Chicken Anemia Virus
Disease Overview
Introduction
Economic importance
Etiology
Transmission
Clinical signs
Post mortem lesions
Diagnosis
Prevention and control
Introduction

Chicken anemia virus is commonly found in commercially produced chickens and has a worldwide distribution.
The chicken anemia virus (CAV) was first described in 1979 by Yuasa et al. (1979) in commercially produced chickens.

Since that time, the virus has been detected by isolation or serology in most other countries in both laying and broiler chickens (von Bulow and Schat, 1997).
Chickens are the natural hosts of CAV.

All ages of chickens can be infected, the most severe clinical signs are seen in chickens younger than 2 weeks of age.

CAV has also been isolated from other species such as turkeys and CAV antibodies have been detected in quails.
The importance of this virus comes from its:

1. Trans-ovarian transmission.
2. Potential for inducing immunosuppression alone or in combination with other infectious agents.
Plan of Talk

- Introduction
- Economic importance
- Etiology
- Transmission
- Clinical signs
- Post mortem lesions
- Diagnosis
- Prevention and control
Economic Importance

- The **immunosuppressive effects** of CAV on broilers are more economically significant than the disease itself.
The economic impact of CAV is mainly due to:

1. Bad performance (poor FCR and reduced weight gain).
   - It has been proven that flocks that appear normal, but suffered from a subclinical CAV infection, performed less well when compared to flocks that remained negative throughout the growing period.
2. Increased condemnation rates at slaughter.
4. Vertical transmission from breeders to their progeny which may result in clinical disease.
The effect of a subclinical CAV infection on the performance parameters of broilers was well documented by McNulty et al.

<table>
<thead>
<tr>
<th>CAV status</th>
<th>% net income</th>
<th>Bonus %</th>
<th>FCR</th>
<th>Weight %</th>
<th>Mortality %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Negative</td>
<td>+2.4</td>
<td>+2.7</td>
<td>-0.4</td>
<td>+0.8</td>
<td>-0.16</td>
</tr>
<tr>
<td>Positive</td>
<td>-10.6</td>
<td>-8.8</td>
<td>+1.6</td>
<td>-1.7</td>
<td>+0.24</td>
</tr>
</tbody>
</table>
Plan of Talk

- Introduction
- Economic importance
- Etiology
- Transmission
- Clinical signs
- Post mortem lesions
- Diagnosis
- Prevention and control
Etiology - Virus Characteristics

› Small, spherical, single stranded, non enveloped DNA-virus.
› Family: Circoviridae.
› Genus: Gyrovirus.
Cont. ...

- Able to withstand pH of 3 and chloroform.
- Not inactivated by heating at 70°C for one hour or after 5 minutes at 80°C.
- Resistant to lipid solvents and to treatment for 2 hours at 37°C with 5% solutions of commercial disinfectants (quaternary ammonium compounds, amphoteric soap and orthodichlorobenzene)
Etiology - Virus Serotypes

› All Chicken Anemia Virus isolates belong to one serotype, although antigenic differences among isolates have been reported in the United States.
Pathogenesis

- CAV can be found latent in SPF flocks where the virus was detected in the ovaries, oviduct, testicles and spleen of birds without obvious seroconversion until the birds came into production.
Plan of Talk

› Introduction
› Economic importance
› Etiology
› Transmission
› Clinical signs
› Post mortem lesions
› Diagnosis
› Prevention and control
Transmission

› Disease usually occurs during the first 3 week of life through;
  1. Vertical transmission when breeder flocks get infected during the production period; this occurs especially in younger flocks.
  2. Horizontal transmission through infected organic material or contaminated equipment.

› Chickens at any age are susceptible to infection by:
  – Oral route.
  – Respiratory route.
Cont. ...

- In younger birds the presence of clinical signs depends on:
  1. The challenge dose.
  2. The level of the maternally derived antibodies.
  3. The presence of other immunosuppressive agents (such as marek's disease virus, reovirus or infectious bursal disease virus).

  - Immunosuppressive agents act synergistically increasing the severity of the disease and overriding the protective effect age and maternal antibodies.
Cont. ...

› **Incubation period**
  - CAV is not highly contagious and it takes few weeks to spread through an entire flock.
  - The incubation period is relatively long under field conditions, with the disease taking **weeks** to spread through the entire flock.

› **Organ affinity**
  - The virus can be isolated from different organs but targets mainly the **thymus**, therefore impairing the maturation of T lymphocytes.
  - Under experimental conditions, the virus is found present in most organs **after one day** (brain, liver, spleen, bursa, bone marrow, rectal contents and serum).
Morbidity and mortality rates are high dependent on the age of the chickens when infected.
Age resistance

- Chickens develop "age resistance" to disease > 2-3 weeks of age, but remain susceptible to infection.
- The period of susceptibility to disease may be extended by early exposure to IBDV, Marek's disease virus, or selected avian reo viruses that interfere with normal immune system development.
Post infection complications
- 8 days post infection haematocrit levels, thrombocytes and red and white cell counts decrease.
- Increased blood clotting time (blood takes longer to clot)
- Between 28 - 36 days post infection the haemathological parameters in recovered birds return to normal.
Introduction
Economic importance
Etiology
Transmission
Clinical signs
Post mortem lesions
Diagnosis
Prevention and control
Clinical Signs

› Birds of all ages are susceptible to infection but the clinical signs are mainly seen in young birds <2 weeks of age.

› Most outbreaks occur in broilers, followed by replacement pullets and are acutely reported at around 2 - 3 weeks of age.

› Outbreaks in older birds (replacement pullets) have been reported when other immunosuppressive agents are involved (like Marek's Disease virus and/or Infectious Bursal Disease virus).
The following may be seen:

1. Young chickens are depressed and huddle under the heat source.
2. The birds appear less developed for their age and anaemic.
Clinical Signs

1. Anorexic.
2. Depressed.
3. Exhibit a marked **pallor** that may extend to the internal organs.
4. Hemorrhages can be observed in the musculature and subcutaneously, with the wing tips frequently affected.
5. The bone marrow is pale or yellow in color and may have a fatty consistency.
6. Thymic atrophy and congestion is common and is considered diagnostic when associated with other typical signs or lesions.
7. Bursal atrophy is generally modest and transitory, typically occurring at 10 to 14 d of age in chickens vertically infected.
Cont. ...

› **Complications**
  - All of the aforementioned lesions are exacerbated and more persistent in chickens coinfect ed with infectious bursal disease virus or other lymphocidal agents.
  - Severely affected birds generally die within 2 to 4 week of age
  - Survivors are often stunted.
CAV and Immunosuppression

› The most important consequence of a CAV infection is immunosuppression.
› CAV infection impairs the immune system, affecting and multiplying in most lymphopoietic organs.
› CAV will have an impact on the generation of cytotoxic T lymphocytes to other pathogens (lymphocyte depletion).
› CAV enhances the effect of other immunosuppressive agents such as Marek's Disease virus and Infectious Bursal Disease virus.
There are also reports of enhanced signs after Infectious Bronchitis infection due to immunosuppression.

The reduction in the development of antibodies after vaccination against Newcastle Disease in CAV infected birds has been well documented.
Because of the immunosuppression, affected chickens frequently develop secondary infections with Clostridium perfringens and Staphylococcus aureus in the subcutaneous tissues and musculature that results in losses due to gangrenous dermatitis.

There may also be an increased susceptibility to adenovirus-associated inclusion body hepatitis and respiratory disease.
Gangrenous dermatitis in the wings - Blue Wing disease (BWD)

The skin lesions begin generally from wings and the adjacent areas.
Haemorrhages on the wing of a young chicken with Chicken Infectious Anemia, hence the name Blue Wing Disease.
Subcutaneous haemorrhages on the wing of a young chicken with Chicken Infectious Anemia. The name "Blue Wing Disease" is as a result of the blue discoloration seen.
Subcutaneous haemorrhage in a young chicken with Chicken Infectious Anemia, resulting in blue discoloration of the hock joint.
Plan of Talk

- Introduction
- Economic importance
- Etiology
- Transmission
- Clinical signs
- Post mortem lesions
- Diagnosis
- Prevention and control
Poste Mortem Lesions

- Focal lesions (mostly in the wings) appear as ecchymotic skin haemorrhages.
- The lesions turn blue and may break, releasing serosanguineous exudate which is prone to secondary bacterial infections, leading to gangrenous dermatitis.
- This can be especially notorious at the end of the wings, hence the name "Blue Wing Disease" used to describe this condition.
- The tips of the wings may appear haemorrhagic and necrotic.
The mortality peaks 5-6 days after the appearance of the clinical signs, declining to normal 5-6 days later.

Thymus atrophy with lobes appearing small and greyish. When observed closely, the medullar part of the lobes predominate over the cortical part.

Bone marrow atrophy, when observed closely it appears pale.
Pale anemic carcass of a young chicken with Chicken Infectious Anemia
Subcutaneous haemorrhages and petechiae in the breast muscle of a young chicken with Chicken Infectious Anemia.
Subcutaneous petechiae on the hock of a young chicken with Chicken Infectious Anemia.
Thymic atrophy
Bone marrow aplasia
Poste Mortem Lesions - Histology

› Bone marrow
  - Decrease in the number haematopoietic cells is observed 4 – 6 days post infection.
  - Followed by the appearance of large blastic cells.
  - The haematopoietic tissue is replaced by adipose tissue; this gives the bone marrow a pale appearance.
Other organs

- Depletion of lymphocytes from the thymus, spleen, Bursa Fabricius and caecal tonsils is observed, followed by hyperplasia of reticular cells.
- Changes in the thymus appear at 4 days post infection, with cortical lymphocytes disappearing and being replaced by reticular cells.
- Medullar lymphocytes are also reduced.
- Recovery starts 20 days post infection in convalescent birds.
- The changes found in the different organs suggest that CAV multiplies in the lymphocytes.
› The bursa of Fabricius and spleen may also be depleted of lymphoid cells, but the involvement and duration is less extensive than seen with the thymus.

› Survivors of CAV infection usually return to normal by approximately 4 week of age, which coincides with the onset of measurable antibody responses.
Diagnosis

- Clinical signs in young chicks are indicative of CAV infection but laboratory tests are required for a definitive diagnosis.
Clinical signs
- Diagnosis on the basis of clinical signs is very difficult.
- Very often the clinical disease may present itself in milder forms making it impossible to make a proper diagnosis.
- Post mortem findings are often not conclusive.
Laboratory tests

- Laboratory tests to identify the viral genome, antigen or antibodies are required for a definitive diagnosis.
- Testing serum samples for example at the time of the clinical signs and 2-3 weeks later provide the best basis for serological diagnosis.
- This is also applicable for monitoring vaccination results.
- The retrospective testing of sera from breeders could be done in cases where vertical transmission is suspected.
Prevention and Control

› Because the widespread distribution of the virus, being very resistant to inactivation and easily transmitted, it is probably unrealistic to assume that exposure can be limited with conventional housing and production parameters.

› Prevention and control of this disease requires a well coordinated approach, balancing biosecurity / hygienic measures and vaccination.
Control measures are directed at:

1. Limiting vertical transmission
2. Preventing coinfections with other lymphocidal agents.
Prevention and Control - Biosecurity

1. Basic management practices
   - Such as limited controlled site access, separate footwear and equipment for each site/house, and footbaths at the entrance to sites/houses all minimize the risk of introducing the virus.

2. Dry clean:
   - Removal and disposal of all organic material from the site.

3. Wet clean:
   - Clearing poultry houses using water at high pressure (35-55 Bar) to ensure removal of all organic material.
   - It is advisable to add detergents to assist the cleaning process.
4. Disinfection:
   – Application of a suitable disinfectant to reduce infectivity of any remaining virus particles.
   – Applying disinfectants at the correct concentration with a suitable contact time is critical.
   – Generally products containing formaldehyde, chlorine releasing agents, or quaternary ammonium compounds are suitable within the context of minimizing virus (and lowering infective dose) in the environment, rather than assuring complete inactivation.

5. The downtime between successive flocks must be maximized (a minimum of 10 days is recommended).
   – The control of CAV on multi-age sites is extremely challenging and requires strict control of the movement of personnel and equipment between houses.
The aim of vaccination is to protect the progeny from early infections (before 3 weeks) by means of maternally derived antibodies, afterward, there is an age resistance.
The immunization of breeding stock, several weeks before onset of lay, will efficiently prevent outbreaks of infectious anemia caused by CAV in their progeny.

Furthermore, if high uniform antibody levels can be induced in breeding stock, the resulting high levels of maternally derived antibodies (MDA) prevent or at least delay horizontal infection in the progeny and thus prevent or decrease economic losses due to subclinical disease.
Cont. …

› **Breeders**
  - Breeders are vaccinated during the rearing period between 8-16 weeks of age with a **live vaccine**.
  - Care should be taken to ensure that vaccination results in high levels of maternally derived antibodies throughout the production period.

› **Broilers and layer pullets**
  - **Broilers and layer pullets** are not commonly vaccinated against CAV in the field.
  - Early protection is achieved by vaccination of the breeders though MDA.
Maternal Immunity

- Maternally derived antibodies (MDA) offer protection against CAV infections, CAV and related infections do not appear in the progeny of immune breeder flocks.
- The level of protection is directly related to the level of MDA, the higher the MDA level, the longer the protection in the progeny.
 › MDA develop after field infections and/or vaccination
 › Field infections do not guarantee high and homogenous levels of MDA
 › Vaccination is the best way of promptly obtaining high and homogenous levels of MDA
Minimum protection level

- Breeders with different levels of MDA were challenged by inoculation with CAV
- Vertical transmission was evaluated by re-isolation of CAV from the faeces as this correlates with transmission through the eggs.
- Birds with VN titers <7 (log$_2$) still shed the challenge virus
- Birds with a VN titer of 7 (log$_2$) sporadically shed the virus
- Birds with VN titers >7 (log$_2$) did not shed the virus
› Conclusion

– To avoid vertical transmission, a MDA titer of $\geq 8 \log_2$ is needed.
– The higher the MDA level, the longer the protection in the progeny.