# ADENOVIRIDAE

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# STRUCTURE OF ADENOVIRUS

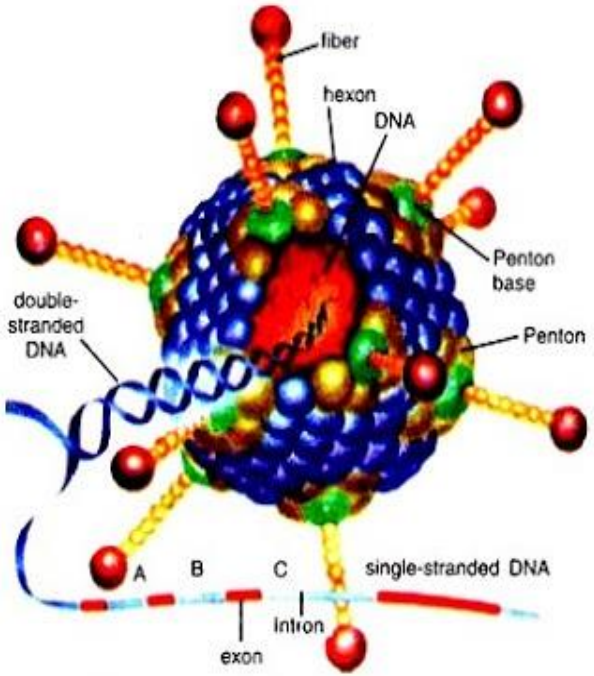



Fig. 17.5 : Adenovirus capsid showing DNA genome, fibres, and penton and hexon capsomeres.

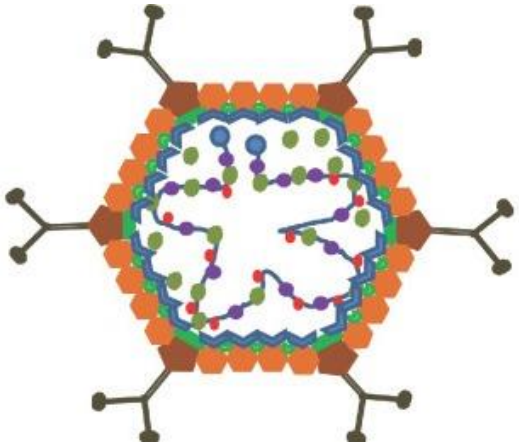


IMAGE SOURCE: GOOGLE

# 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 occurrs 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

# CLASSIFICATION

## 05 GENERA

| S.No | Genera | Host Affected |
|---|---|---|
| 1 | Mastadenovirus | Mammals (bats, dogs, ruminants, horses, humans, swine and mice) |
| 2 | Aviadenovirus | Birds |
| 3 | Atadenovirus | Reptiles, birds, opossums and ruminants |
| 4 | Siadenovirus | Birds, reptiles and amphibians |
| 5 | Ichtadenovirus | Fish |

# ADENOVIRUSES OF VETERINARY IMPORTANCE

| S.No. | Virus | Disease |
|---|---|---|
| 1 | Canine adenovirus 1 | Infectious canine hepatitis, lesions arising from direct cytopathic effects and immune complex formation |
| 2 | Canine adenovirus 2 | Involved in infectious tracheobronchitis (kennel cough), a highly contagious respiratory disease |
| 3 | Equine adenovirus A | subclinical or mild respiratory infection; associated with pneumonia in Arabian foals with combined immunodeficiency disease |
| 4 | Bovine adenoviruses | Associated with occasional outbreaks of respiratory and enteric disease |
| 5 | Ovine adenoviruses | Associated with occasional outbreaks of respiratory and enteric disease |

| S.No. | Virus | Disease |
|---|---|---|
| 6 | Porcine adenoviruses | Usually subclinical infections, occasionally cause diarrhoea |
| 7 | Fowl adenoviruses | Associated with quail bronchitis and inclusion body hepatitis |
| 8 | Egg drop syndrome virus | Causes egg drop syndrome in laying hens |
| 9 | Pheasant adenovirus | Causes marble spleen disease with sudden deaths in 2-8 month old birds; acute pulmonary oedema and splenic Necrosis |
| 10 | Turkey haemorrhagic enteritis virus | Causes turkey haemorrhagic enteritis, dysentery in 4-12 week-old poults; mortality rate up to 60% |

# INFECTIOUS CANINE HEPATITIS VIRUS

- 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.

# PATHOGENESIS

**Canine adenovirus 1**

↓

Ingestion

↓

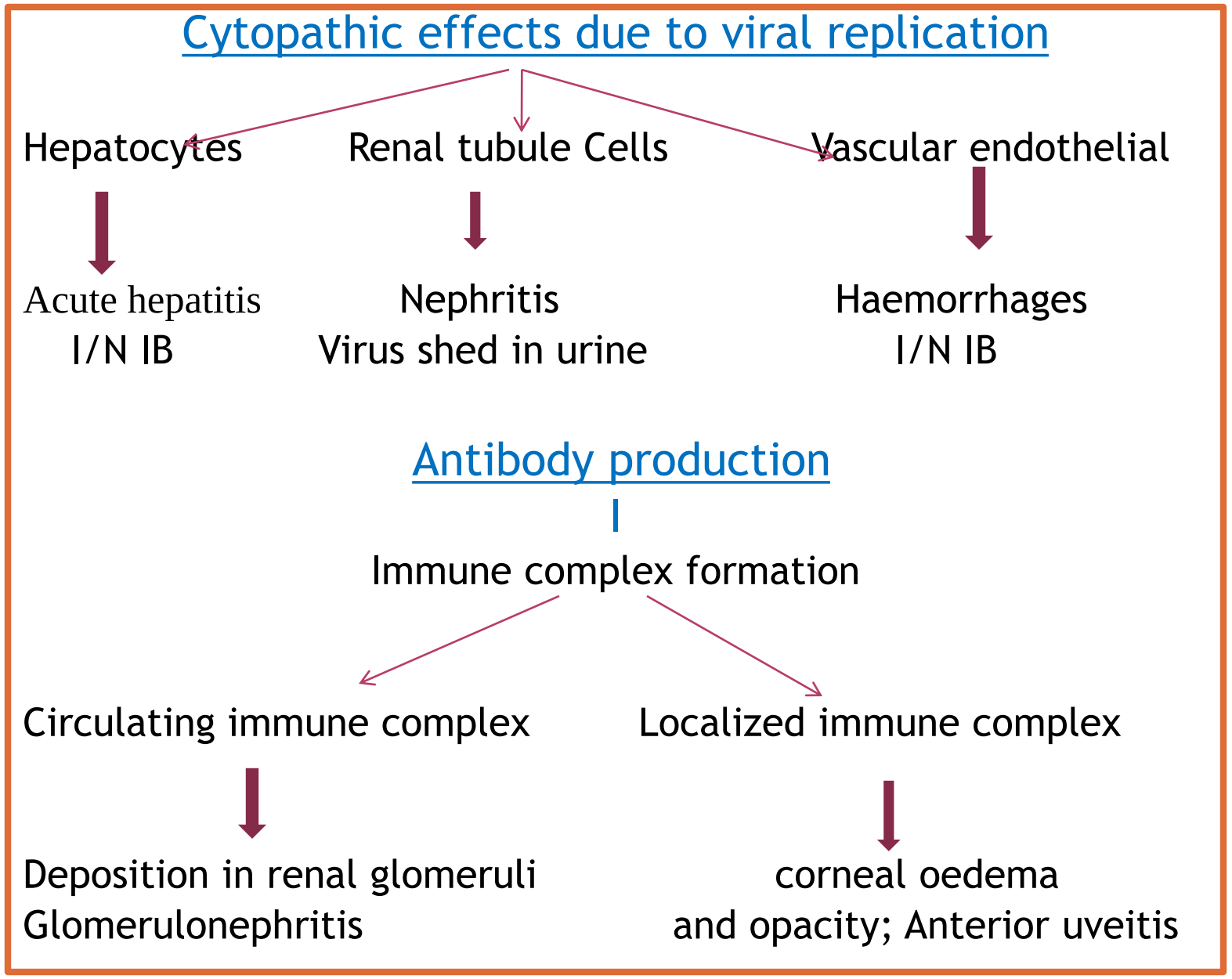
Local replicaton in tonsils and Peyer's patches

↓

Viraemia

Cytopathic effects due to viral replication

Antibody production


Cytopathic effects due to viral replication
Hepatocytes
Renal tubule Cells
Vascular endothelial
Acute hepatitis
I/N IB
Nephritis
Virus shed in urine
Haemorrhages
I/N IB
Antibody production
Immune complex formation
Circulating immune complex
Localized immune complex
Deposition in renal glomeruli
Glomerulonephritis
corneal oedema
and opacity; Anterior uveitis

# CLINICAL SIGNS

- 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

# LESIONS

- 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

# DIAGNOSIS

- History of fever, sudden collapse and abdominal pain in young, unvaccinated dogs- suggestive of ICH.
- Marked reduction in neutrophils an 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

# PREVENTION AND CONTROL

- 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.

# LESIONS

- 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

# DIAGNOSIS

- 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

# FOWL ADENOVIRUS

## 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

In young birds-

- respiratory distress, open-mouth breathing
- nasal discharge, coughing, sneezing, rales, lacrimation and conjunctivitis
- Mortality may be 100% in young birds

In older birds-

- There is also diarrhea
- Less than 25% in birds aged more than 4 wks

# 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.

# INCLUSION BODY HEPATITIS

- 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

# PATHOGENESIS

- 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

# CLINCICAL 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.

# DIAGNOSIS & CONTROL

- 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