Inclusion Body Hepatitis

Inclusion body hepatitis (IBH) was first described in the U.S. in 1963 concurrent with a severe respiratory outbreak in broilers and since then has been reported in many countries around the world. Subsequently, it was determined to be associated with avian adenovirus (AAV) infection.

•The first AAV associated with clinical disease was isolated from an outbreak of respiratory disease in quail in 1950 . Since that time, AAVs have been found to be ubiquitous in all types and breeds of chickens (normal flora)

In Pakistan Inclusion Body Hepatitis, or hydropericardium syndrome first appeared in the commercial broiler chickens during late 1987,causing high mortality. Later the syndrome has also affected medium weight laying strains and broiler breeder strains of the chicken. The disease primarily affects liver, heart, kidneys and lungs.

The syndrome is characterized by an accumulation of clear, straw-coloured fluid in the pericardial sac, with enlargement and discoloured liver and enlarged kidneys.

The syndrome is an acute disease of young chickens associated with anemia, hemorrhagic disorders, and hydropericardium. It is a common disease in several countries, where broilers are severely affected, resulting in high mortality rates.

Avian adenoviruses (AAVs) in chickens are the etiologic agents of two important diseases known as inclusion body hepatitis (IBH) and hydropericardium syndrome (HP).
Although in some cases each disease is observed separately, the two conditions have been frequently observed as a single entity; therefore, the name hepatitis hydropericardium has been widely used to describe the pathologic condition.

Initially, it was thought that IBH could only be caused by adenovirus if the bird's immune system was first weakened by exposure to immunosuppressive agents such as infectious bursal disease (IBD) and chicken anemia virus (CAV). Recent work, however, has demonstrated that virulent strains alone can produce the disease.

Etiology

The AAVs classified in the genus *Aviadenovirus* (formerly group I), are the etiologic agents of this condition. Although there are 12 different serotypes of AAV, the most common viruses isolated in cases of IBH/HP belong to serotypes **4 and 8**.

These AAVs are capable of producing the disease without the immunosuppressive effects of associated viruses such as infectious bursal disease or other immunosuppressive agents. However, the association with immunosuppressive viruses such as IBDV and chicken anemia virus will result in a more severe disease.

Transmission

Both vertical and horizontal transmission plays a role in the spread of **IBH**.

Most outbreaks are initiated by transmission of the virus through the **embryonated eggs** and hens exposed during production will typically shed the virus to their progeny for **three to six weeks** until development of immunity occurs.

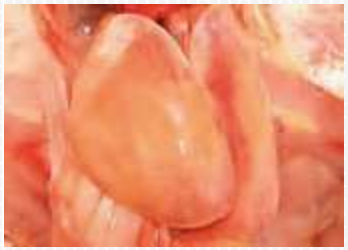
Horizontal transmission has also been demonstrated; young chicks in contact with infected chicks can die of peracute IBH/HP.

Horizontal spread occurs primarily from contact with infected feces. This seems to occur quite frequently and most cases are not diagnosed because they do not become a clinical problem.

Commercial hatching eggs may be a mechanism of spread of endemic AAV from one area to another. There is evidence that adenovirus infections can become latent and that periods of stress, such as the onset of egg production, will reactivate viral shedding.

Infection with some strains of AAVs may result in minimal hepatic disease; however, if birds have been infected with immunosuppressive viruses (IBDV, CAV, Marek disease), the clinical disease becomes evident.


www.PoultryMed.com




www.PoultryMed.com

Clinical Signs

1-Birds usually found dead.

2-Shanks and comb become very pale.

3-Reduced growth rate.

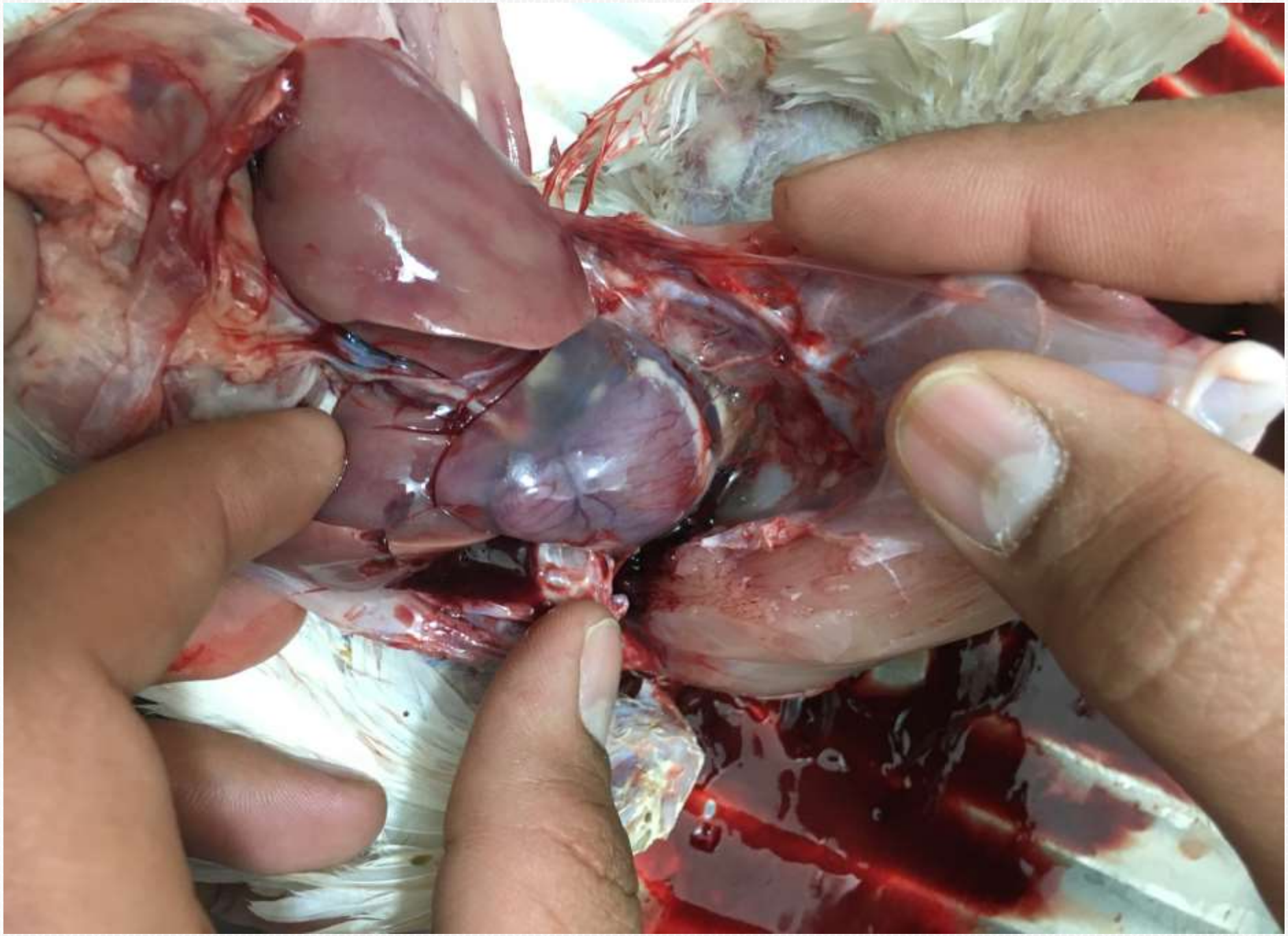
4-Sudden mortality usually is seen in chickens <4 wk old and as young as 4 days of age. Mortality normally ranges from 2%–40%, especially when birds are <3 wk old. However, in some outbreaks, mortality has reached 80%. Mortality rates also vary depending on the pathogenicity of the virus and infection with other viral or bacterial agents.

5-Flocks of 3- to 5-wk-old broilers with HP may not show specific clinical signs, but sudden onset of mortality, **lethargy**, **huddling with ruffled feathers**, and **yellow, mucoid droppings** may be seen.

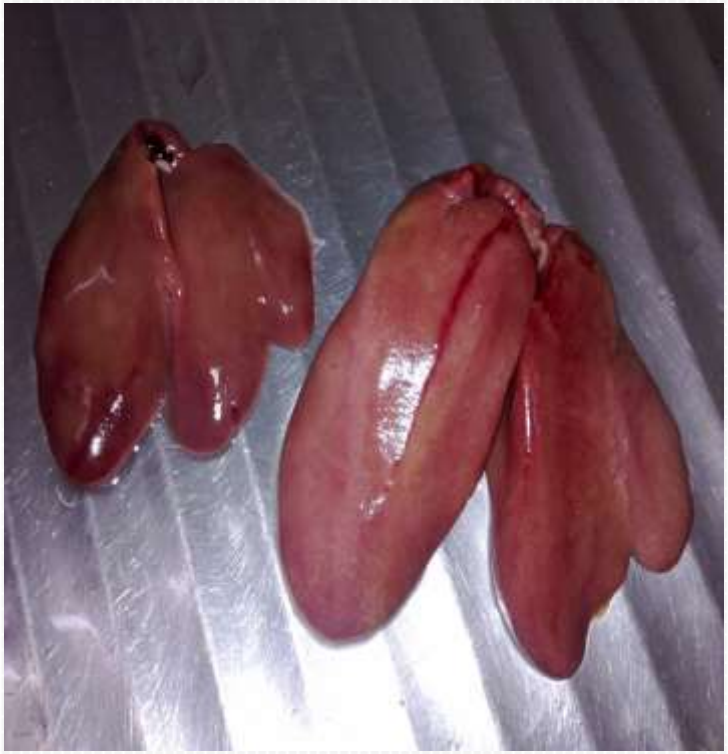
The duration of the infection usually ranges from 9–14 days with morbidity of 10%–30% and a daily mortality of 3%–5%.

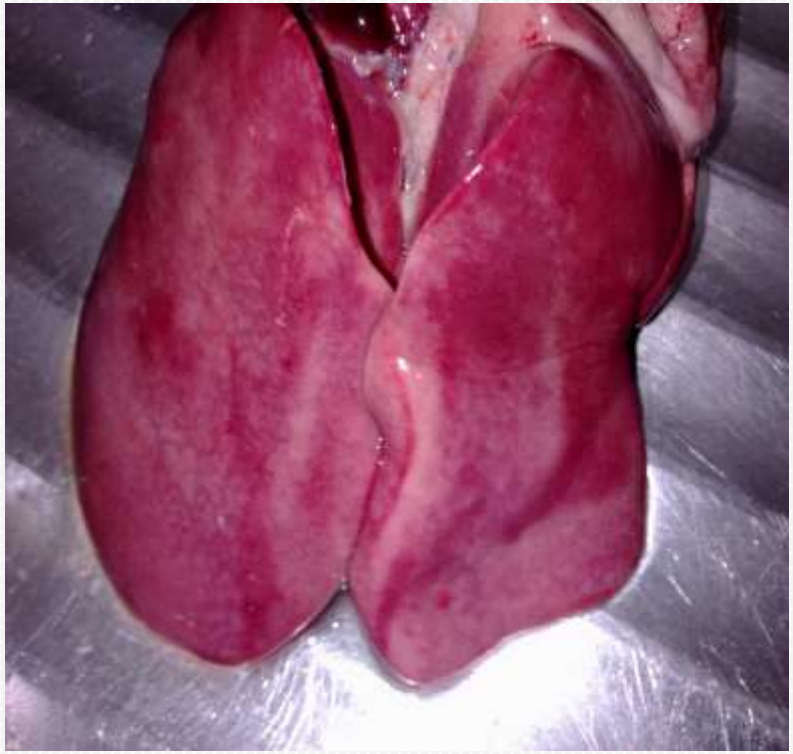
Gross lesions

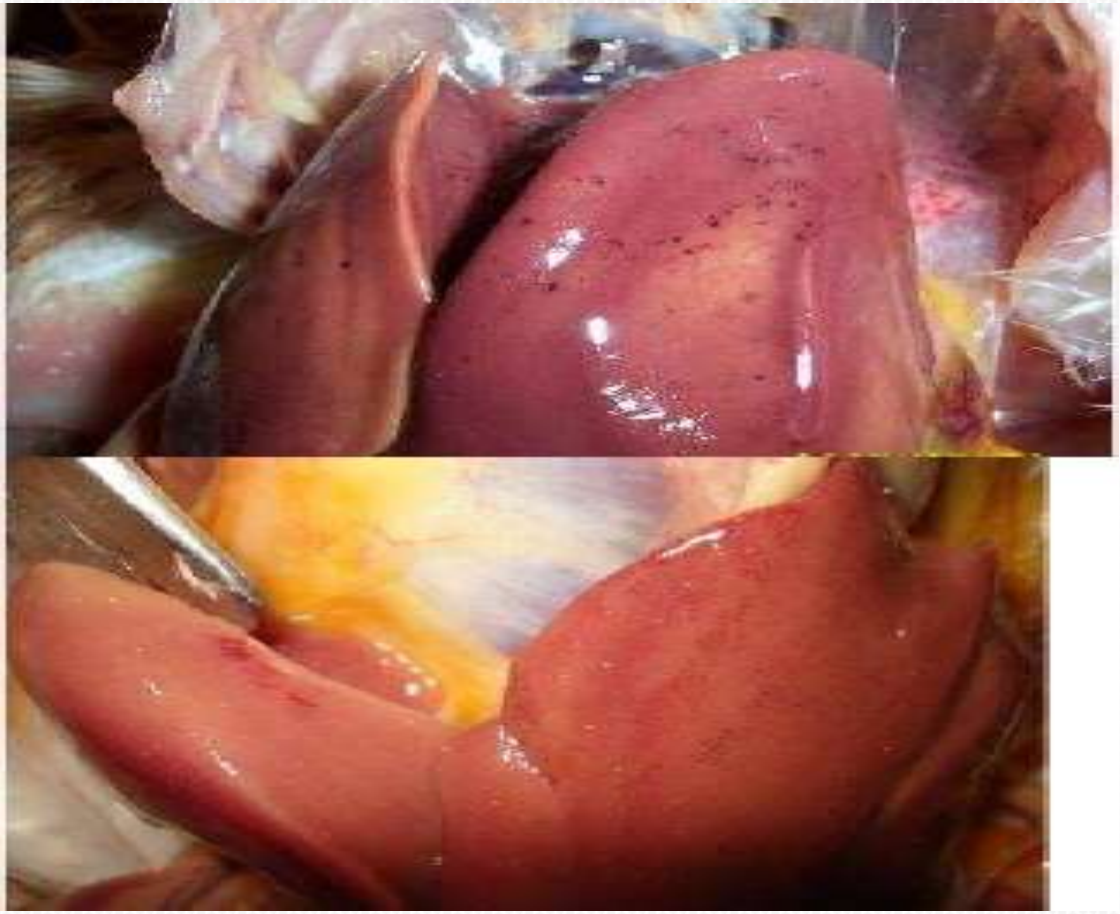
1-Disease is characterized by the accumulation of about 10 mL of a clear straw-colored fluid is present in the sac surrounding the heart.



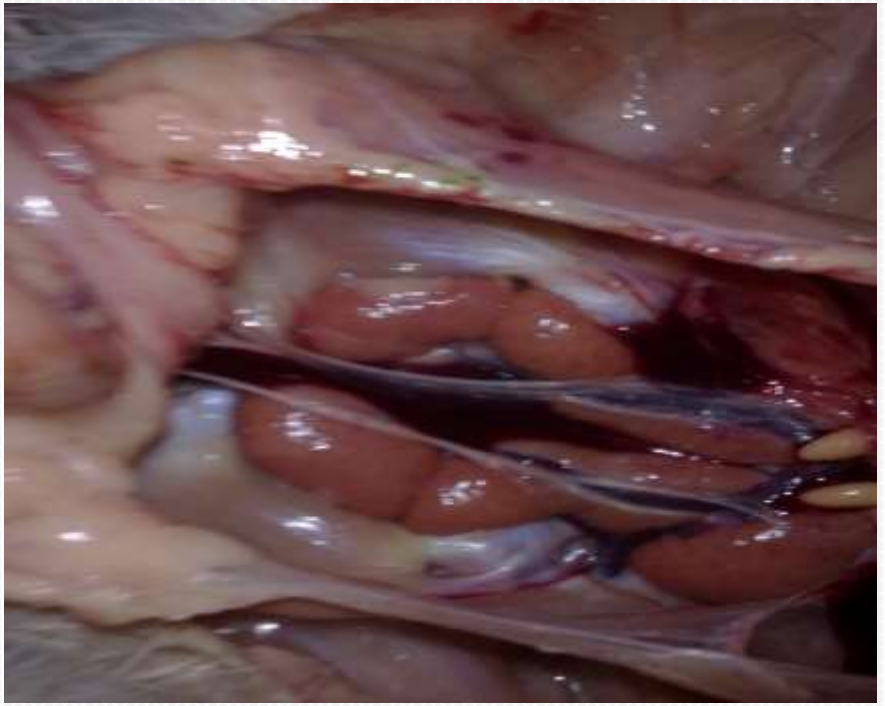
2-The most consistent findings in this condition were encountered in the liver. On gross examination, the appearance of the livers was very similar in all outbreaks. The liver is the primary organ affected in these birds which is enlarged, pale yellow with multiple petechial haemorrhages. The parenchyma of the liver was soft in consistency

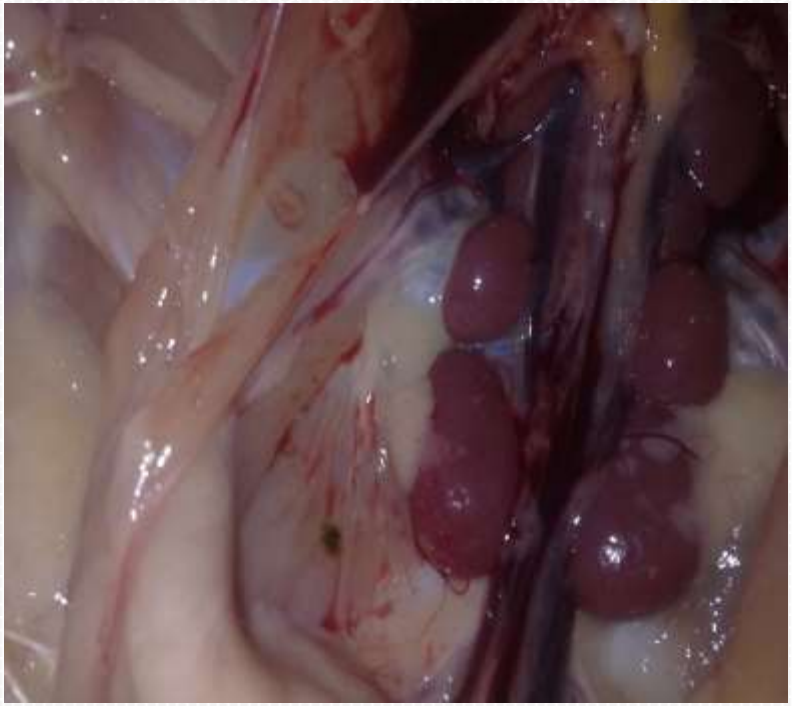


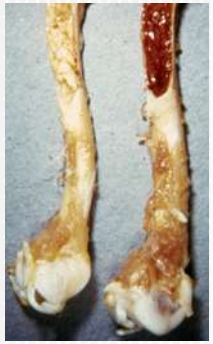




3-Kidneys enlarged, swollen and pale with some hemorrhage.
4-Bone marrow very pale.
5-Small bursa of Fabricius.



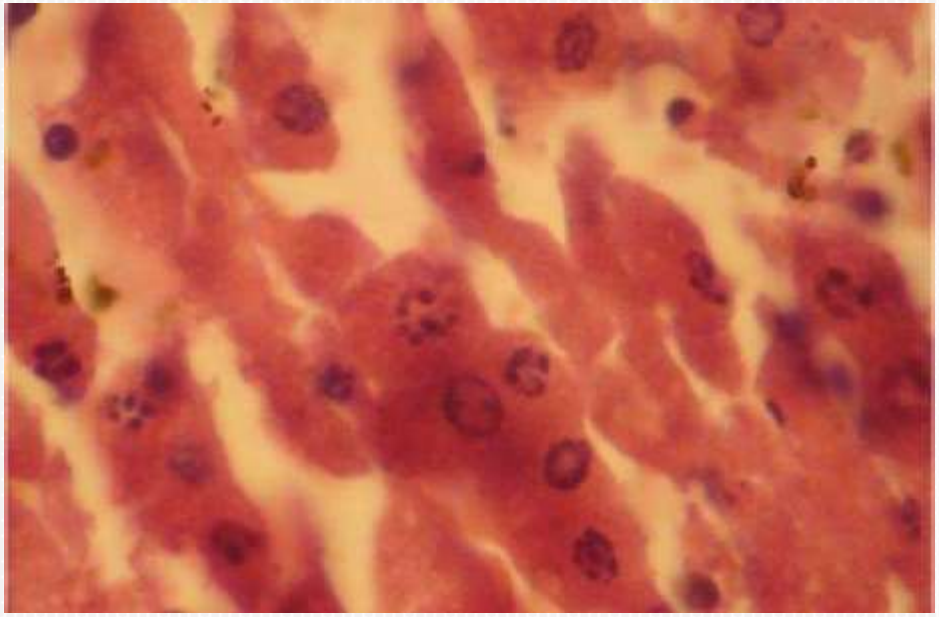




Histopathologic lesions

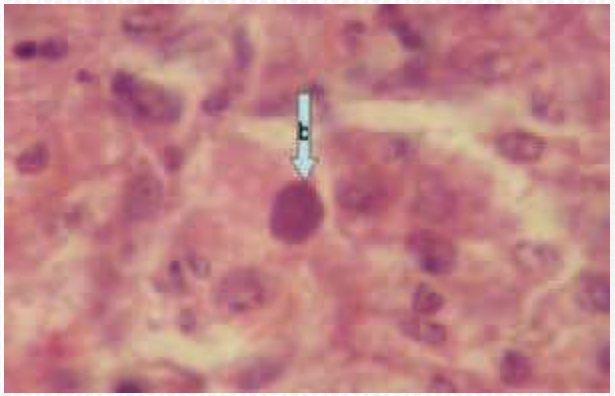
Varying degrees of pyknosis, karyorrhexis and karyolysis were observed in the majority of the hepatic cells.

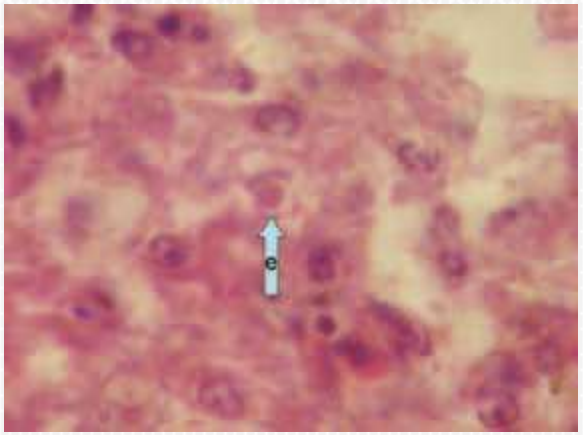
Multiple subcapsular hemorrhages, multifocal groups of hepatocytes and lipid degeneration were also present. In some hepatic cells, swelling of the nuclei and margination of the chromatin were apparent.



Histopathologic lesions

In the nuclei of hepatocytes, basophilic or eosinophilic inclusion bodies are detected. The basophilic inclusion bodies are usually dense and occupy the entire nuclear inner space (arrow b), whereas the eosinophilic ones are round or irregularly shaped and surrounded by a light halo (arrow e).





Diagnosis

Is based upon the typical gross lesions and the history records.

A principal approach in IBH diagnostics is the

1- **histological investigation** that helps to detect the intranuclear inclusion bodies.

2-IBH should be distinguished mainly from IBD and chicken infectious anaemia (CIA).

3- A tentative diagnosis is based on typical microscopic findings and confirmed by isolating adenoviruses from the liver.

4-Viral Isolation, need SPF eggs. Some embryos will have liver lesions grossly and microscopically.

4-Serology, restriction enzyme analysis, and PCR are used to classify adenoviruses isolated from clinical cases. This information is used for epidemiologic studies.

Treatment and Prevention

1-As with many other viral diseases, there is no treatment. **Antibiotics may help** prevent secondary bacterial infections.

Sulfonamides are contraindicated if evidence of hematologic disease or immunosuppression is seen.

2-With regard to IBH prevention and control, the eggs of broiler parent flocks, where the disease is consecutively appearing in the progeny, should not be used for hatching.

3-The access of wild birds should be prevented as they are potential carriers and distributors of the virus.

4- The most important steps in IBH prevention are the control of IBD and CIA.

5-Vaccines against IBH/HP are not commercially available in the USA; however, in other countries both live and inactivated vaccines are used to control the syndrome. The AAV serotypes most frequently used to prepare commercial vaccines are serotypes 4 and 8.

In other countries, including Mexico, Pakistan, **Iraq** and many countries in South America, inactivated vaccines are routinely used to vaccinate breeders and broilers.

When breeders are properly vaccinated, antibodies generated by the vaccine are transmitted to the progeny, providing protection against field infections and clinical disease. Broilers are vaccinated at <10 days of age when their parents either do not have serotype-specific adenovirus antibodies or maternal antibody transmission is irregular because of improper vaccination procedures that result in a substantial number of unvaccinated birds.