Necrotic enteritis

- Necrotic enteritis is an acute enterotoxemia.
- Necrotic enteritis (NE) is found worldwide
- The clinical illness is usually very short and often the only signs are a sudden increase in mortality.
- The disease primarily affects **broiler** chickens (2–5 wk old) and turkeys (7–12 wk old) raised on **litter** but can also affect commercial layer ***pullets*** raised in **cages**.

Etiology

- The causative agent is the gram-positive, obligate, **anaerobic** bacteria *Clostridium perfringens*.
- There are **2** primary C. perfringens types, **A and C**, associated with necrotic enteritis in poultry.
- Toxins produced by the bacteria cause damage to the small intestine, liver lesions, and mortality.
- *C. perfringens* is often found in the intestinal tract of healthy birds it can cause outbreaks of disease.

- Clostridiosis occurs in **two forms**
  - **acute clinical disease**, necrotic enteritis (NE), in 2−4-week-old chickens, causing high mortality
  - **subclinical disease** with focal necrosis in the intestines or as C. perfringens -associated hepatitic change with cholangiohepatitis or fibrinoid necrosis in the liver.

Transmission

- C perfringens is ubiquitous ,found in soil, dust, feces, feed, and used poultry litter→ feco-oral
- It is also a **normal inhabitant** of the intestines of healthy chickens and turkeys.

- The enterotoxemia that results in clinical disease most often occurs either from
  - **alteration in the intestinal microflora**
  - condition that results in **damage to the intestinal mucosa** (eg, coccidiosis, mycotoxicosis, salmonellosis, ascarid larvae, Gumboro).

ϖ High dietary levels of animal by products (e.g, fish meal), wheat, barley, oats, or rye predispose birds to the disease.

ϖ Anything that promotes excessive bacterial growth and toxin production or slows feed passage rate in the small intestine could promote the occurrence of

Grain Source and Quality

- **Wheat, Barley, Rye** → Increased Intestinal Irritation/Decreased Digestibility/More Nutrients for the Clostridia Organism /Increased Mucous Production → Favors CP
- **Higher the Protein** –the easier to induce NE outbreak
  - High Levels of Animal By-Products (Fishmeal, Meat & Bone Meals)
- The alterations are particularly in the **jejunum and the ileum** because of their **higher pH** and the **lower oxygen** content in these areas

Clinical Findings (Symptoms)

- Rapid and unexplained death of the bird.
- severe depression, decreased appetite, **dark colored diarrhea**, closed eyes or ruffled feathers appear they are often short-lived because birds die

Lesions

- ♣ jejunum may be ballooned/distended, friable, and contain a foul-smelling, brown fluid (brownish watery content, mixed with gas bubbles).
- ♣ The mucosa is covered with a tan to yellow (bile stained) pseudomembrane often referred to as a "**Turkish towel**" in appearance.

Two types of liver lesions have been described

- ϖ An inflammation affecting the bile tree (cholangiohepatitis) is the most common lesion type.
- ϖ Intrahepatic parts of the bile tree are most frequently affected, but gall bladder and extrahepatic bile duct changes may also be found.
- ϖ Histologically, fibrinoid necroses with or without an inflammatory response.
- ϖ Focal, subcapsular nodules (including fibrinoid necroses)

Diagnosis

♣ The presence of birds with cholangiohepatitis among bird from such flocks is another helpful indicator.

- Histological and microbiological examinations may be required for a definitive diagnosis.
- Necrotic enteritis must be differentiated from lesions produced by **Coccidia** (Eimeria) and also from **ulcerative enteritis**.
- Uncomplicated coccidiosis rarely produces lesions as acute or severe as those seen with necrotic enteritis.
- Ulcerative enteritis caused by *C colinum* usually produces ***focal lesions*** from the distal portion of the small intestine (ileum) to the ceca and is almost always

Prevention, Control, and Treatment

- Prevent coccidiosis, especially *Eimeria acervulina* and *E maxima.*
- Treatment for necrotic enteritis is most commonly administered in the drinking water, with
  - bacitracin (200–400 mg/gal. for 5–7 days)
  - penicillin (1,500,000 u/gal. for 5 days)
  - lincomycin (64 mg/gal. for 7 days) most often used.
- Avoiding drastic changes in feed and minimizing the level of fishmeal, wheat, barley, or rye in.
- In each case, the medicated drinking water should be the sole source of water.
- Moribund birds should be removed on time, as they can serve as a source of toxicosis or infection due to

¬ When higher amounts of wheat, barley, or rye are necessary, use of enzymes in the feed (improve digestibility)

Manage by:

ϖ Feeding lower protein and higher (synthetic) amino acid

ϖ Feed all vegetable diets to flocks at risk

ϖ Litter Type and Condition- *Increased Litter Moisture/Increased Cocci Challenge/Increased Bacteria Growth*

Vaccine

- Oil emulsion for injection & Contains *Clostridium perfringens* type A alpha toxoid (>6.8 IU HIA)
  - 0.5 ml administered by intramuscular injection into the breast
  - First dose to be given at 10 to 14 weeks of age

Gangrenous dermatitis (malignant edema)

- **It** occurs worldwide in chickens and turkeys.
- The syndrome is precipitated by various detrimental microbial, nutritional and environmental factors.

Characterized by:

- a sudden onset, sharp increase in mortality
- gangrenous necrosis of the skin over the wings, thighs, breast, and head.

Cause: It is associated with

♣ *Clostridium septicum* , *Clostridium perfringens* type A, and *Staphylococcusaureus* , either singly or in combination are most often involved.

- Immuno-suppressed by infectious bursal disease or chick anemia virus are predisposed.
- Skin lesions due to trauma, wet litter, picking, or treading wounds may provide entry sites for causative

Clinical signs

- sudden dramatic increase in mortality in the affected flock(10-60%)
- depressed, lethargic, and die within 8-24 hr.

♣ Red to black patches of moist, gangrenous skin are seen over the breast, abdomen, wing tips, or thighs.

♣ Feather loss or sloughing of the epidermis is common.

♣ Palpation of the affected areas often reveals crepitation due to gas bubbles in the subcutis and musculature.

Gross lesion

- Accumulation of gaseous, serosanguineous fluid in the subcutis, and the musculature has a pale cooked appearance.
- The liver and spleen are enlarged

Histopathology

- Gangrenous necrosis with numerous coccoid bacteria or large, gram-positive rods with or without spores in affected tissues is sufficient to confirm a clinical diagnosis.

Diagnosis

- Isolation of the etiologic agent, together with the history and clinical findings.

Differential diagnosis

- Exudative diathesis.
- Squamous cell carcinoma of the skin of chickens

Prevention

- Maintaining proper litter condition
- minimizing mechanical injury
- controlling cannibalism
- Vaccinate breeders against infectious bursal disease.

Avian mycoplasmosis

Characteristics of Mycoplasmas:

- Quite host specific
- Some infect only a single species of animal, and others may have the ability to infect several different animal species.
- Smallest free living organisms
- Lack a cell wall
- Non-hardy outside the host
- Resistant to penicillin act on cell wall /resistance to antibiotics that affects cell wall synthesis and complex nutritional requirements.

Etiology

- *M. gallisepticum* In chicks and turkey
- *M.synoviae*

Mycoplasma meleagridis in turkeys only

Disease occurs in two forms

- Respiratory mycoplasmoa is (Chronic respiratory disease i.e. CRD
- Infectious synovitis.

Respiratory mycoplasmosis (chronic reparatory disease) (CRD)

ϖ *M. gallisepticum* infections are commonly known as chronic respiratory disease in chickens and infectious sinusitis in turkeys.

ϖ These diseases affect poultry worldwide and may be more severe during cooler months, causing the most significant economic losses in large commercial operations.

Etiology

- Caused by *Mycoplasma gallisepticum*.
- The organism is coccoid and 0.25 -0.5 microns in diameter.
- Mycoplasmas have no cell wall.

Infection mostly is complicated by

- *E.coli*(pathogenic strains)
- New Castle Disease virus
- Infectious bronchitis virus
- *Haemophilus gallinarum*

Transmission & Epidemiology

- **Egg transmission** (transovarian) is a major source of infection.
  - The transmission rate then lessens and is inconsistent (0–5%) until the end of production.
- Transmission may be **direct contact** with birds

- Spread is slow between houses and pens suggesting that aerosols are not normally a major route of transmission.
- Fomites appear to a significant factor in transmission between farms.
- ***Recovered birds remain infected for life***; subsequent stress may cause recurrence of disease.
- Intercurrent infection with respiratory viruses (IB, ND), virulent *E. coli*, *Pasteurella* spp. *Haemophilus*, and inadequate environmental conditions are predisposing factors.

Clinical Findings

ϖ Respiratory distress, slight to marked **rales, coughing, sneezing.**

ϖ Nasal discharge.

ϖ Swelling of the infraorbital sinuses.

Lesion

- ¬ Uncomplicated infections **mild** catarrhal sinusitis, tracheitis, and **airsacculiti**s.
- ¬ E. coli infections are often coexisting and result in ***severe air sac thickening and turbidity, with exudative accumulations, fibrinopurulent pericarditis, and perihepatitis, particularly in broilers***.
- mucopurulent sinusitis and varying degrees of tracheitis and airsacculitis.
- The mucous membranes are ***thickened, hyperplastic, necrotic, and infiltrated with inflammatory cells***.
- Lymphofollicular areas are found in the submucosa.

Mycoplasmosis

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68 *M. gallisepticum*. Swelling of the infra-orbital sinuses in a turkey.

69 *M. gallisepticum*. Infra-orbital sinus of a turkey opened to show sticky exudate in an acute case.

Microscopic lesions

- Lymphofollicular reaction is a marked feature in the respiratory tract.
- Pneumonia, myocarditis, sinusitis, hepatitis, etc.

Diagnosis

- Serology by agglutination and ELISA methods are commonly used for surveillance.
- *M.gallisepticum* should be confirmed by isolation and identification or by PCR in samples from infraorbital sinuses, nasal turbinates, choanal cleft, trachea, air sacs, lungs, or conjunctiva.

Differential diagnosis

- E.coli, ND, influenza, and other respiratory diseases (eg, IB in chickens)

Treatment and Control

- Most strains of *M gallisepticum* are sensitive to a number of antibiotics, including macrolides, tetracyclines, fluoroquinolones, and others, ***but not penicillins or those that act on the cell wall***.
- Tylosin or tetracyclines have been commonly used to reduce egg transmission or as prophylactic treatment to prevent respiratory disease in broilers or turkeys.
- Antibiotics may alleviate the clinical signs and lesions but do not eliminate infection.
- In valuable breeding stock, treatment of eggs with antibiotics or heat has been used to eliminate egg transmission to progeny.

- ♣ **Vaccines** are available in most countries; these help prevent egg production losses but not infection.
- Inactivated, oil-emulsion **vaccines** are available in most countries; these help prevent egg production losses but not infection.

Prevention

- Depopulation of infected premises and cleaning and disinfection should precede the introduction of a "clean" flock.
- All in all out
- Test foundation breeders and eliminate reactors
- Quarantine measures must be strictly enforced and good management and sanitation must be practiced

Mycoplasma synoviae (**infectious synovitis**)

- A mild respiratory disease results in inflammation of the synovial sheaths.
- This disease is known as infectious synovitis and occurs in both chickens and turkeys.
- It is less virulent but spreads faster than MG.
- It is common in commercial laying flocks.

Etiology: *Mycoplasma synoviae*

ϖ Most frequently cause a subclinical URT infection

ϖ It may cause air sac infection when combined with ND, IB or other

Clinical signs

- ¬ Pale comb, lameness and retarded growth
- ¬ ruffled feather and shrunken comb
- ¬ Swelling around joints/ Hock joint and foot pads are principally involved/Lameness is the most prominent sign in turkeys
- ¬ Brest blisters are common
- ¬ Normal appetite
- ¬ Greenish droppings with large amount of uric acids
- ¬ Slight rales and air saculitis if respiratory tract is involved

Transmission

- **Lateral transmission occurs readily by direct** contact
  - Entry via the respiratory tract

Lesions

- **Viscous creamy to gray exudates (even caseous)** in the joints (hock, wing, keel bursa)
- Hepatosplenomegaly, Kidneys are usually swollen, mottled and pale
- Air

73 *M. synoviae*. Two swollen footpads (centre and left) of broilers. This feature may be very pronounced. There is frequently swelling of the hock and main wing joints. As in other mycoplasma infections, egg transmission occurs and the diagnosis should be carefully confirmed, both serologically and culturally.

74 *M. synoviae*. Footpad exudate in an acute infection of a commercial layer. The exudate is characteristically glairy and tenacious.

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Diagnosis

1. Suggestive -Typical signs and gross lesions with a positive plate agglutination test on sera from a few birds in the flock.

2. Confirmation -Isolation and identification of the bacteria by ELISA /PCR

- The isolated Mycoplasma can be identified by direct immunofluorescent antibody techniques.

Differential diagnosis

- CRD/, s.*aureus, E.coli, pasteurella and salmonella can* cause synovitis

¬ **Prevention & treatment similar to CRD**

Avian Tuberculosis

- ♣ Avian tuberculosis occurs throughout the world in many avian, mammalian species and domestic poultry.
- ♣ It is generally seen in mature stock kept in conditions of poor management.
- ♣ Tuberculosis is a slowly spreading, chronic, granulomatous bacterial infection, characterized by gradual weight loss.
- All birds appear to be susceptible, although to variable degrees
- pheasants seem to be highly susceptible, while the disease is uncommon in turkeys.

Etiology

- Mycobacterium avium complex (MAC) - **Mycobacterium avium subspecies avium** , (often simply called *M. avium*), principle cause of avian tuberculosis in birds.
- Other species of mycobacteria may be involved such as the closely related *M intracellulare*, or *M. genavense*

Transmission

¬ The main source of infection is infected birds as they shed large amounts of organism into the environment.

¬ The bacilli are exuded from ulcerated lesions of the intestine and are voided in droppings.

¬ The most common route of infection is the

- Persistence within flocks is associated with keeping **older stocks**
- Maintaining birds closely confined under stressful conditions favores spread of the disease.

- The organism persist in the environment for ***many years***, especially in soil and litter favor the disease transmission.

- Litter, pens, equipment, and pasture contaminated with excreta and the hands, feet, and clothing of attendants play an important role in disease transmission.

Clinical signs

- The disease may follow a slow course (without signs).

¬ In advanced cases, birds may develop symptoms like progressive **weight loss**, ***depression***, ***white diarrhea*** with soiled feathers, increased ***thirst***, respiratory distress, ***fatigue***, and decreased egg production

¬ Feathers are often dull or ruffled and comb, wattle, and earlobes often appear pale, thinner and dry.

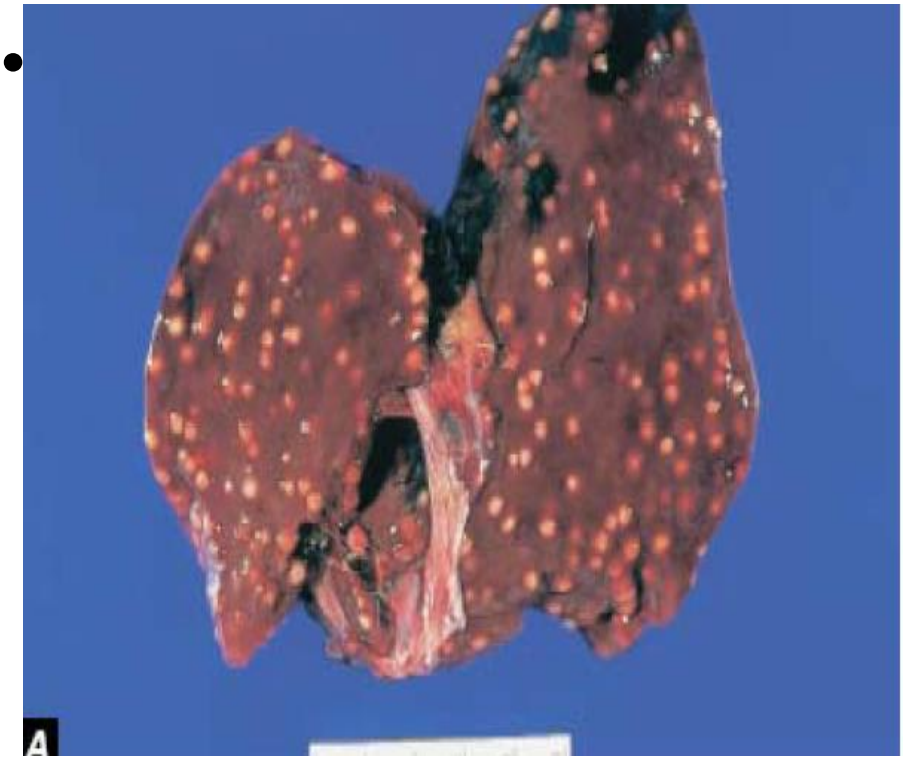
¬ Birds eventually become lethargic and emaciated with marked ***atrophy of breast muscles*** manifested as

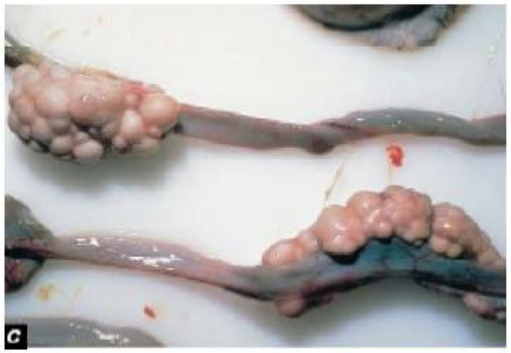
- A **jerky hopping gait** with unilateral **lameness** (presence of tubercular lesions in bone marrow of the leg bones or joints).

- Birds may adapt a sitting position & Tuberculoses arthritis can even lead to paralysis.

- Fatal results often occur due to massive

Lesions

- Irregular **grayish white or grayish yellow nodules of varying size along the** intestine, on the liver, spleen and bone marrow
- hypertrophy of liver and spleen (rarely)
- 



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52 A pale granuloma within the marrow cavity of a femur. Lameness often occurs in affected birds due to the development of such lesions, particularly at the distal end of the femur.

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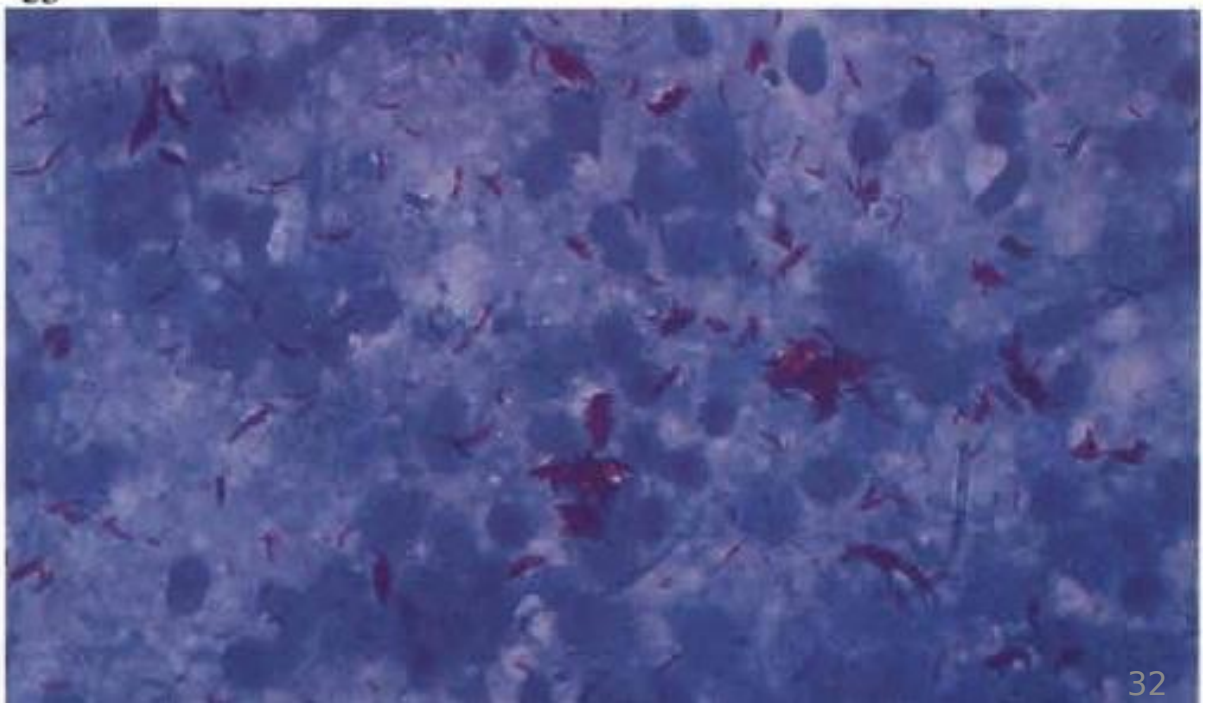

53 Large numbers of acid fast bacilli are present in smears from most avian lesions. Smears are best made by crushing individual nodules between two glass slides. Ziehl-Neelson.

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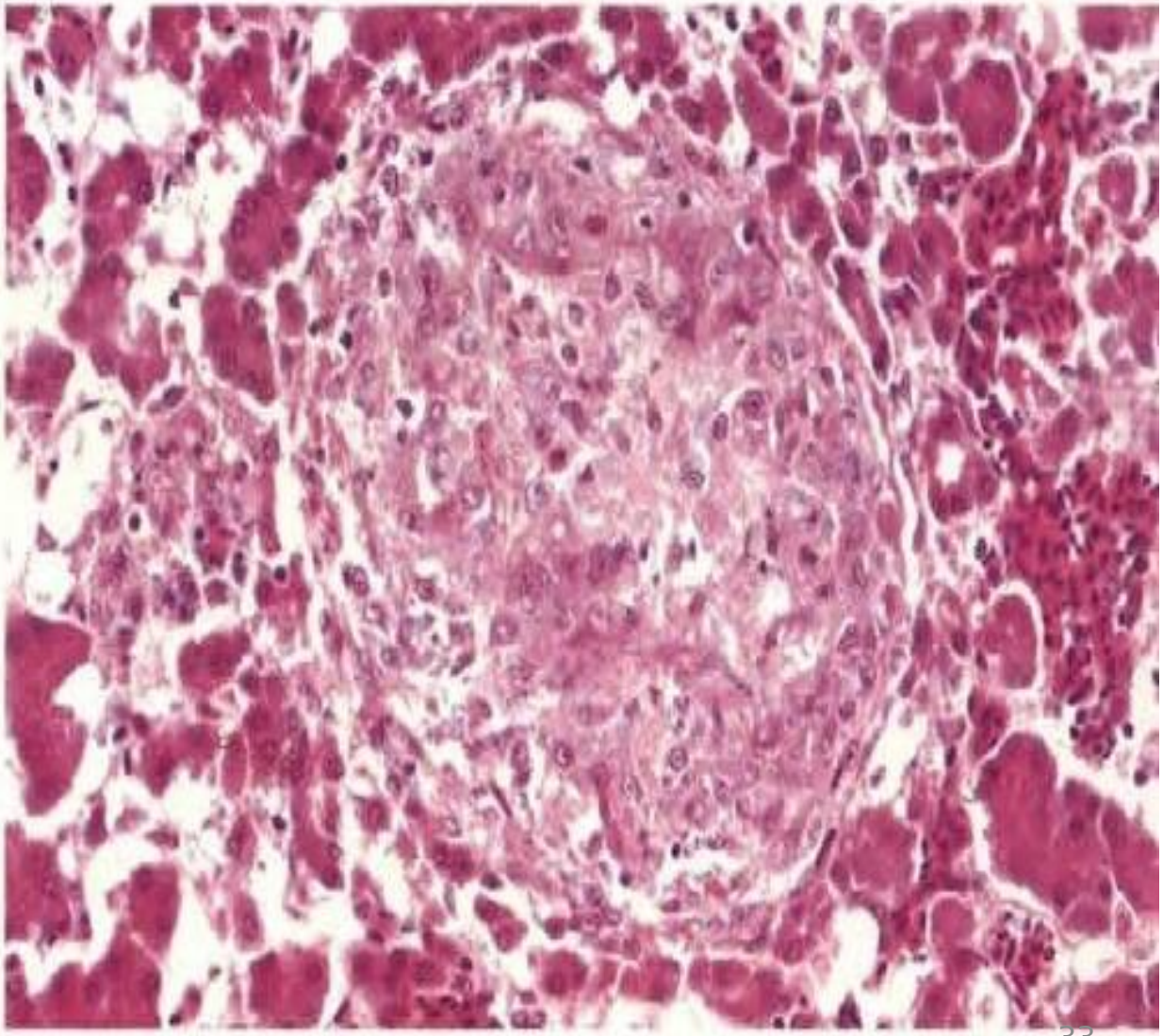

54 Developing tubercle in liver of hen. At this stage the central part of the lesion is mainly composed of pale-staining epithelioid cells.

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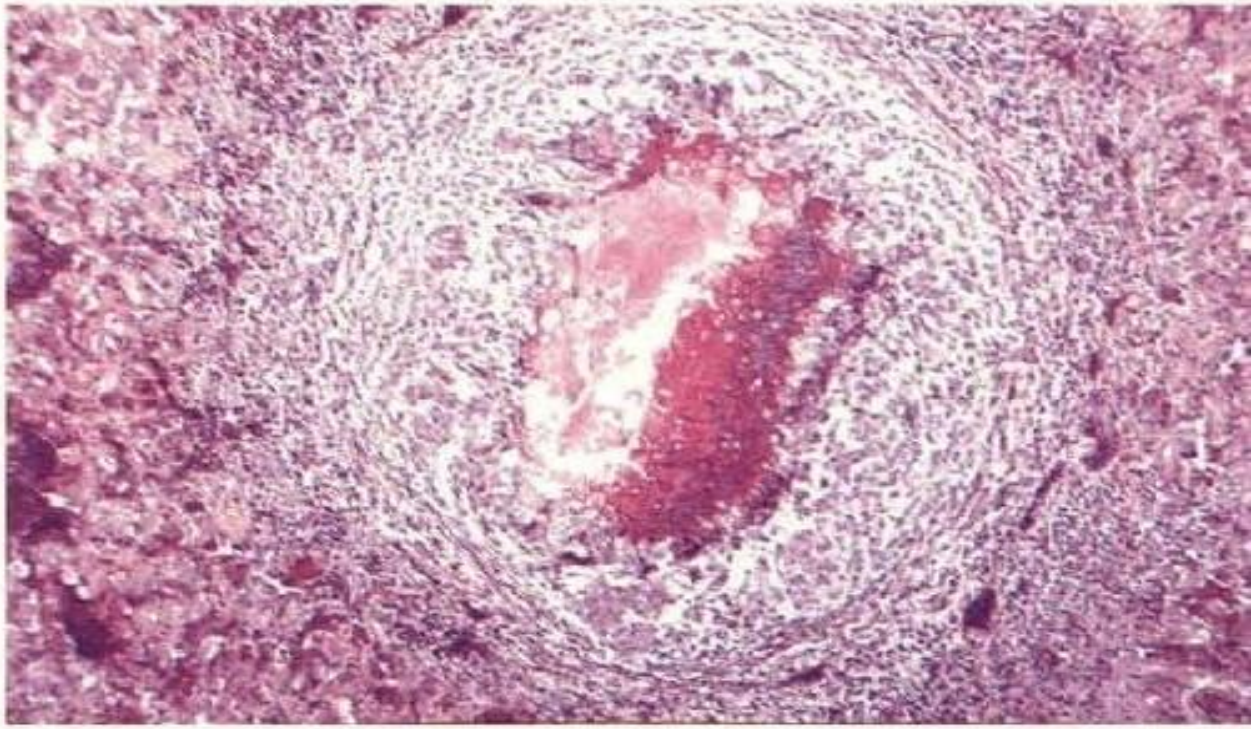

55 A more advanced lesion in which central necrosis has taken place. Giant cells are starting to pallisade round the necrotic zone. Numerous macrophages are present peripherally. Connective tissue encapsulation has not yet occurred.

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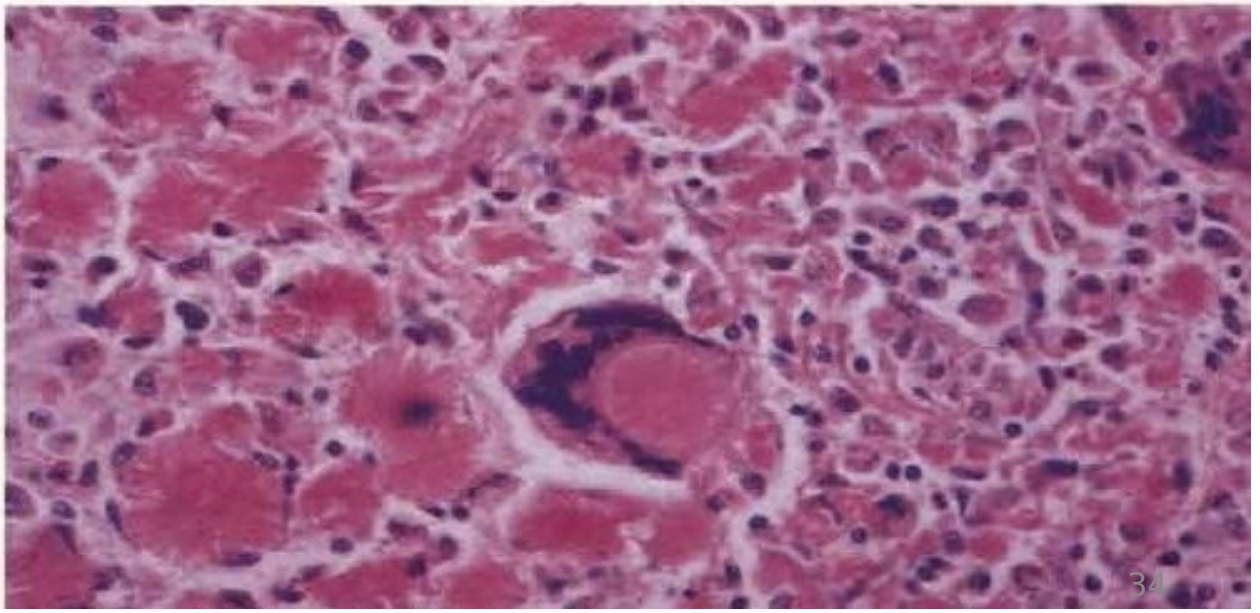

56 A large mass of granulation tissue was present at the thoracic inlet of a commercial layer. Many giant cells were scattered through the lesion in which there was considerable deposition of amyloid. A giant cell is seen engulfing an amyloid focus.

Differential diagnosis

- Aspergillosis, salmonellosis
- fowl cholera, colibacillosis
- In neoplasm, the cut surface of the lesion is uniform and without necrosis.

Public health significance

- Mycobacterium avium may cause disease in man thus care must be taken.

Diagnosis

- Lesions and signs
- Ziehl-Neelsen staining, of what??
- Isolation and identification, media?
- Tuberculin test, where and how??
- Serology like rapid agglutination test and ELISA

Treatment, prevention and control

- Combination of ethambutol, rifampcin and isoniazid ➔ but remission was observed
- Prevention and control
  - **Test and remove** (tuberculin) – early and good to reduce bacterial dissemination
  - **Abandon old equipment**
  - **Proper fencing/ biosecurity**
  - **Eliminate old flock, burn carcasses** of dead birds (with lesion)
  - **Eliminate from swine herds** all reactors to avian and mammalian tubercle