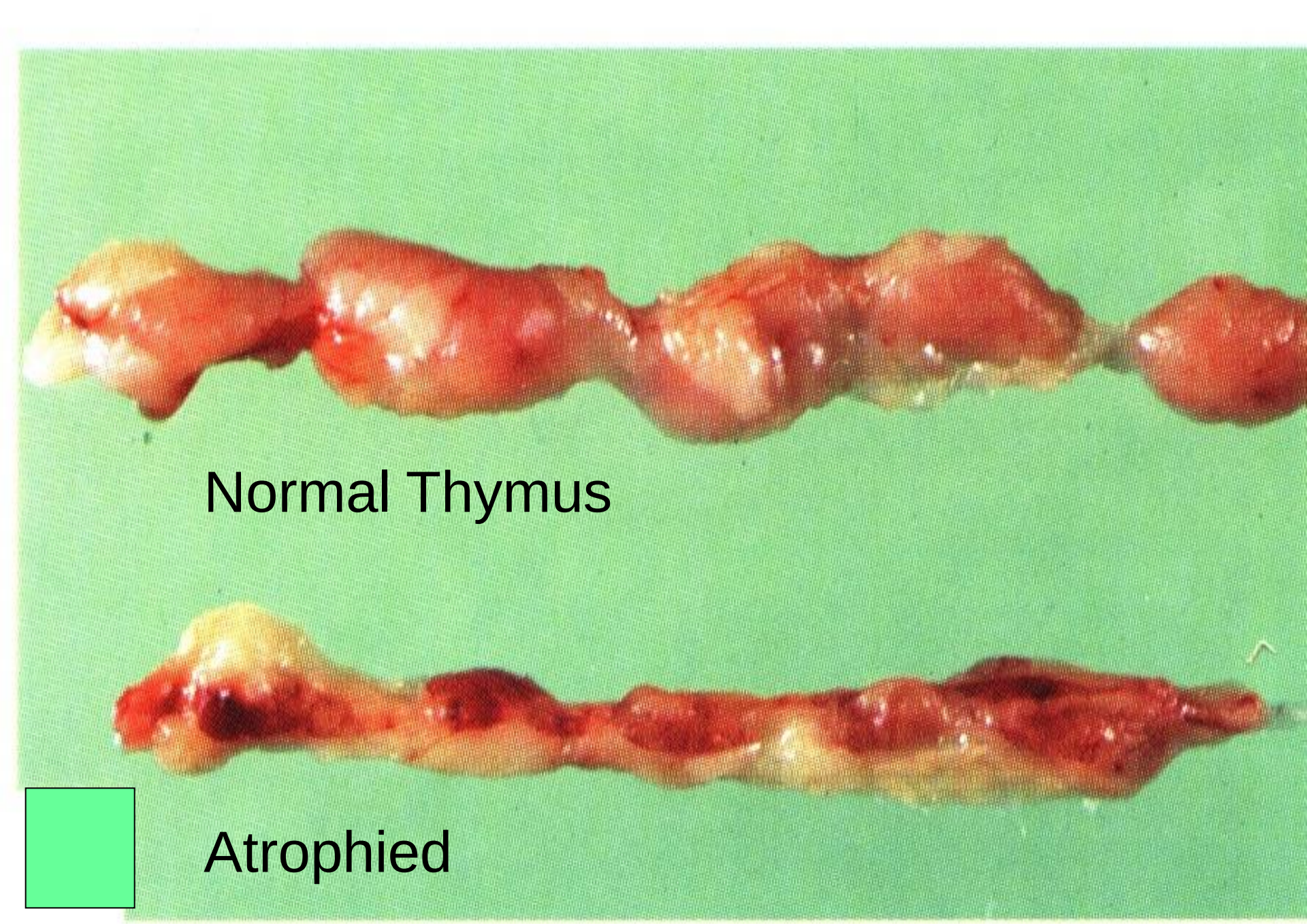
- ⟶ Age resistance to Infectious Anemia is delayed by e.g. simultaneous infection with IBDV

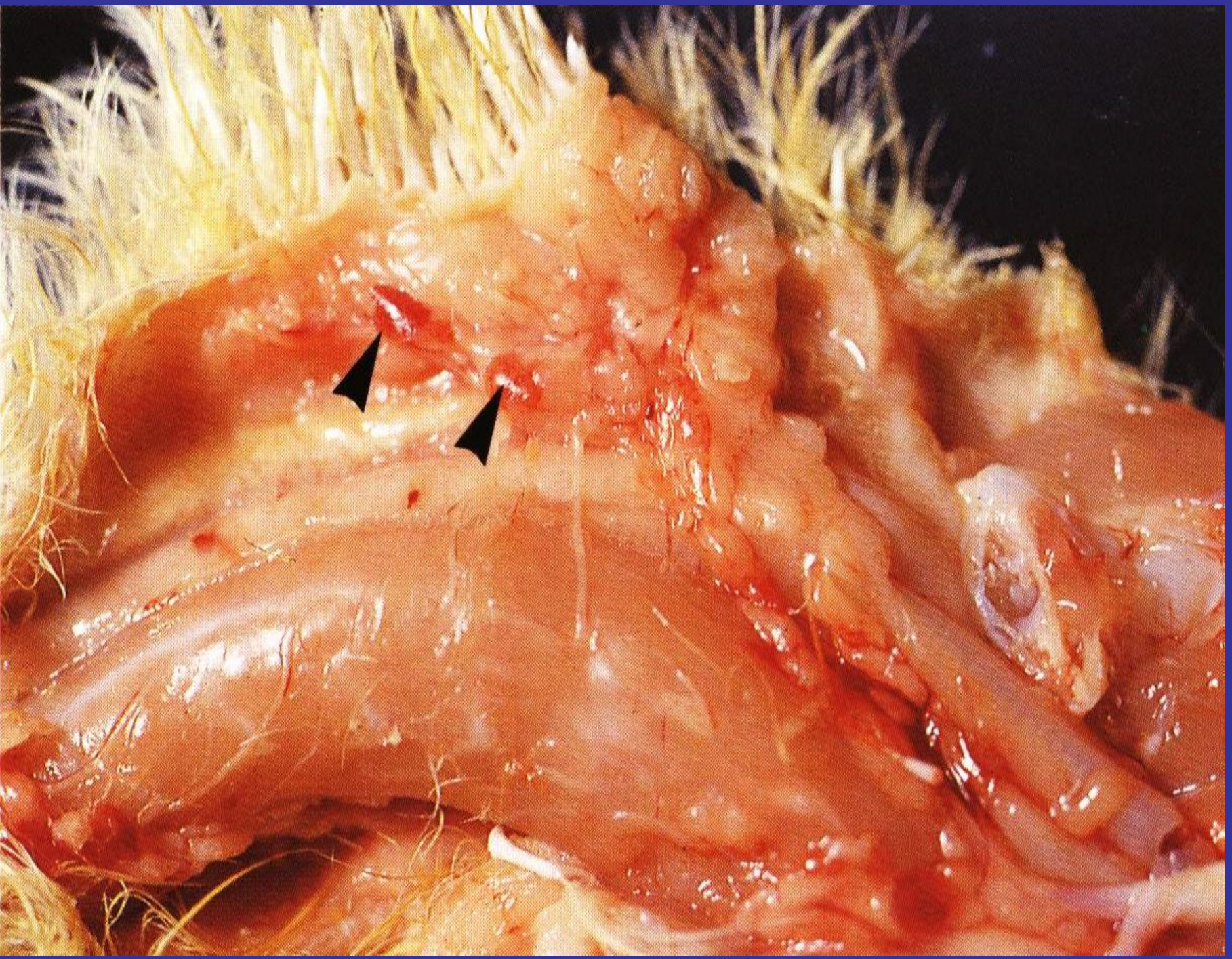
# GROSS LESIONS

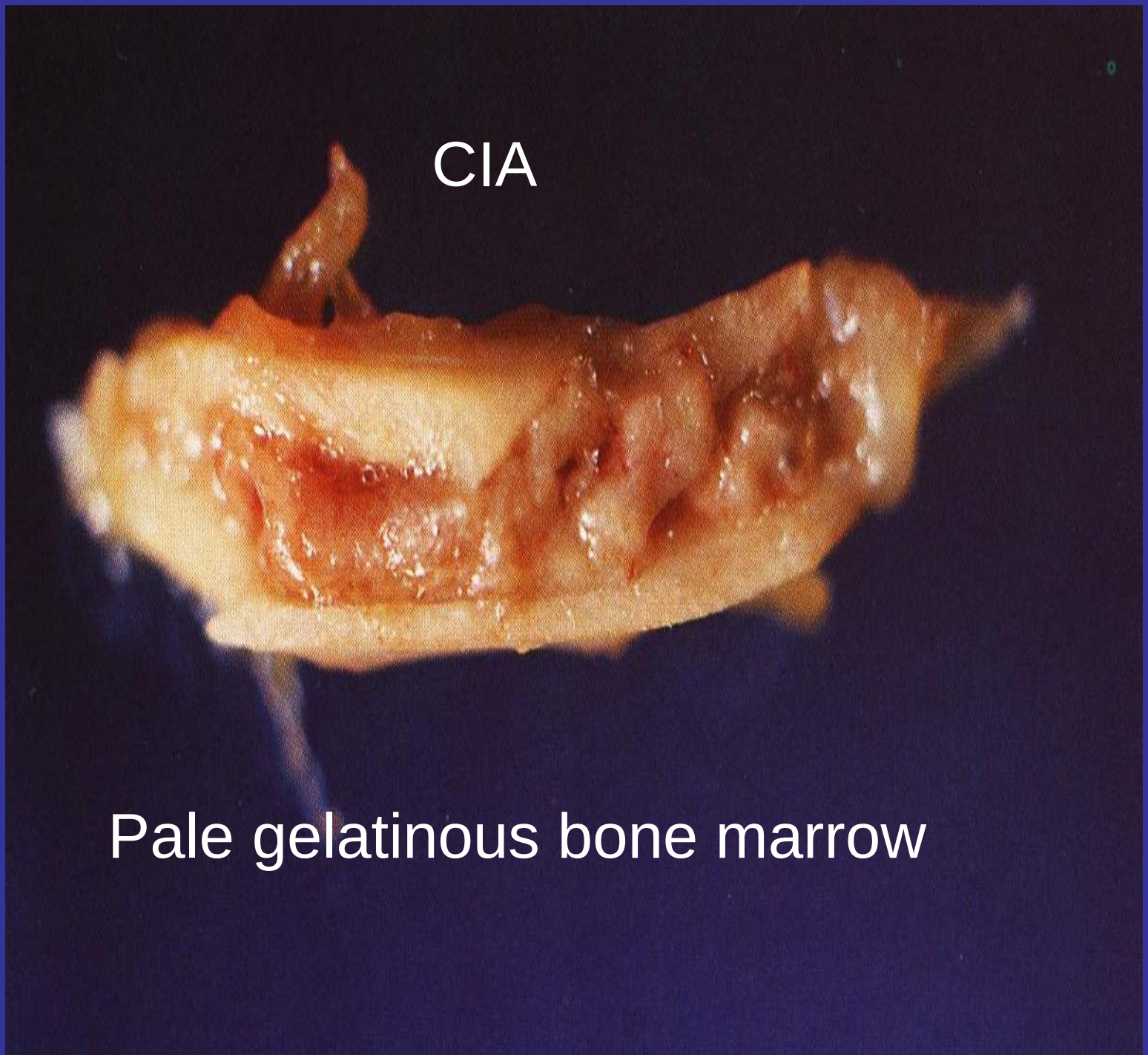
* Atrophy of :
    * Thymus (consistent ) .
    * Bone marrow ( charactristic)
    * Bursa of Fabricius (less obvious).
* Haemorrhages in:
    * Proventricular mucosa .
    * Subcutaneous and intramuscular tissu.
*Swollen and mottled liver .






Normal Thymus
Atrophied




CIA
Pale gelatinous bone marrow

# HISTOPATHOLOGY

* **Severe lymphoid depletion in :**
    * **Thymus .**
    * **Bursa of Fabricius .**
    * **Spleen .**
    * **Cecal tonsils and other tissues .**
* **Bone marrow :**
    * **Atrophy**
    * **Aplasia**

    **Haematopoietic cells are replaced by adipose tissues or proliferating stroma cells .**

# HAEMATOLOGY

**Blood : watery .**

**Clotting time : increased .**

**Blood plasma : pale .**

**Haematocrit values : less than 27 % .**

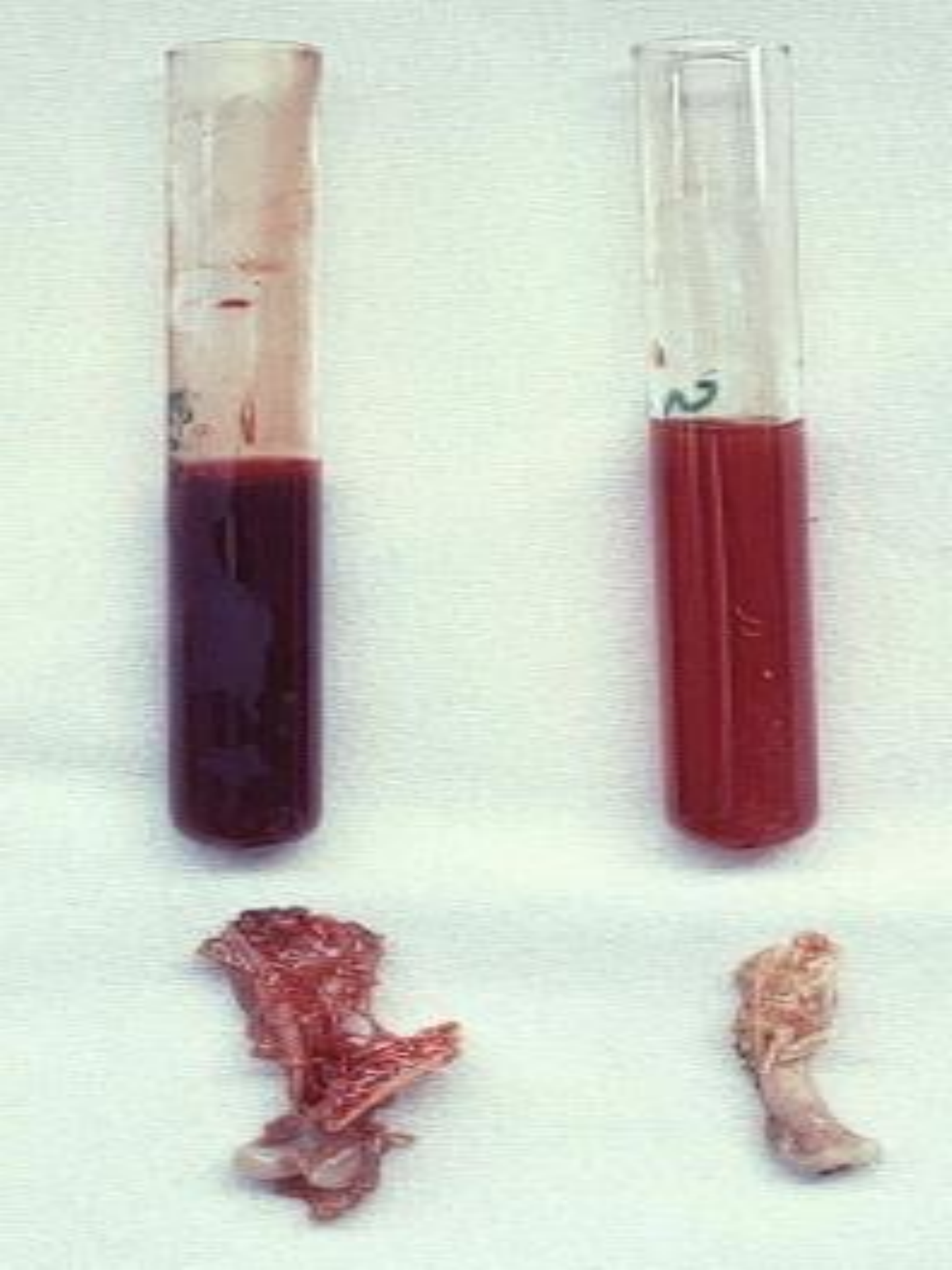
**( versus 29-35 % normal )**

*** Low haematocrit value is due to decreased number of :**

**** Erythrocytes .***

**** White blood cells .***

**** Thrombocytes .***



# DIAGNOSIS

1- Flock history .
2- Clinical signs .
3-Haematological changes .
4- Gross and micoscopic lesions .
5- Isolation and identification of the virurs :

* Samples:

- whole blood and buffy coat.
- rectal contents .
- liver homogenate (preferred)

* Methods :

a- Isolation in day old chicks (most specific)
b- Isolation in cell culture (MSB1 cells )

# DIAGNOSIS

6- Detection of **viral antigens** in tissues (thymus is prefered ) using FAT and IP .

7-Detection of **viral nucleic acid** using DNA probes and **PCR .**

8- Detection of specific **CIAV antibodies** :

- **Neutralization** test .
- Indirect FAT .
- ELISA and indirect IP .

# DIFFERENTIAL DIAGNOSIS

1-Sulfonamide intoxication & **mycotoxicosis** can result in **aplastic anaemia** and haemorrhagic syndrome .

2- **MDV and IBDV** : induce atrophy of lymphoid tissues .

3- Adenovirus : a major cause of previously designated Inclusion Body Hepatitis .

4- **Osteopetrosis** and **Erythroblastosis** may induce anaemia .

# PREVENTION AND CONTROL

1- Improve management and hygiene to :

* prevent immunosuppression .
* prevent or minimize early and heavy exposure to CIAV .

2- Immunization of parent flocks several weeks before egg production .

# PREVENTION AND CONTROL

* **Two types of live vaccines** are available :

a- Virulent CIAV given in drinking water at 13-15 weeks of age .

b- Attenuated CIAV (i/m or s/c or w/w)

* Monitoring of breeder flocks for the presence of CIAV antibodies 3-4 weeks before onset of production to decide the necessity of or efficacy of vaccination

# CAV - vaccine Nobilis

## *Opportunities*

- Reducing the responsibility of the hatchery for CAV breaks
- Preventing early CAV infection in the progeny
- → Better production figures in the progeny
- High maternal immune chickens
- less problems with - Mareks
- - Reo
- - Gumboro
- Field infection breeders → still vertical transmission possible
- More biosecurity (IQC) → more flocks with
- infections during lay

# Nobilis® CAV P4

## *Conclusions*

- The vaccine is safe
- High and uniform levels of antibodies are obtained after i..m., s.c. and w.w. administration
- Titers remain high until the end of the production period
- No interference with live vaccines against Reovirus, (AE +) Pox and inactivated vaccines
- The progeny of vaccinated breeders is protected

# CAV - vaccine Nobilis

***Strong points***

- First attenuated CAV vaccine in the world
- Safe
- Uniform high immunity
- Quick seroconversion
- Combination possible with:
  - reo and pox live vaccines
  - inactivated viral vaccines

# CAV - Drop of MDA