ADENOVIRIDAE

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STRUCTURE OF ADENOVIRUS

Fig. 17.5: Adenovirus capsid showing DNA genome, fibres, and penton and hexon capsomers.

IMAGE SOURCE: GOOGLE
### CHARACTERISTICS

- Greek word *Adenos* = gland
- first isolated from explant cultures of human adenoids
- Adenovirus virions are nonenveloped
- genome single linear molecule of ds DNA with inverted terminal repeats
- Hexagonal in outline, with icosahedral symmetry
Has 252 capsomers: 240 hexons on faces and edges of 20 equilateral triangular facets of icosahedron

12 pentons (vertex capsomers) that occupy the vertices.

From each penton projects a penton glycoprotein fiber with a terminal knob.

Avian adenoviruses have two fiber proteins per vertex
Replication takes place in the nucleus

Virions are released by cell lysis

Intranuclear inclusion bodies are formed, containing large numbers of virions

Many adenoviruses agglutinate red blood cells

Hemagglutination occurs when the tips of penton fibers bind to cellular receptors and form bridges between cells.
Adenoviruses are relatively stable in the environment

inactivated easily by common disinfectants

In cell culture, CPE characterized by rounding and clumping of affected cell, resemble “bunches of grapes”

Many adenoviruses cause acute respiratory or gastroenteric disease of varying severity

Some viruses are oncogenic in laboratory animals, but not in their natural hosts
## 05 Genera

<table>
<thead>
<tr>
<th>S.No</th>
<th>Genera</th>
<th>Host Affected</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Mastadenovirus</td>
<td>Mammals (bats, dogs, ruminants, horses, humans, swine and mice)</td>
</tr>
<tr>
<td>2</td>
<td>Aviadenovirus</td>
<td>Birds</td>
</tr>
<tr>
<td>3</td>
<td>Atadenovirus</td>
<td>Reptiles, birds, opossums and ruminants</td>
</tr>
<tr>
<td>4</td>
<td>Siadenovirus</td>
<td>Birds, reptiles and amphibians</td>
</tr>
<tr>
<td>5</td>
<td>Ichtadenovirus</td>
<td>Fish</td>
</tr>
<tr>
<td>S.No.</td>
<td>Virus</td>
<td>Disease</td>
</tr>
<tr>
<td>-------</td>
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<td>-------------------------------------------------------------------------</td>
</tr>
<tr>
<td>1</td>
<td>Canine adenovirus 1</td>
<td>Infectious canine hepatitis, lesions arising from direct cytopathic effects and immune complex formation</td>
</tr>
<tr>
<td>2</td>
<td>Canine adenovirus 2</td>
<td>Involved in infectious tracheobronchitis (kennel cough), a highly contagious respiratory disease</td>
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<tr>
<td>3</td>
<td>Equine adenovirus A</td>
<td>Subclinical or mild respiratory infection; associated with pneumonia in Arabian foals with combined immunodeficiency disease</td>
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<tr>
<td>4</td>
<td>Bovine adenoviruses</td>
<td>Associated with occasional outbreaks of respiratory and enteric disease</td>
</tr>
<tr>
<td>5</td>
<td>Ovine adenoviruses</td>
<td>Associated with occasional outbreaks of respiratory and enteric disease</td>
</tr>
<tr>
<td>S.No.</td>
<td>Virus</td>
<td>Disease</td>
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<tr>
<td>6</td>
<td>Porcine adenoviruses</td>
<td>Usually subclinical infections, occasionally cause diarrhoea</td>
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<tr>
<td>7</td>
<td>Fowl adenoviruses</td>
<td>Associated with quail bronchitis and inclusion body hepatitis</td>
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<tr>
<td>8</td>
<td>Egg drop syndrome virus</td>
<td>Causes egg drop syndrome in laying hens</td>
</tr>
<tr>
<td>9</td>
<td>Pheasant adenovirus</td>
<td>Causes marble spleen disease with sudden deaths in 2-8 month old birds; acute pulmonary oedema and splenic Necrosis</td>
</tr>
<tr>
<td>10</td>
<td>Turkey haemorrhagic enteritis virus</td>
<td>Causes turkey haemorrhagic enteritis, dysentery in 4-12 week-old poult's; mortality rate up to 60%</td>
</tr>
</tbody>
</table>
Canine adenovirus 1, genus *Mastadenovirus*

- Causes **Infectious canine hepatitis (ICH)**
  - In dogs, the virus may cause respiratory or ocular disease.

- Also an important pathogen of foxes, wolves, coyotes, skunks and bears

- Virus was first recognized as the cause of fox encephalitis
INFECTIOUS CANINE HEPATITIS

- Ist recognized by Rubarth in 1947, also known as Rubarth’s disease.

- Transmission by direct contact with infected animal.

- Urine, faeces or saliva from infected animals—source of infection

- Infectious canine hepatitis, involves massive destruction of hepatocytes, resulting in peracute death.
Canine adenovirus 1

Ingestion

Local replication in tonsils and Peyer's patches

Viraemia

Cytopathic effects

Antibody production due to viral replication
Cytopathic effects due to viral replication

- Hepatocytes
  - Acute hepatitis
  - I/N IB
- Renal tubule Cells
  - Nephritis
  - Virus shed in urine
- Vascular endothelial
  - Haemorrhages
  - I/N IB

Antibody production

- Immune complex formation
  - Circulating immune complex
  - Deposition in renal glomeruli
    - Glomerulonephritis
  - Localized immune complex
    - corneal oedema
    - and opacity; Anterior uveitis
most frequently encountered in young dogs.

mortality rate ranges from 10 to 30% in mature dogs and up to 100% in young pups.

In peracute disease-

- rapid death

- without apparent preceding illness or, after an illness lasting only 3 or 4 h
ACUTE DISEASE

- Fever, depression, anorexia, increased thirst, vomiting and diarrhoea

- Conjunctivitis, serous discharge from eyes and nose

- Petechial hemorrhages of gums, pale mucous membranes

- Icterus (jaundice), abdominal pain

- There may be tachycardia, leukopenia, prolonged clotting time and disseminated intravascular coagulation

- may be bleeding around deciduous teeth

- Severely affected dogs may convulse
MILD DISEASE

- may actually be a vaccine-modified disease—result of partial immunity

- 7-10 days after acute signs disappear, about 25% of affected dogs develop

- bilateral corneal opacity ("blue eye") seen, usually disappears spontaneously

- characteristic and diagnostically useful

- caused by deposition of virus antibody complexes in small blood vessels of ciliary body

- Interferes with normal fluid exchange within the cornea
Edema and hemorrhage of superficial LN

- hemorrhages on serosal surfaces
- liver, spleen enlarged, mottled splenic parenchyma
- wall of gallbladder - thickened, edematous
- cortical renal hemorrhages, pulmonary consolidation
- Centrilobar necrosis of hepatocytes and I/N IB in hepatic parenchyma - pathognomonic
- Intranuclear inclusions in Kupffer’s cells, hepatocytes, in endothelial cells of kidney
History of fever, sudden collapse and abdominal pain in young, unvaccinated dogs - suggestive of ICH.

Marked reduction in neutrophils and lymphocytes during the febrile stage.

Clotting time may be prolonged.

I/N IB in hepatocytes, Kupffer cells and endothelial cells are confirmatory.

Virus isolation - cell lines of canine origin (e.g., Madin Darby canine kidney cells)

PCR, ELISA
DIAGNOSIS

- Suitable specimens for virus isolation
  - **During febrile stage**- oropharyngeal swabs, blood, urine and faeces.
  - **At postmortem**- Spleen, LN and kidney

- Due to high levels of arginase activity, liver samples are unsuitable for cell culture.

- A rising antibody titre, using virus neutralization or HI tests- indicative of active CAV-I infection.
TREATMENT

- Supportive treatment may allow time for hepatocellular regeneration.

- Intravenous fluids administration to counteract dehydration and control shock.

- In severely ill animals, blood transfusions to prevent or control haemorrhage.
Both inactivated and live-attenuated canine adenovirus 1 vaccines

Live-attenuated CAV 1 vaccines results in mild nephropathy with shedding of virus in urine and, in some instances, corneal opacity.

CAV 2 vaccine-
- No side effects
- Stimulate effective long lasting immunity to CAV-1.
EGG DROP SYNDROME VIRUS

- classified in the genus *Atadenovirus*

- likely originated in ducks and spread to chickens through a contaminated vaccine.

Causes egg drop syndrome, first reported in 1976

- virus grows to high titer in embryonating eggs of ducks or geese

- Grow in cell cultures derived from ducks, geese, or chickens—esp. in duck kidney, duck embryo liver and duck embryo fibroblasts
EGG DROP SYNDROME

- Chickens - major species affected, looks apparently healthy

- **Transmission**-
  - Through contaminated eggs
  - Droppings, contaminated fomites
  - By needles used for vaccinations
  - Virus is transmitted vertically in eggs
IRREGULARLY SHAPED EGGS, SOFT-SHELLED OR SHELL-LESS EGGS AND DEPIGMENTATION OF EGGS

IMAGE SOURCE: GOOGLE
SYMPTOMS

- loss of color in pigmented eggs
- soft-shelled, thin-shelled, and shell-less eggs
- Thin-shelled eggs may have rough or even sandpaper-like surface
- production of soft-shelled and shell-less eggs
- drop in egg production or failure to reach peak production is seen.
Characteristic lesions in pouch shell gland and oviduct

- Their epithelial cells become necrotic and contain intranuclear inclusion bodies
- There is associated inflammatory infiltration
- These findings are virtually pathognomonic
By virus isolation-
✓ In embryonated duck or goose eggs by allantoic cavity route

✓ In cell culture

▪ Hemagglutination inhibition

▪ Virus neutralization test

▪ FAT
PREVENTION AND CONTROL

- Preventing contact with other birds, especially waterfowl
- Disinfecting all equipment regularly
- Chlorination of water
- Inactivated vaccines for chickens before they begin laying eggs
- Vaccination only reduce, rather than eliminate virus
Quail bronchitis virus

- Causes Quail bronchitis, an important disease of quail
- Etiologic agent is avian (fowl) adenovirus 1 (now designated as fowl aviadenovirus A)
- Belongs to genus Aviadenovirus
- Virus is highly contagious and spreads rapidly through flocks
### CLINICAL SIGNS

<table>
<thead>
<tr>
<th>In young birds-</th>
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<tbody>
<tr>
<td>➢ respiratory distress, open-mouth breathing</td>
<td></td>
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<tr>
<td>➢ nasal discharge, coughing, sneezing, rales, lacrimation and conjunctivitis</td>
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<tr>
<td>➢ Mortality may be 100% in young birds</td>
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</tbody>
</table>

<table>
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<tr>
<th>In older birds-</th>
<th></th>
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<tbody>
<tr>
<td>➢ There is also diarrhea</td>
<td></td>
</tr>
<tr>
<td>➢ Less than 25% in birds aged more than 4 wks</td>
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</tbody>
</table>
LESIONS

- Necrotic or hemorrhagic tracheitis, with distinct large basophilic intranuclear inclusions
- Air sacculitis, multifocal hepatic necrosis
- Gaseous mucoid enteritis

Control

- Strict isolation, quarantine of introduced birds
- Decontamination of premises and equipment
- Long lasting immunity in recovered birds.
By fowl adenovirus 4, genus *Aviadenovirus* (now designated fowl aviadenovirus C)

Disease is also known as-
- Hydropericardium Syndrome
- Angara Disease
- Litchi disease

Broilers of 3-5 wks are commonly affected, infection through oral route

Virus can spread by horizontally and vertically
Severe disease require co-infection with an immunosuppressive agent or exposure to immunosuppressive aflatoxins

- Virus multiply in RBC and hepatic cells

- Infection of liver cells affects synthesis of albumin

- Hydropericardium condition is seen in affected birds
**CLINICAL SIGNS & LESIONS**

- **Sudden mortality** is a feature of the disease
- Depression, ruffled feathers, yellowish faeces
- Reluctant to move, chest and beak rest on ground

**Lesions**
- Enlarged liver with haemorrhages and necrosis
- Intramuscular haemorrhage and anaemia
- Intranuclear inclusions in hepatocytes are prominent.
Provisional diagnosis based on symptoms and lesions

Intranuclear inclusion bodies in hepatocytes

AGPT, ELISA, PCR

Virus isolation-
- In embryonated hen’s eggs- by CAM or yolk sac route
- Chicken embryo’s liver or kidney cell culture

Control by vaccination, use of immunomodulator, sanitation
Thanks