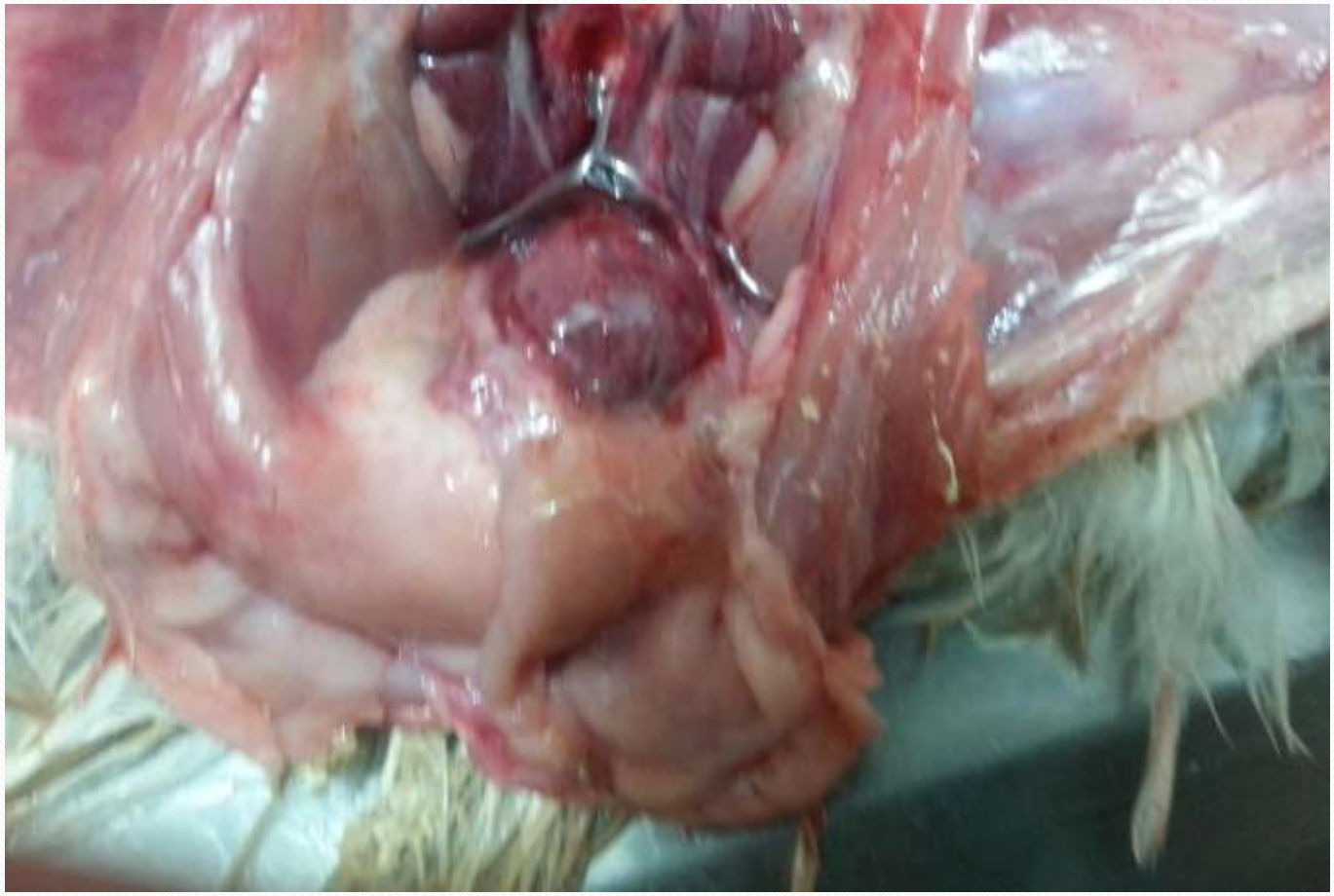
Infectious Bursal Disease
(GUMBORO OR IBD)



Infectious Bursal Disease (Gumboro or IBD)

is an acute, highly contagious viral infection in chickens manifested by inflammation and subsequent atrophy of the bursa of Fabricius, various degrees of nephroso-nephritis and immunosuppression. Clinically the disease is seen only **in chickens older than 3 weeks**. The feathers around the vent are usually stained with faeces containing plenty of urates.

This form can result in high mortality. Infection prior to 3 weeks of age results in **immunosuppression** and bursal atrophy due to destruction of undifferentiated lymphocytes.

in **1962** was recognized and referred to as **"avian nephrosis**" because of the extreme kidney damage found in birds that succumbed to infection.

Since **the first outbreaks** occurred in the area of Gumboro, Delaware in USA, "Gumboro disease" was a synonym for this disease and is still frequently used. It is economically important to the poultry industry worldwide due to increased susceptibility to other diseases and negative interference with effective vaccination.

No obvious clinical disease causes by the virus but it can produces severe immunosuppression with bursal atrophy regardless of age of infection.

Disease is of economic importance for 2 reasons

1-As it causes heavy mortality in the chickens of 3 weeks of age & older.

2- It causes **immunosuppression** which leads to vaccination failure, E.coli infection, gangrenous dermatitis, & Inclusion body hepatitis.

The virus does not affect man and has no public health significance.

Etiology:

RNA virus, a member of the **Birnaviridae** family.

The family has 3 genera:

1-Aquabirnavirus included infectious pancreatic necrosis virus, which infects fish.

2-Avibirnavirus whose type species is infectious bursal disease virus, which infects birds and this genera have two serotypes (serotype 1 and serotype 2) which they differentiated by virus neutralization test.

3-Entomobirnavirus whose type species is Drosophila X virus, which infects insects.

The Avibirnavirus viral genomes was nonenveloped with icosahedral symmetry and a diameter varying from 55—65 nm, consisting of 2 segments of double stranded RNA (dsRNA).

There are two distinct serotypes of the virus (1 and 2), but only **serotype 1** viruses cause disease in poultry.

At least six antigenic subtypes of IBDV serotype 1 have been identified by in vitro by cross-neutralization assay.

Viruses belonging to one of these antigenic subtypes are commonly known as **variants**, which were reported to break through high levels of maternal antibodies in commercial flocks, causing up to 60 to 100 percent mortality rates in chickens.

IBD is highly contagious and very difficult to remove from a house. It tends to reoccur on the same farm.

It survives for long periods (at least 6 months) in poultry houses even where thorough cleaning and disinfection procedures are followed.

Subclinical infection in young chicks results in:

Deficient immunological response to **Newcastle disease**, **Marek's disease** and **Infectious Bronchitis**; susceptibility to **Inclusion Body Hepatitis** and **gangrenous dermatitis** and increased susceptibility to **CRD**.

The route of infection

Is usually oral by contaminated feed,water , servicemen and trucks , but may be via the conjunctiva or respiratory tract, with an incubation period of 2-3 days.

The disease is highly contagious. Mealworms and litter mites may harbour the virus for 8 weeks, and affected birds excrete large amounts of virus for about 2 weeks post infection.

There is no vertical transmission.

The bursa of Fabricius is an immune organ that produces B lymphocytes which migrate to secondary immune organs (cecal tonsil, Harderian gland, etc.) for the purpose of antibody production.

Embryos are immunocompetent by 18 days and B-cells begin to leave the bursa at this time.

However, full B-cell production peaks at 3 weeks and this is when the birds are most susceptible to the virus and classic IBD infection.

Susceptibility to Physical and Chemical Agents:

Infectious bursal disease virus is very stable.

IBDV resisted treatment with **ether and chloroform**, was inactivated at pH 12 but unaffected by pH 2, and was still viable after 5 hours at 56°C. Only the iodine complex had any deleterious effects.

Certainly, the hardy nature of this virus is one reason for its persistent survival in poultry houses even when thorough cleaning and disinfection procedures are followed.

Morbidity and Mortality

In fully susceptible flocks, the disease appears suddenly, and there is a high morbidity rate, usually approaching 100%.

Mortality may be nil but can be as high as 20—30%, usually beginning on day 3 postinfection and peaking and receding in a period of 5—7 days.

Pathogenicity:

Chickens are the only animals known to develop clinical disease and distinct lesions when exposed to IBDV. Field viruses exhibit different degrees of pathogenicity in chickens. Vaccine viruses also have varying pathogenic potential in chickens.

Incubation Period and Clinical Signs

The incubation period is very short, and clinical signs of the disease are seen within 48-72 hours and the course of disease extend from 5-7 days after exposure.

1-Vent picking

2- Whitish or watery diarrhea

3- Anorexia and depression.

4-Ruffled feathers

5- Trembling, severe prostration, and finally, death.

6-Affected birds became dehydrated, and in terminal stages of the disease, had a subnormal temperature.

Gross Lesions

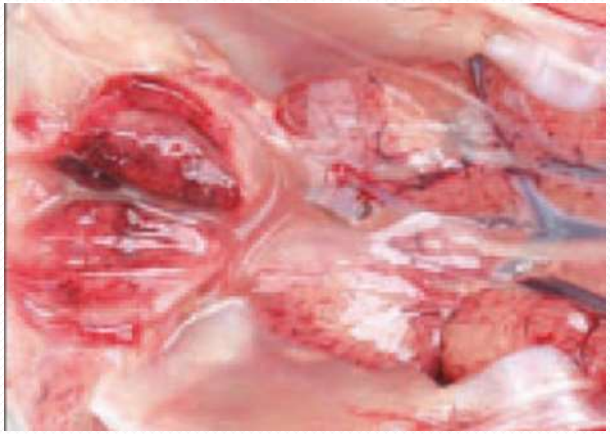
1-Birds that succumb to the infection are dehydrated, with darkened discoloration of pectoral muscles.

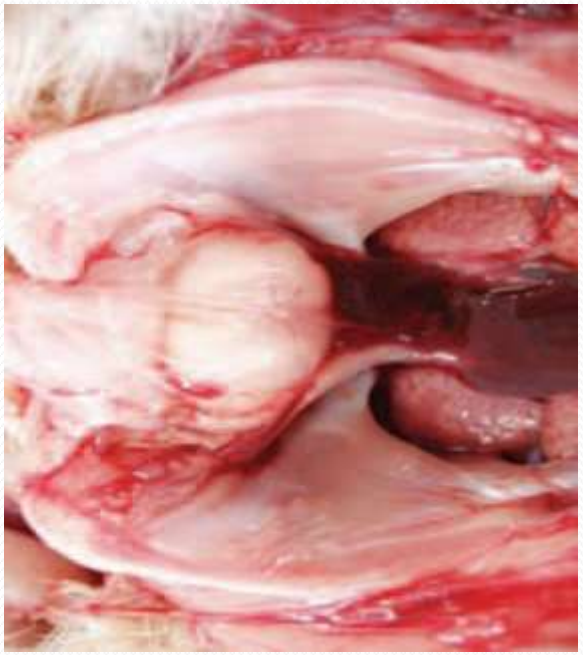
2-Frequently, hemorrhages are present in the thigh and pectoral muscles.

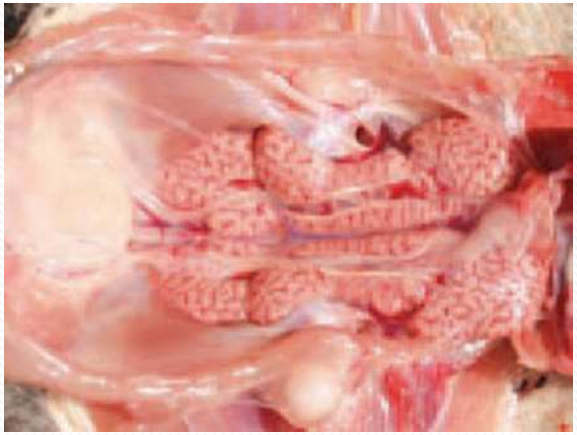
3-By day 2 or 3 postinfection, the bursa has a gelatinous yellowish transudate covering the serosal surface. Longitudinal striations on the surface become prominent, and the normal white color turns to cream color. The transudate disappears as the bursa returns to its normal size, and the organ becomes gray during and following the period of atrophy.

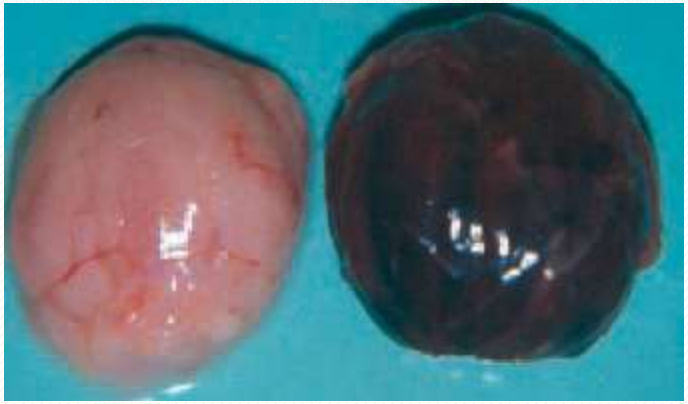
4-Kidneys are swollen and filled with urates

Depressed Chicks

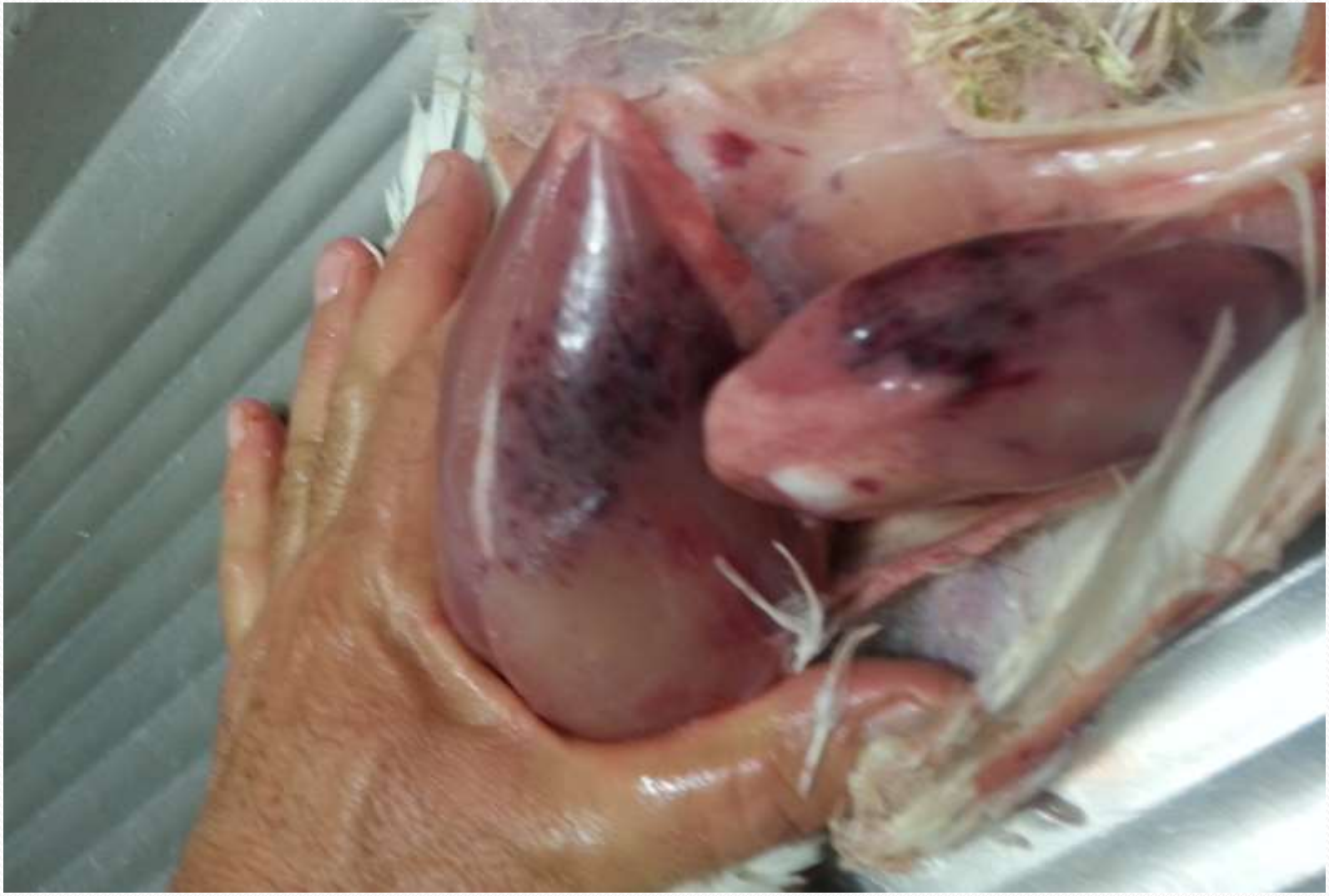


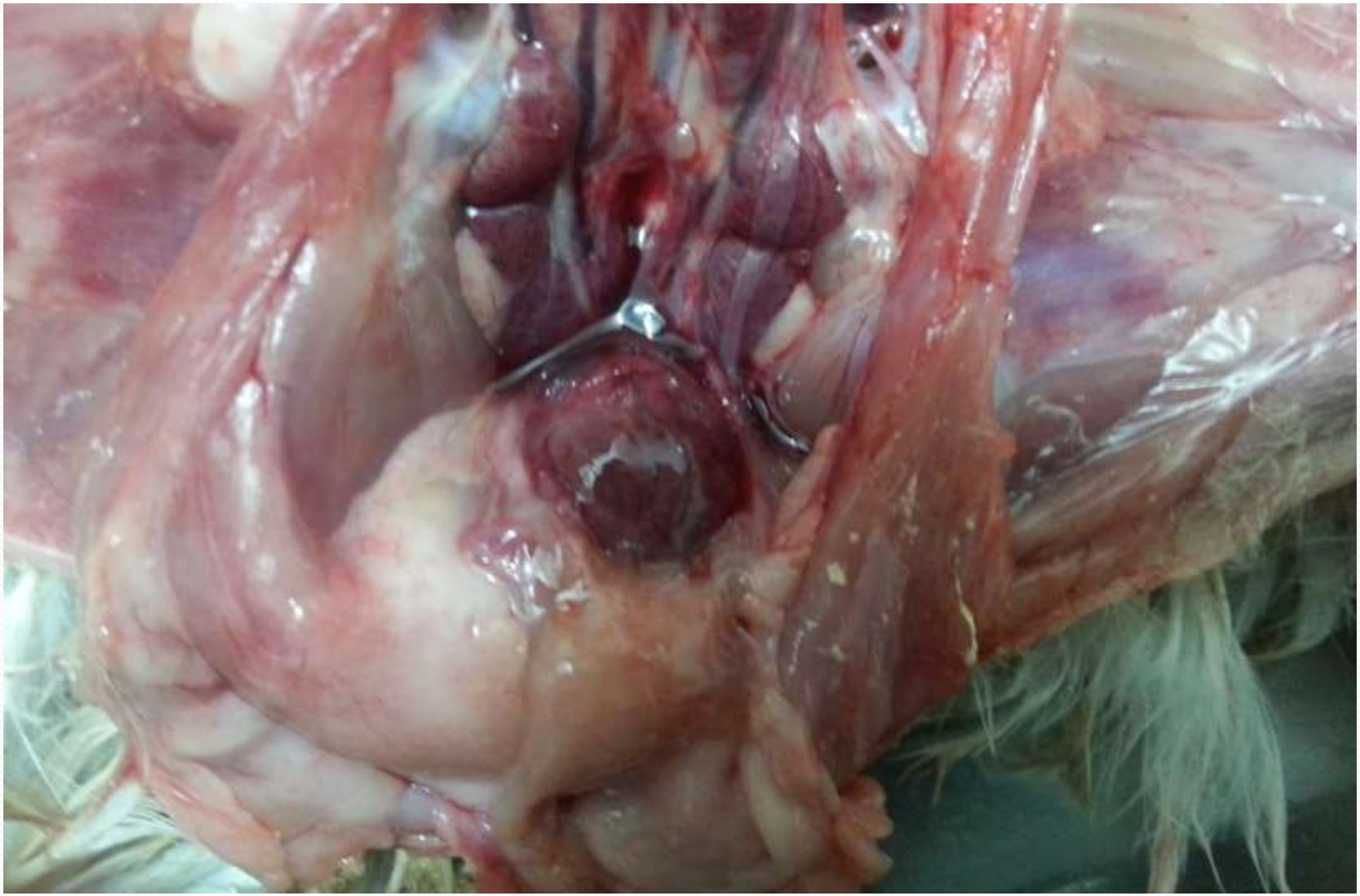


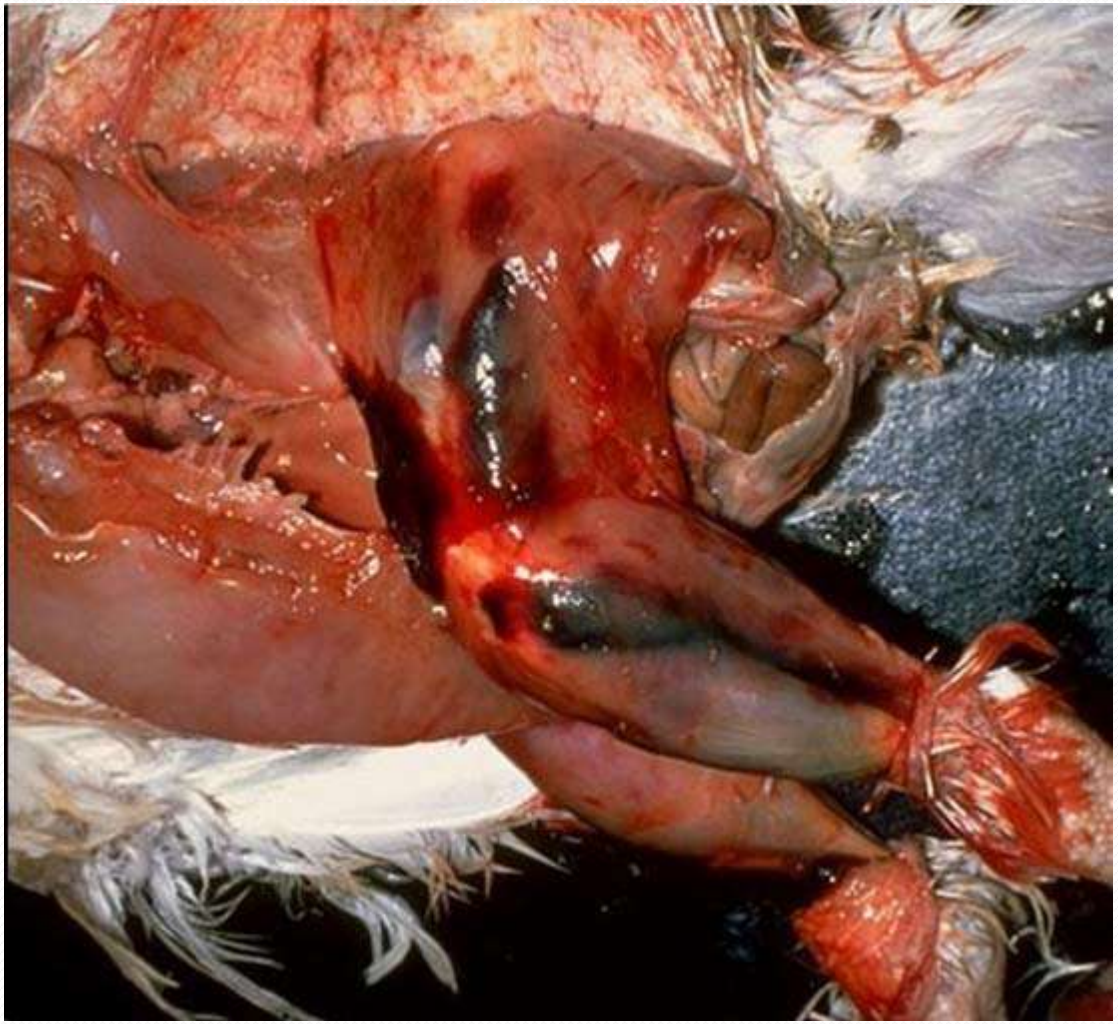


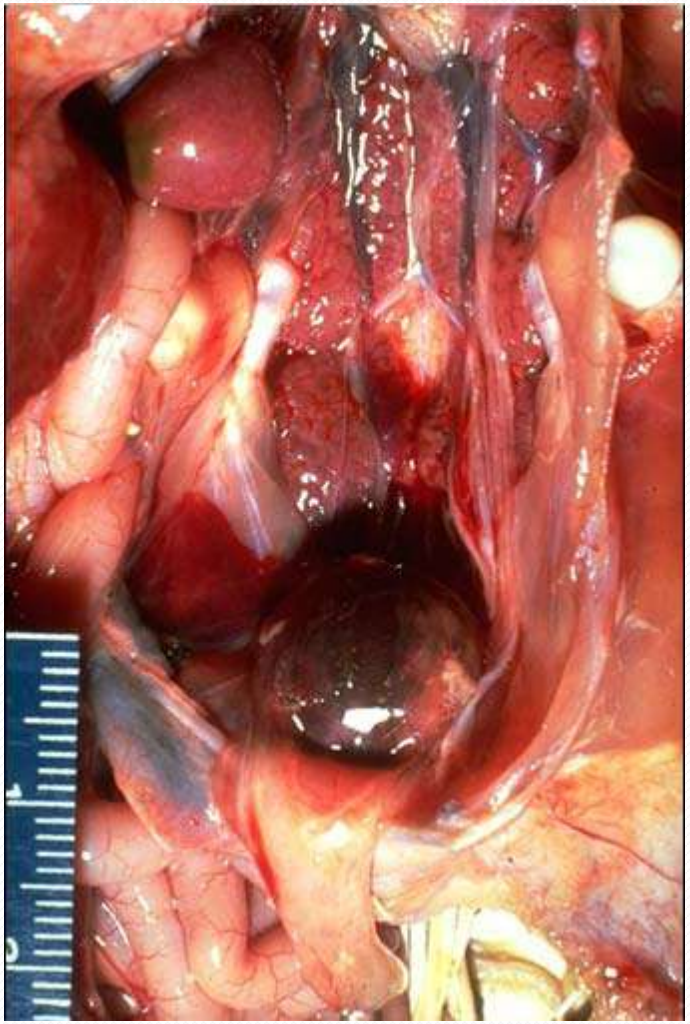


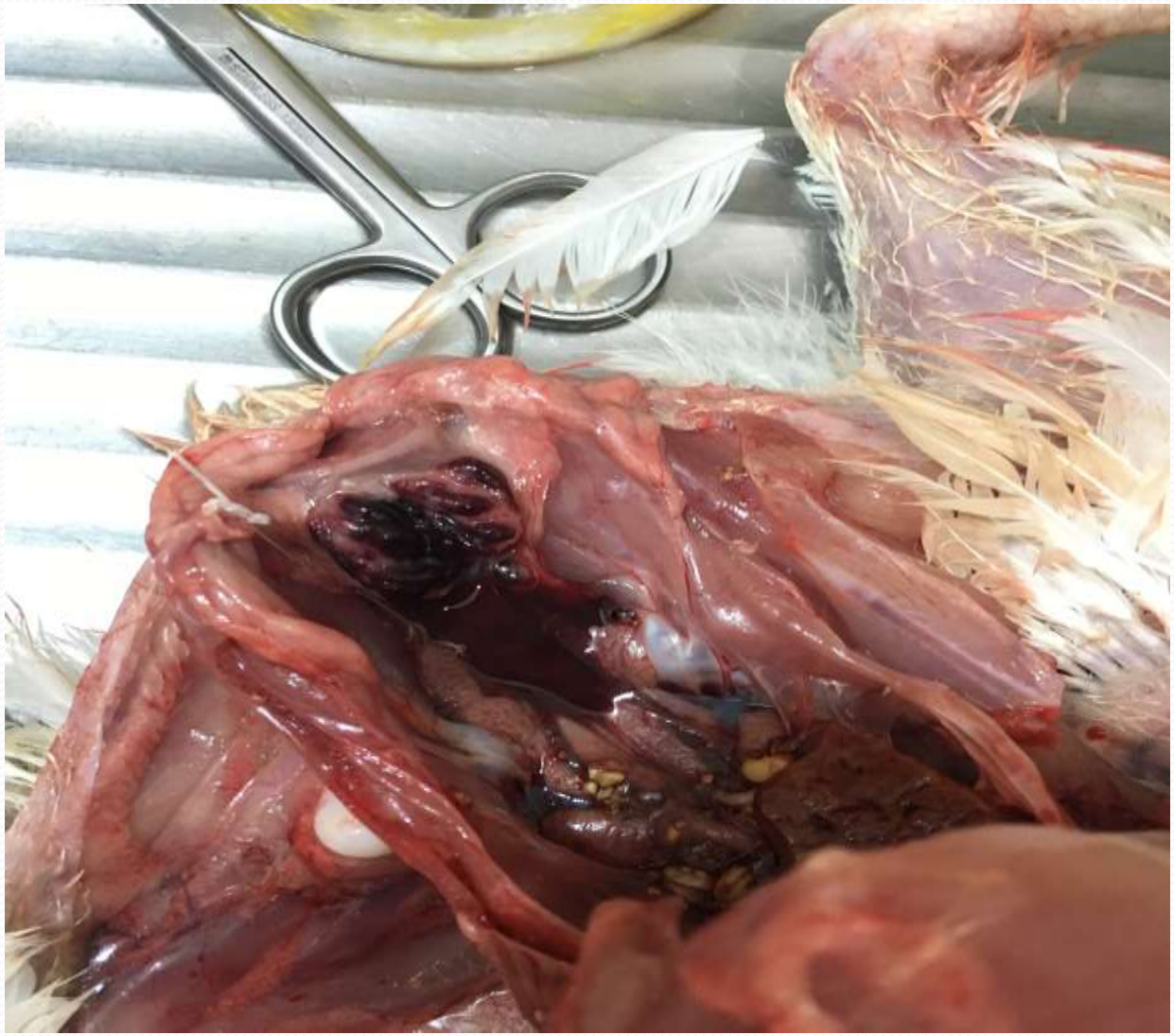
Vent Picking

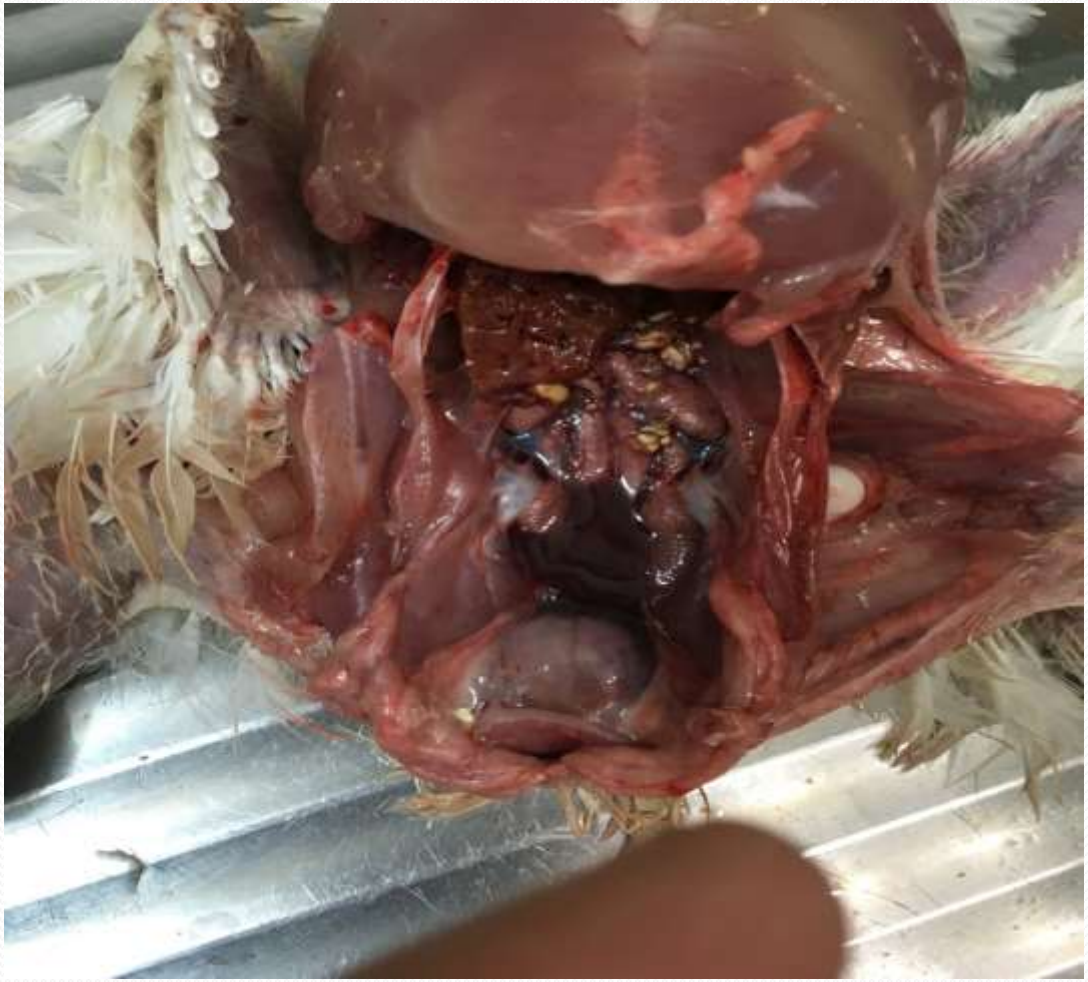


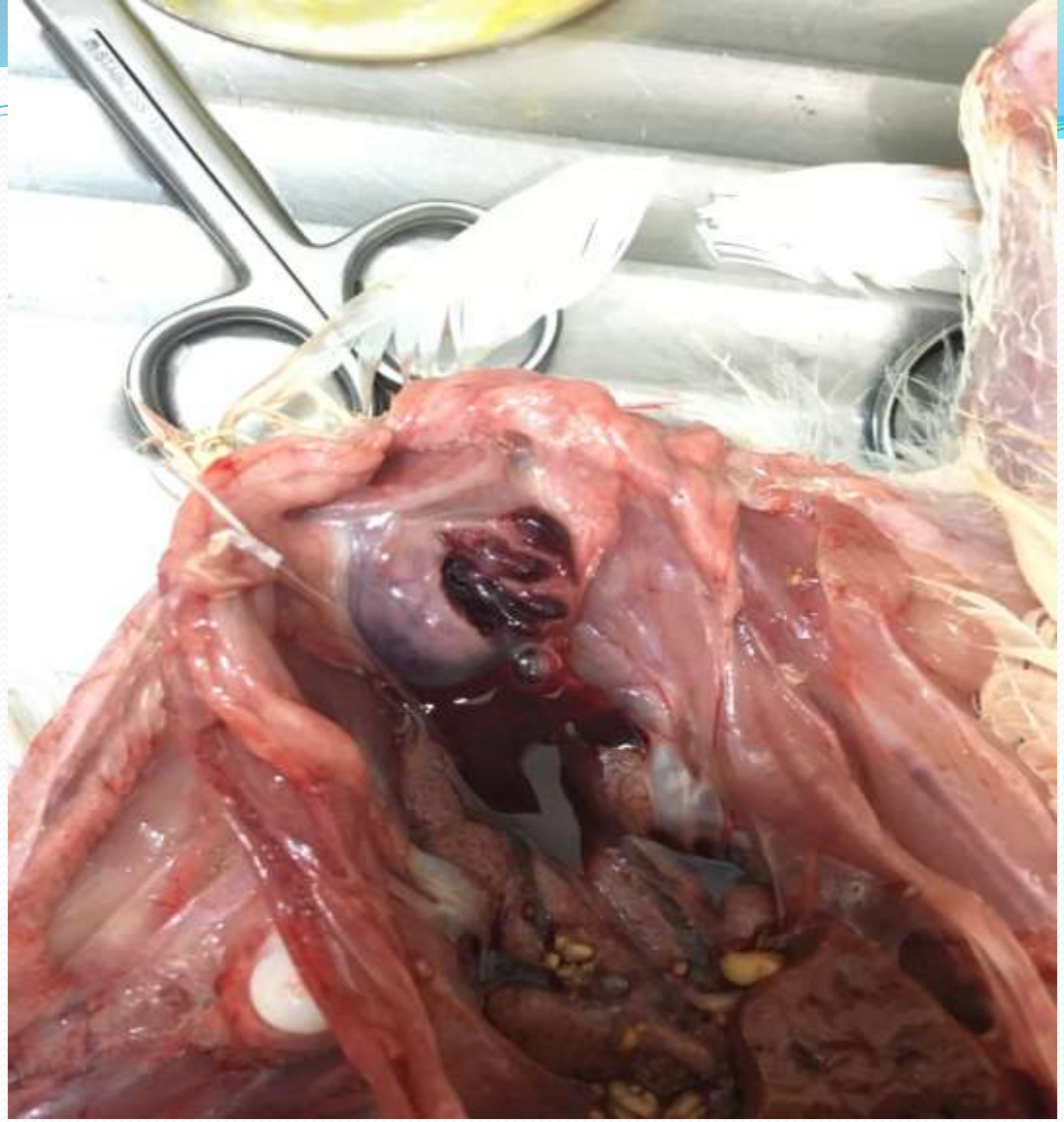












Histopathology

1-Degeneration and necrosis of lymphocytes in the medullary region of the bursal follicles occur as early as 1 day post infection.

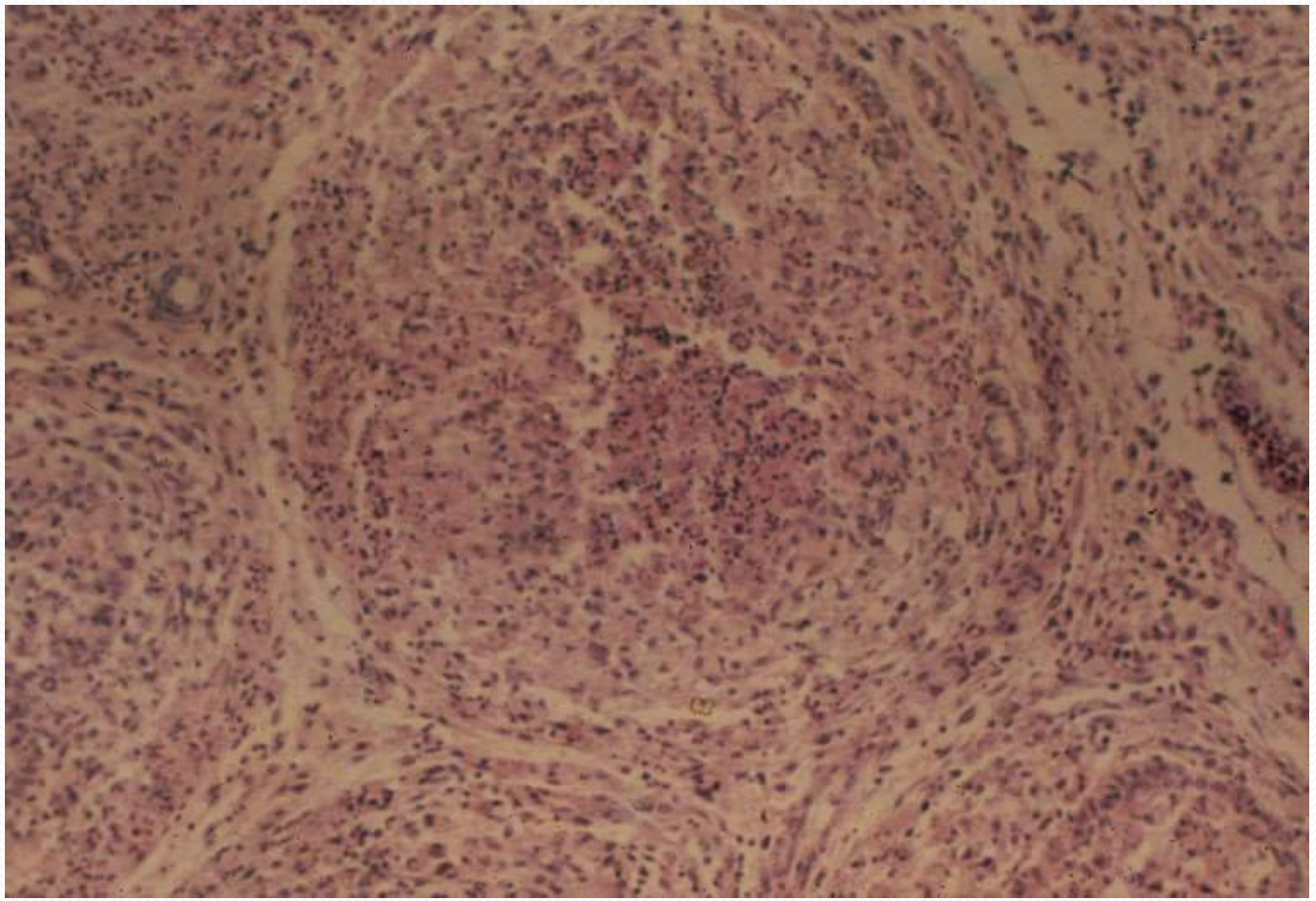
2-Lymphocyte degeneration is a ccompained by by nuclear pyknosis and formation of lipid droplets in the cytoplasm.

2-The increase in bursal weight seen at this time was caused by severe edema, hyperemia, and marked accumulation of heterophils occur by 3 to 4 days post infection.

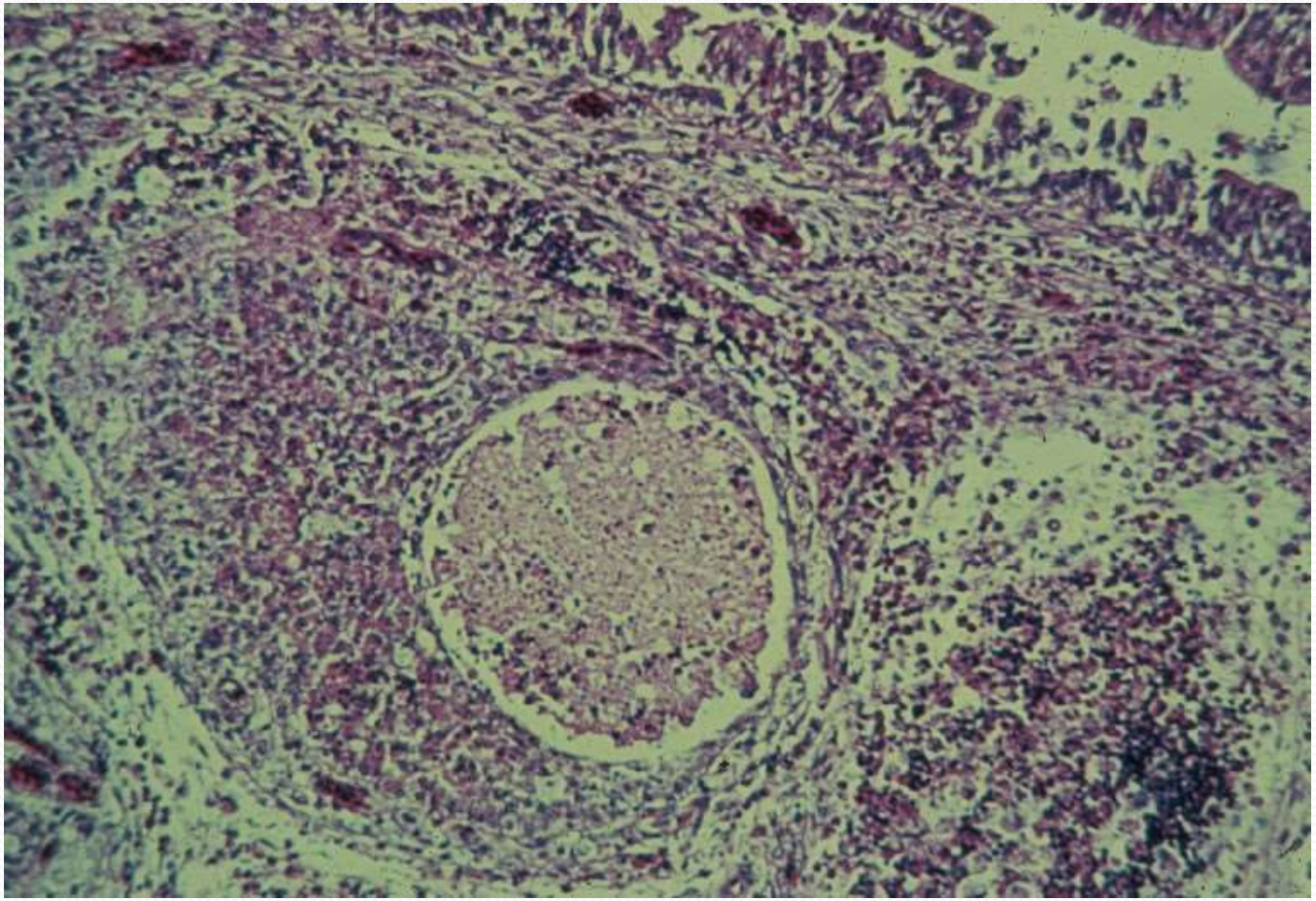
3- As the inflammatory reaction declined, cystic cavities developed in medullary areas of follicles; necrosis and phagocytosis of heterophils and plasma cells occurred; and there was a fibroplasia in interfollicular connective tissue

Normal

Necrosis, loss of lymphocytes



Atrophy



DIAGNOSIS

1-Acute clinical outbreaks, rapid onset, high morbidity of IBD in fully susceptible flocks, and rapid recovery (5—7 days) from clinical signs are characteristics of this disease.

2-Histopathology

3-Viral isolation from infected bursa, cecal tonsils, spleen in in cell culture after propagation of IBDV.

4-Serology – despite the destruction of antibody producing cells, titers to IBD will be high.

5- Identification of the virus by direct immunofluorescent staining of affected organs or direct examination by electron microscopy have proven to be an adjunct to the isolation and identification of IBDV

TREATMENT

No therapeutic or supportive treatment has been found to change the course of IBDV infection . Because of the rapid recovery of the affected flock.

PREVENTION AND CONTROL

Once on farm, the disease tends to reoccur. It cannot be eradicated. it is known that contact with infected birds and contaminated fomites readily causes spread of the infection.

The relative stability of this virus to many physical and chemical agents increases the likelihood that it will be carried over from one flock to a succeeding flock.

The sanitary precautions that are applied to prevent the spread of most poultry infections must be rigorously used in the case of IBD which included restracted all vectors like the lesser mealworm, mosquitos, and rats.

Immunization

1-Immunization of chickens is the principal method used for the control of IBD in chickens

2-Immunization of breeder flocks so as to confer parental immunity to their progeny because maternal antibodies protect the chick from early immunosuppressive for 1-3 weeks.

3-Many choices of live vaccines are available based on virulence and antigenic diversity.

Live Attenuated:

According to virulence, vaccines that are classed as **mild**, **intermediate**, and **hot** forms. The vaccine of intermediate virulence is most commonly used. The most common procedure for vaccinated of broiler chickens is to give the vaccines at 7-14 days old in drinking water or for spray, and when there is a heavy infection its important to revaccinated the flocks after 3 weeks by intermediated strain.

Killed in Oil

Breeders vaccinated at 12-18 weeks of age.

Prevention Cont.

Vaccination, including passive protection via breeders, vaccination of progeny depending on virulence and age of challenge. In most countries breeders are immunised with a live vaccine at 6-8 weeks of age and then re-vaccinated with an oil-based inactivated vaccine at 18 weeks. A strong immunity follows field challenge. Immunity after a live vaccine can be poor if maternal antibody was still high at the time of vaccination.

When outbreaks do occur, biosecurity measures may be helpful in limiting the spread between sites, and tracing of contacts may indicate sites on which a more strong vaccination programme is indicated.