

CHAPTER TWO

Major bacterial diseases of poultry

SALMONELLA INFECTION IN POULTRY

⌘ Infections with bacteria of the genus *Salmonella* are responsible for a acute and chronic diseases.

⌘ **Intestinal microfloras** are an important factor influencing infection and disease by salmonellae in poultry

⌘ *Salmonella pullorum* - Pullorum disease

⌘ *Salmonella gallinarum* - Fowl typhoid

⌘ *Salmonella typhimurium* - Paratyphoid infections (Salmonellosis)

⌘ **public health significance** : consumption of contaminated poultry product

⌘ *S.pullorum* and *S.gallinarum* are highly host-adapted to chickens and turkeys.

⌘ *S.arizonae* is most important in turkeys, with chickens

Pullorum disease

- ✚ It is usually symptomatic only in young birds
- ✚ Characterized by white diarrhea, high mortality in young birds.
- ✚ It is infectious, egg transmitted disease of poultry, especially chicks and turkey poults.

Susceptible species

- ✚ Chicken and turkeys, *S. pullorum* is a specific bacteria, but it can affect other bird species.

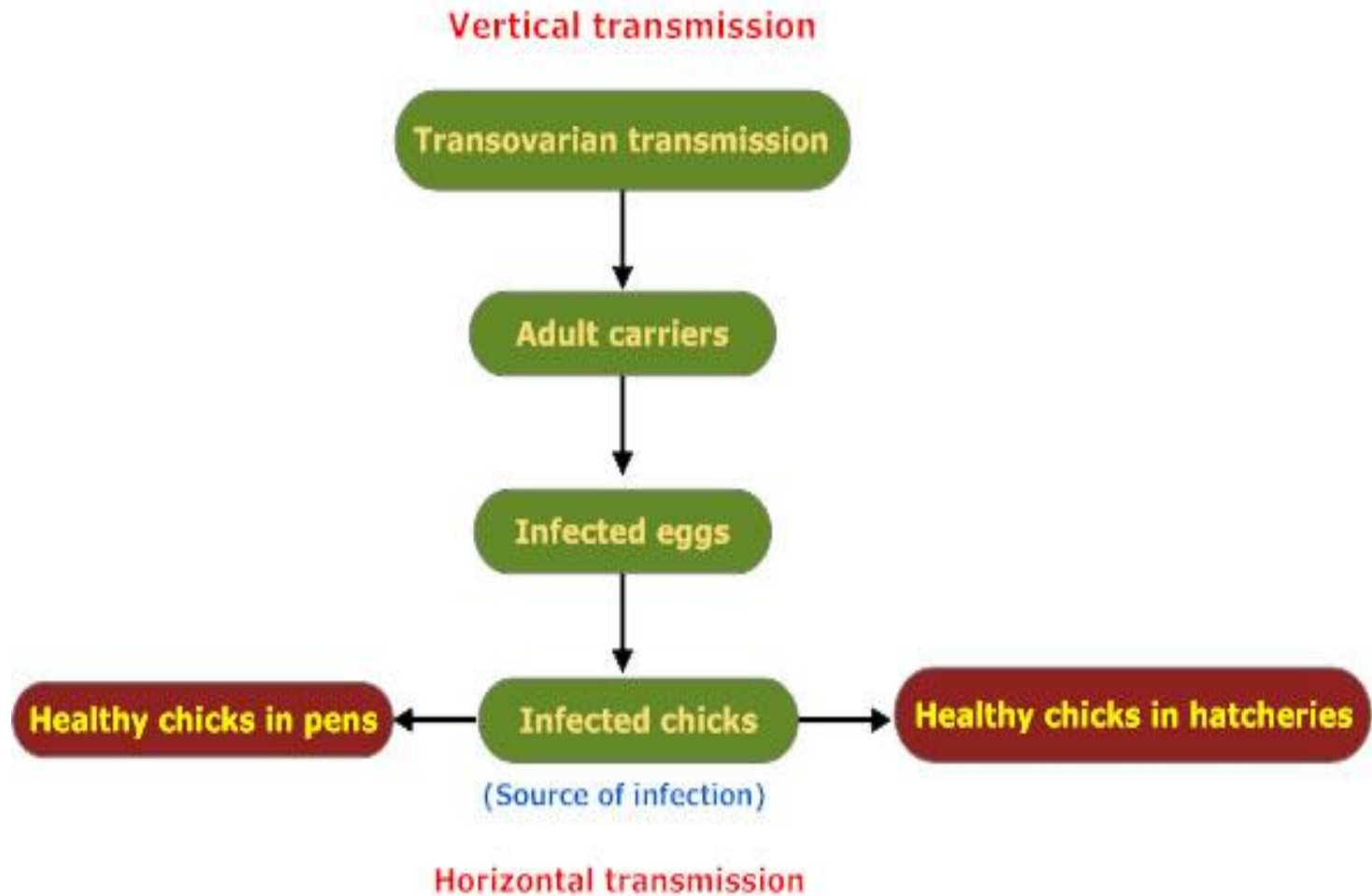
Etiology: Salmonella pullorum.

- Young chicks and turkey poults commonly affected
- High mortality (up to 100%); adults - asymptomatic carriers and not significant in other birds.

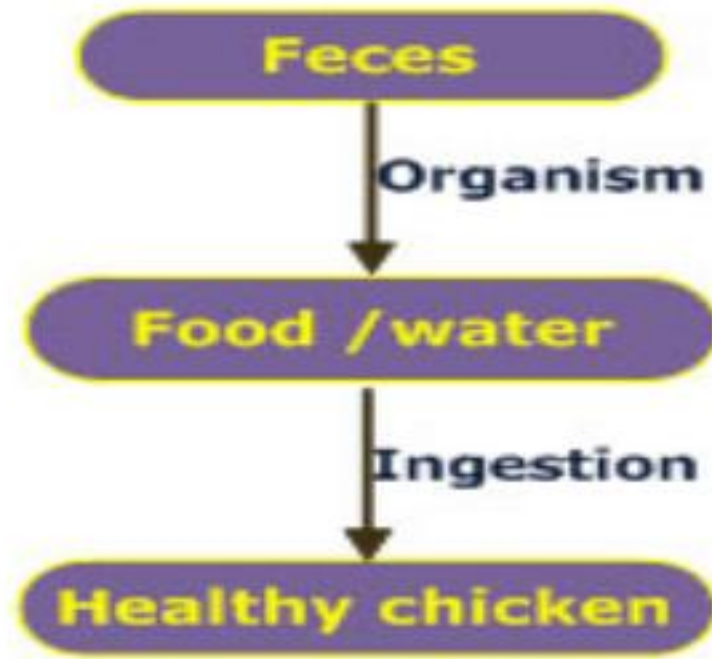
Occurrence and Economic Significance

- Pullorum disease (or “bacillary white diarrhea”, BWD) is potentially world-wide in distribution
- Infection results in high mortality in young chicks

Types of transmission



Transmission through fecal contamination



- Wild birds
 - Mammals
 - insects can act as mechanical or biological vectors.
- Transmission between farms is due to **poor biosecurity**

Clinical signs

Excessive numbers of **dead-in-shell chicks & death soon after hatching**

Chicks:

- Depression with a tendency to huddle
- respiratory distress
- lack of appetite
- **Copious white diarrhea and accumulation of fecal material adherent to the plumage surrounding the vent.**
- Mortality varies considerably, and 100% in severe cases

Growers - Sub acute form:

⌘ Lameness and swollen hock joint Poor growth rate.

In older birds:

♣ pale & shrunken comb, Listless (depressed, dull).

Layers - Reduced egg production

♣ stunting, poor feathering and frequently lameness due to arthritis. **Paleness, depression, Emaciation**

Paleness, depression, Emaciation



Post Mortem Lesion

Chicks:

→ Peritonitis with an inflamed, **unabsorbed yolk sac**

Lungs: congested; pyro-granulomatous pneumonia due to pullorum disease in a chick.

Liver:

- ♣ Dark, enlarged, mottled with multiple **miliary** necroses
- ♣ haemorrhages on the surfaces
- ♣ nodules, necrosis necrogranulomatous hepatitis multiple nodules in heart.

Growers:

- Arthritis - Hock joint - Enlarged due to the presence of excess orange colored gelatinous material around the joints
- swollen hock joint containing yellow viscous fluid.

Adults:

- ♣ **Abnormal ovary:** Ovary with multiple misshapen grey nodular follicles.
- ♣ follicles are deformed & thick pendulating masses.
- ♣ Peritonitis, arthritis and nodular lesions in the heart , yellowish pericardium
- ♣ fibrinous to granulomatous pericarditis.

Layer:

- the ovary shows **marked degeneration**



Liver: Nodules, necrosis
Necrogranulomatous hepatitis

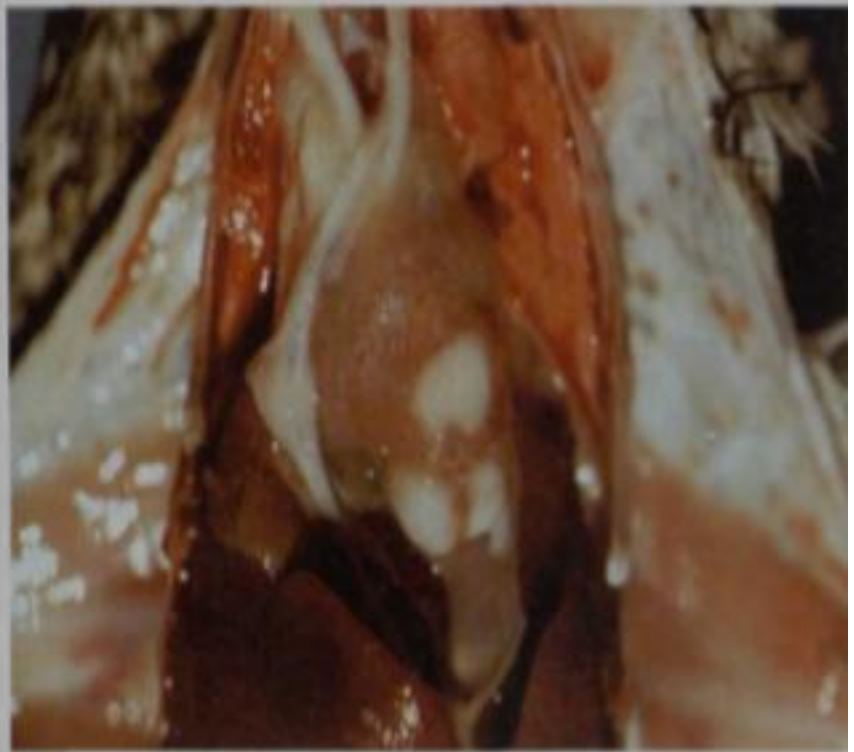
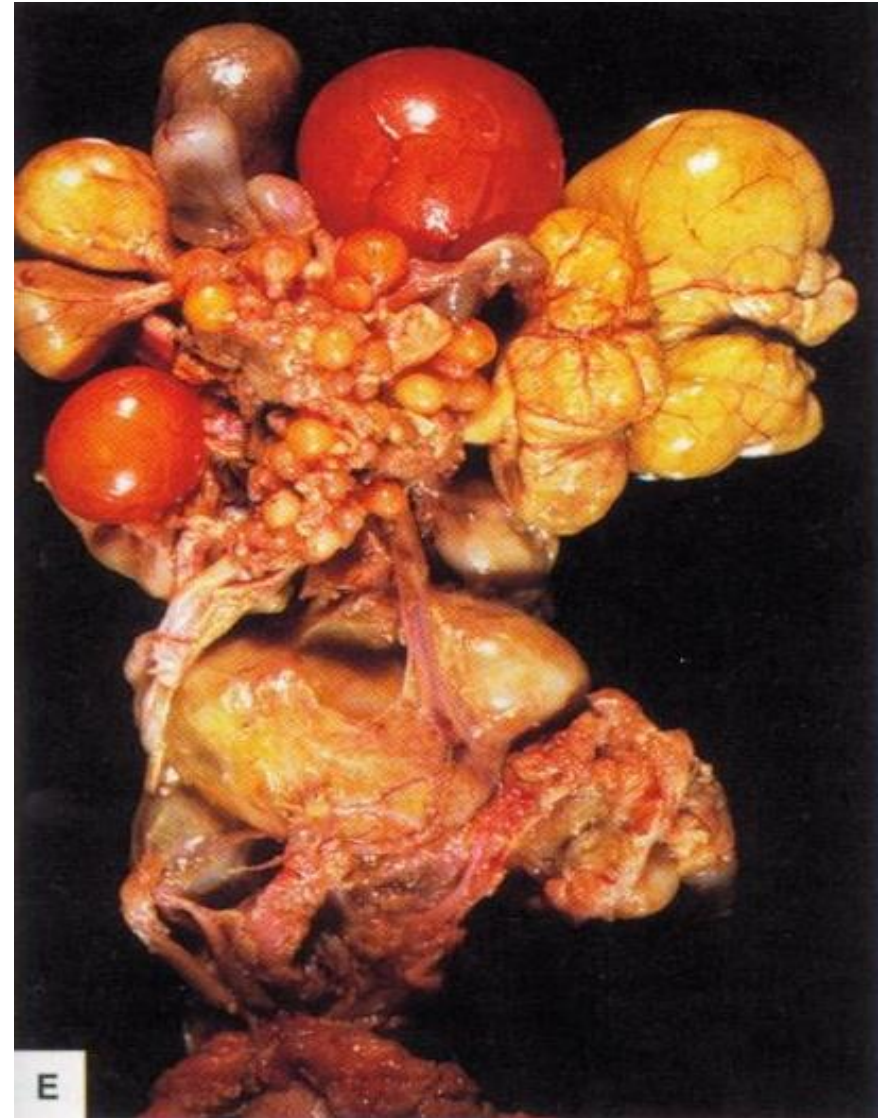


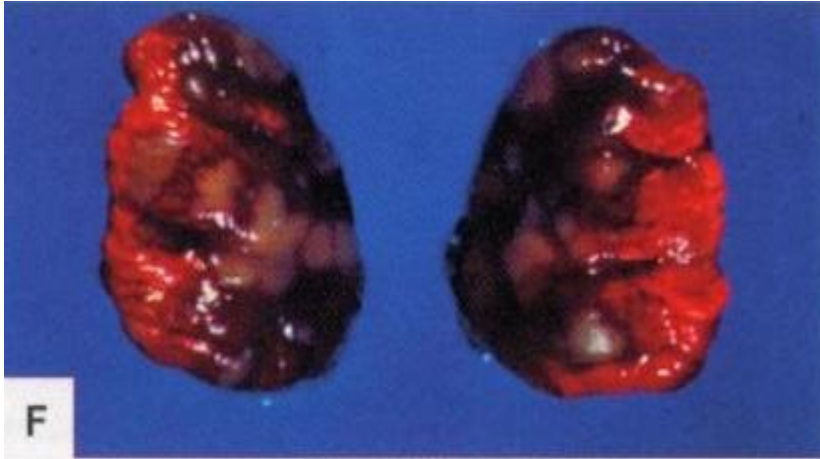
Fig. 6
Heart with prominent white nodules resembling tumours in the myocardium of a five-week-old chick affected by pullorum disease



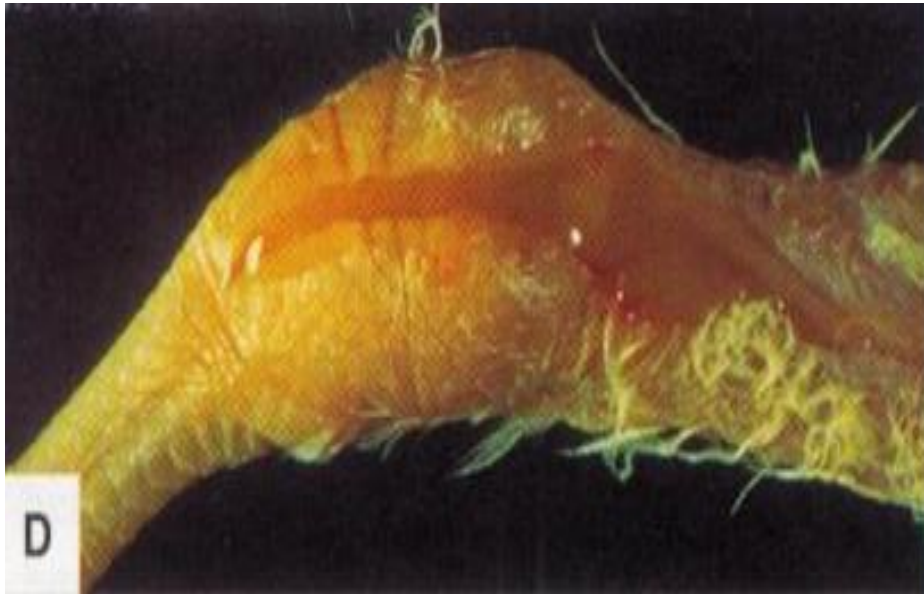
Spleen Nodules
Granulomatous splenitis



Ovarian lesions and salpingitis



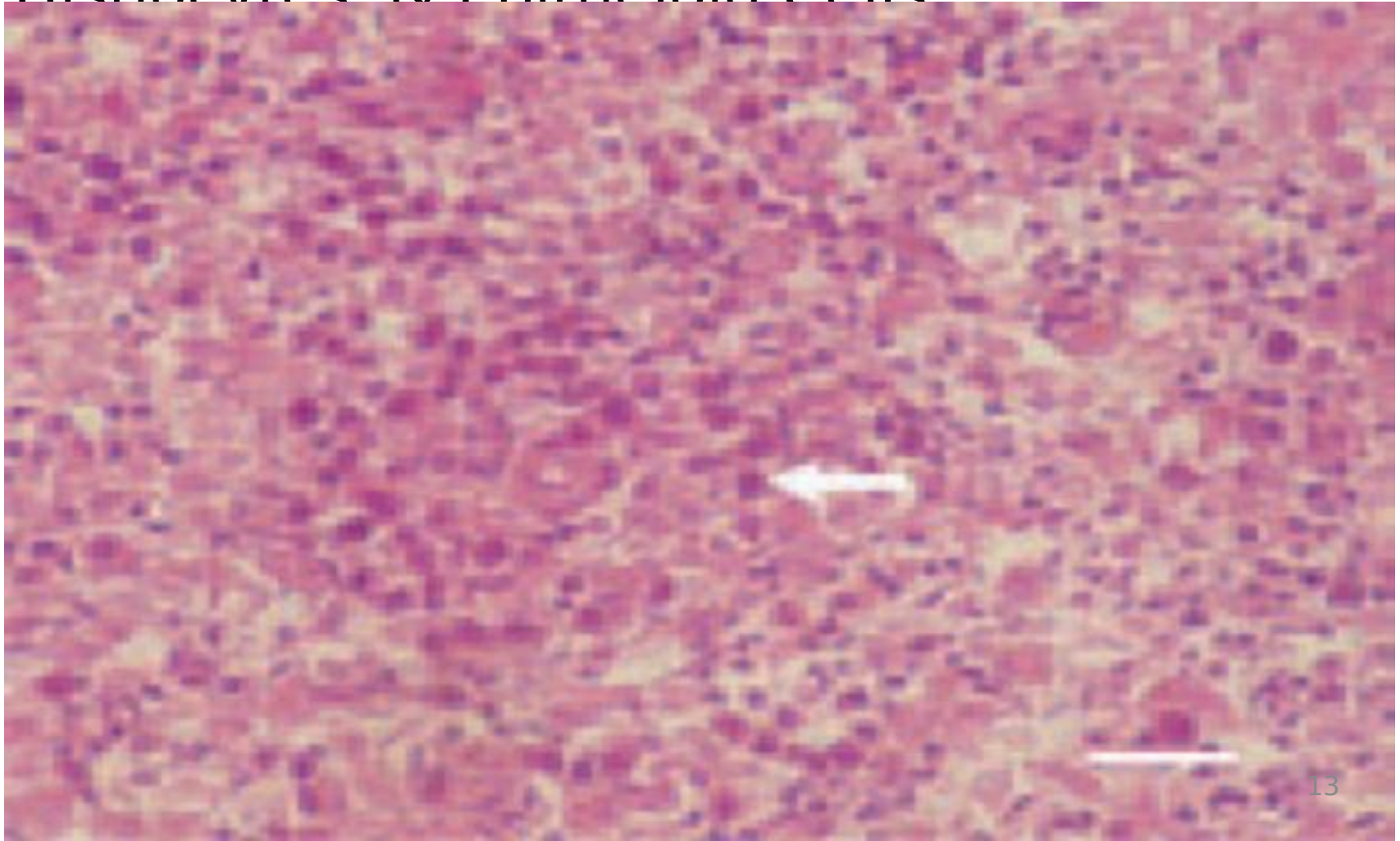
Lungs with
pyrogranulomatou
s pneumonia due
to pullorum
disease in a chick



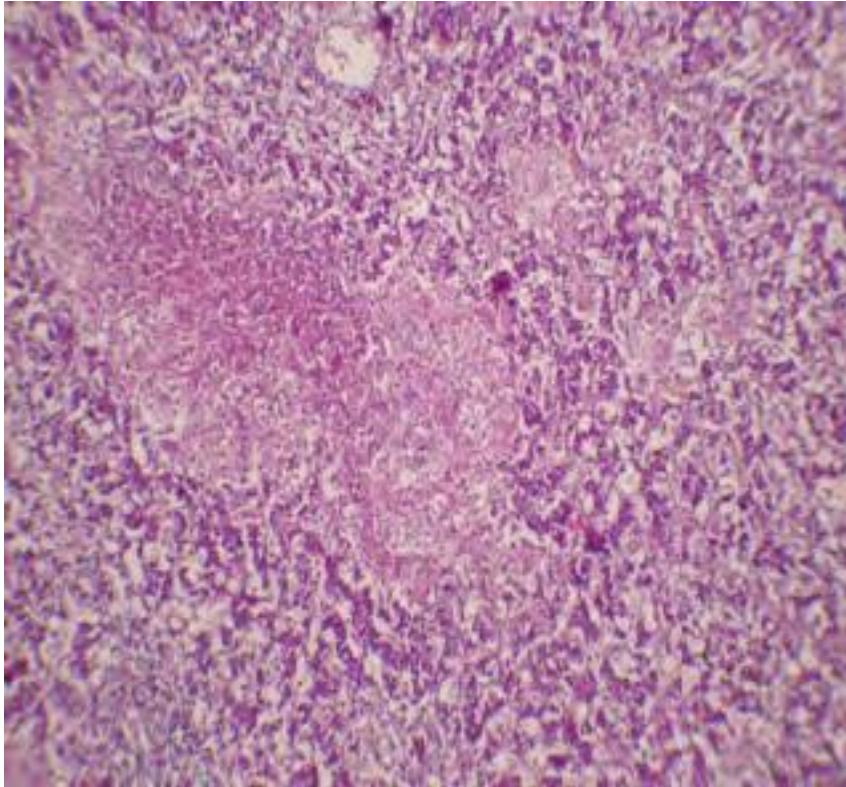
Swollen hock joint
containing yellow
viscous fluid

Microscopic Lesion

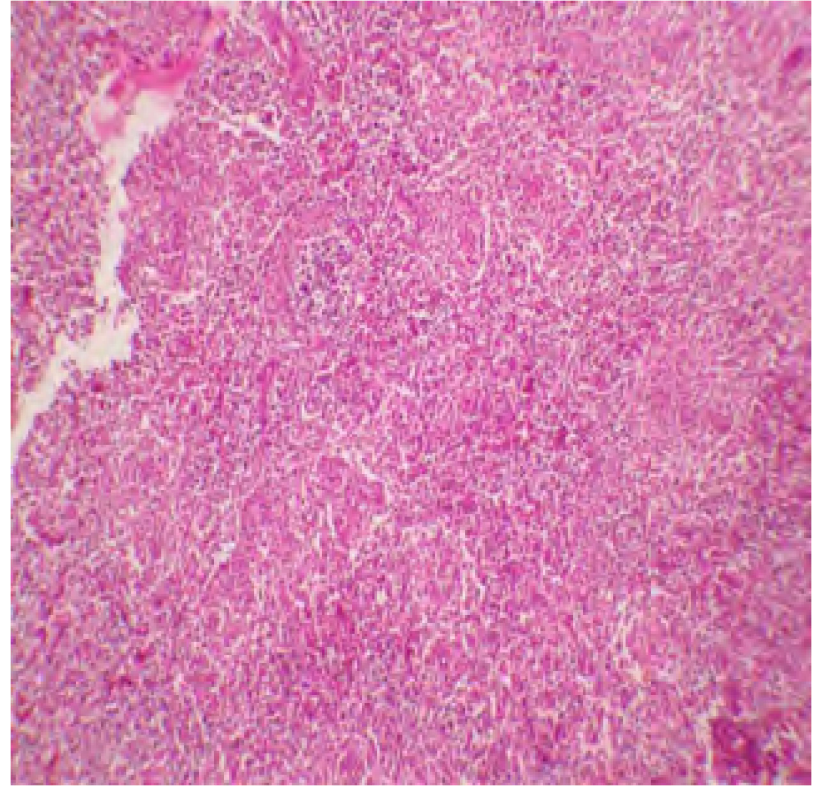
- Perivascular Proliferative Inflammatory Focus in the Liver, Consisting Mainly Of Histiocytes & Epithelioid Cells



Liver: Showing large area of coagulative necrosis, surrounded by leucocytes (*S. Gallinarum* infection).



Spleen: Showing multiple necrotic areas (*S. Gallinarum* infection)



Diagnosis

⌘ Tentative diagnosis based on **history**, **clinical signs** and **post-mortem**

- The clinical signs, flock history, **High mortality** in chicks and post-mortem lesions can be **suggestive**,
- **Laboratory confirmation is essential.**

⌘ ***Laboratory tests***

- Definitive /Positive Diagnosis – **Isolation & identification** of agent.
- grow on standard nonselective media & selective media(MacConkey, brilliant green &xylose lysine deoxycholate agars)

Differential diagnosis

- Fowl typhoid
- Fowl cholera
- Erysipelas

Samples to collect

- Culture is more likely to be successful in birds that have not been treated with **antimicrobials** for approximately 2 to 3 weeks.
- In live birds, **cloacal swabs** may be taken
- **swabs or tissue samples** are collected from **grossly** abnormal tissues at necropsy. (**liver**, **spleen**, yolk

Commonly used specimens required for diagnosis:

- ⌘ Tissue and faeces samples can be submitted for bacteria identification through culture or genetic techniques.
- ⌘ Serological tests are satisfactory for establishing the presence and estimating the prevalence of infection within a flock.

Risk of introduction:

- └ Pullorum could be introduced by importation of live infected chicken, hatching eggs.
- └ The bacteria can also be found in poultry meat but contamination of poultry flocks through this route is at low risk

Treatment

- Results of treatment of pullorum disease are poor.
- Treatment is for flock salvage only.
- Several sulfonamides, antibiotics, and antibacterials are effective in reducing mortality, but none eradicates the disease from the flock.
- Mortality can be controlled with:
 - Antibiotics: tetracyclines, gentamycin, and

Control

- Establish and maintain **Pullorum-free breeders**.
- **Purchase chicks from hatcheries that free of the disease or tested before adding them to a flock. .**
- Rodents and wild birds should be excluded, and insects, particularly flies should be controlled.
- Organism **in hatchery** can be killed by **formaldehyde fumigation**.
- Voluntary regulatory program (+) reactors must be disposed of under supervision.
- the entire flock is **depopulated** and the premises
(building are cleaned and disinfected before restocking

Prevention

- θ Breeding stock and chicks should be **purchased** from suppliers and hatcheries **certified free of *S. Pullorum***
- θ Breeder flocks can be monitored using the rapid **whole-blood plate agglutination** test.
- θ **Strict biosecurity**
- θ **Rodent eradication** is an important

Dead of chicken due to Pullorum disease



Fowl typhoid

Etiology: *Salmonella gallinarum*

- It is a **septicemia disease** affecting mainly chickens and turkeys
- It is transmitted through eggs
- Mortality in young birds is similar to *S. pullorum* infection but may be higher in older birds.

Occurrence and Economic Significance

- ♣ **world-wide in distribution** and frequently encountered in subsistence or semi-commercial flocks.
- ♣ The serious **economic losses** in commercial units in endemic areas.
- ♣ mortality in both mature and immature flocks
- ♣ **loss of egg production and increased**

Transmission

∞ Vertical and lateral transmission

Clinical signs:

Acute

- ♣ Increase in mortality, drop in feed consumption
- ♣ depression, ruffled feathers and **closure of eyes**, respiratory distress with rapid breathing, **watery to mucoïd yellow diarrhea**

Chronic

- ✚ Progressive loss of condition
- ✚ Intense anemia, pale combs and wattles
- ✚ Rapid spread, losses of 50% or more

Sub acute: Sporadic mortality , Occasional

Gross lesion

Acute form

- Carcass appears **septicaemic and jaundiced**
- *Subcutaneous blood vessels:* **hyperaemic and engorged**
- *Skeletal muscles:* Congested and dark in colour
- *Consistent finding:* Dark reddish or almost blackish swollen friable liver
- *Spleen:* Enlarged
- **Small intestine:** Catarrhal enteritis with **viscous, slimy, bile-tinged materials**
- *Characteristic feature:* Dark brownish bone marrow

Chronic form

- ⌘ Emaciated and anemic carcass,
Greyish white necrotic foci in
myocardium, intestines, pancreas and
liver.
- ⌘ *Characteristic feature:* Pericarditis with
yellow turbid fluid in the pericardial sac
- ⌘ fibrin attached to the surface of the
heart
- ⌘ *Young chicks:* Discrete necrotic foci in
lungs and gizzard.
- ⌘ *Cockerels:* Well defined necrotic foci in
testicles.

Microscopic lesion

Liver:

- Showing large area of coagulative necrosis, surrounded by leucocytes *infection*).

Spleen:

Showing multiple necrotic areas.

Diagnosis

- Based on tentative diagnosis, history, signs and lesions
- Confirmatory diagnosis: Isolation and identification of organisms.
- Serological tests - Detection of antibodies
 - Stained antigen whole blood (WB) test.
 - Macroscopic Tube agglutination (TA) test
 - Micro agglutination (MA) test
 - ELISA: can be employed for screening large number of samples
 - Rapid plate agglutination test or whole blood test: Using *S.pullorum* (to detect carriers of *S.gallinarum*)

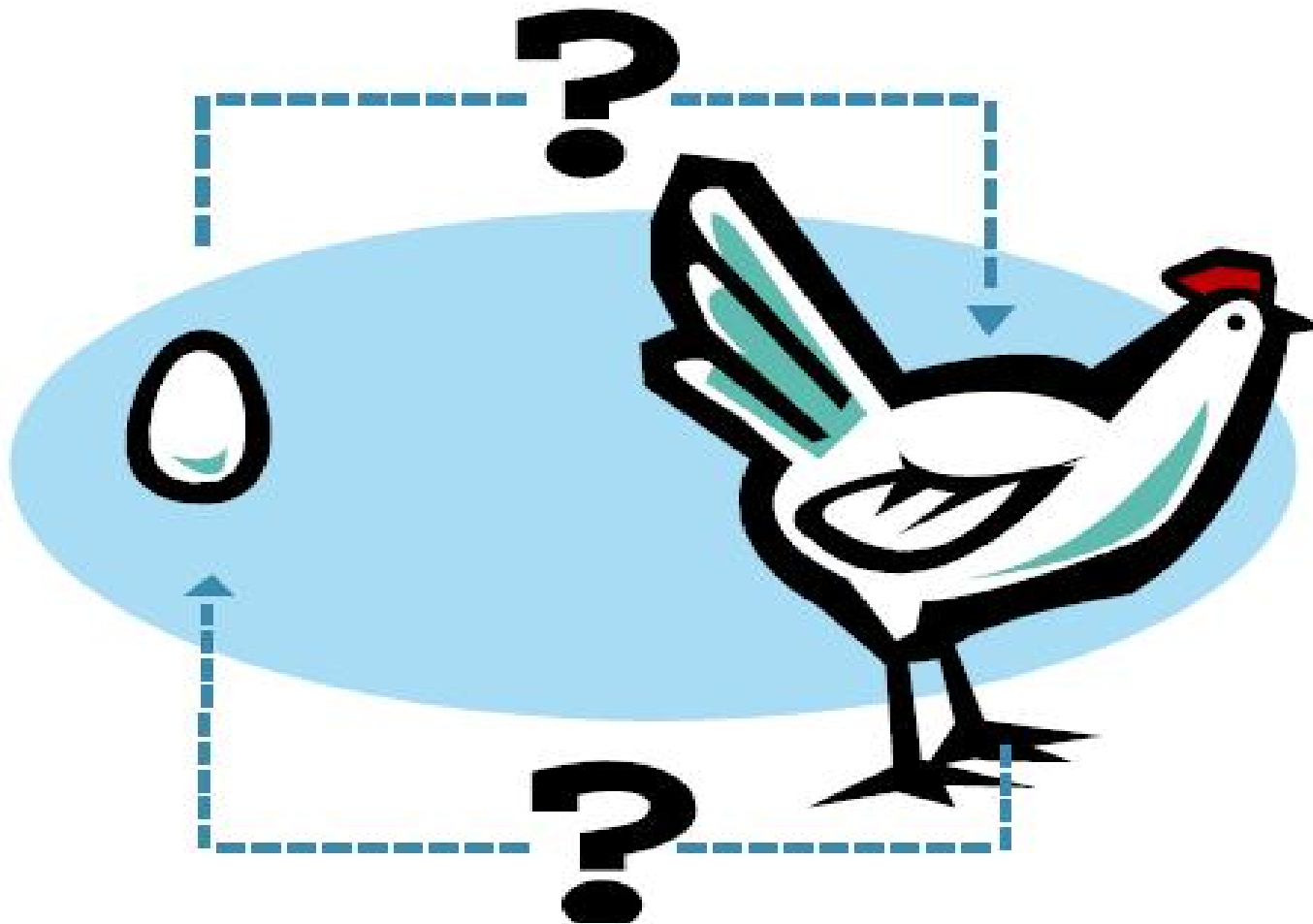
Treatment and control

- There are no federally licensed vaccines
- Commercial laying flocks may be salvage under specific conditions by administering furazolidone or tetracycline in feed at 400 g/ton, for two weeks, where permitted.

Prevention

- Appropriate biosecurity measures should be implemented as for *S. pullorum* infection
- Administration of live 9R strain *S. gallinarum* vaccine during the rearing period will eliminate outbreaks of clinical disease
- Bacterins are generally ineffective in preventing fowl typhoid.

Questions?



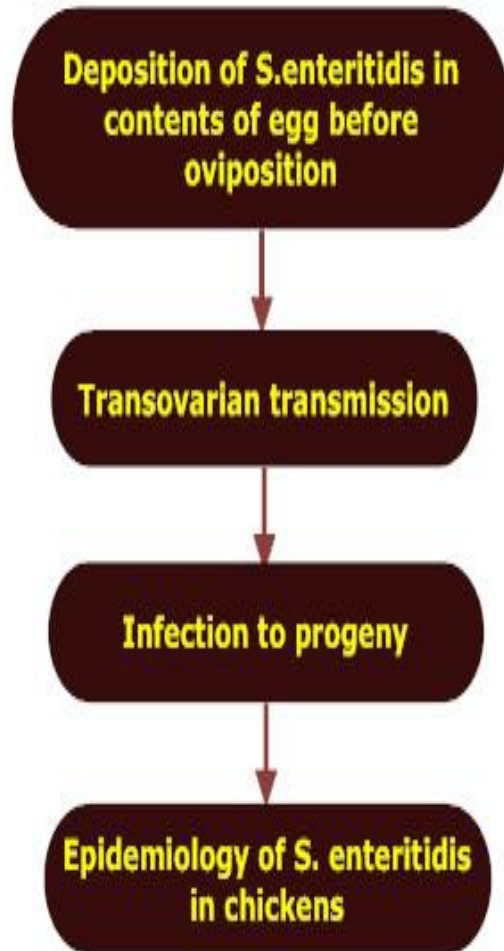
Paratyphoid in poultry

Etiology

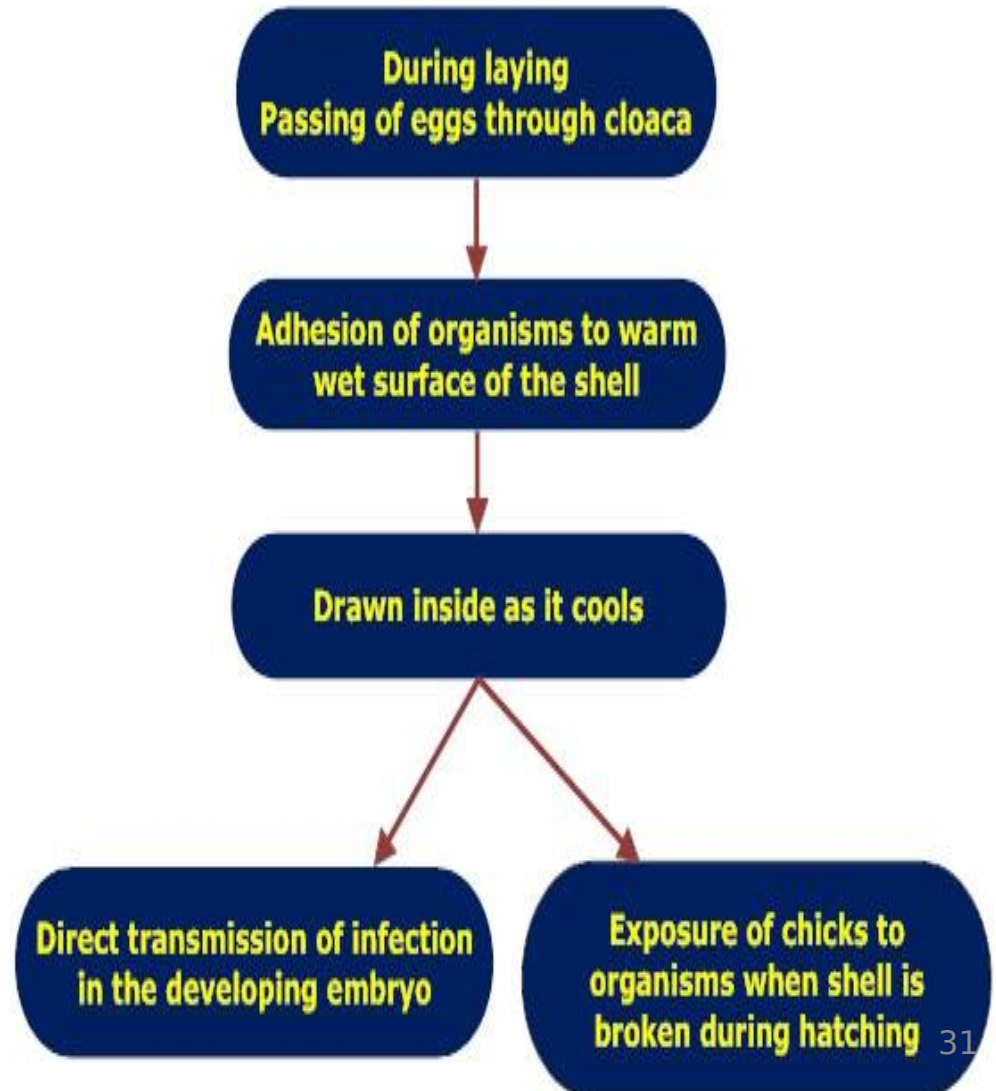
- ♣ Motile Salmonella serotypes, other than those in the *S. arizonae* sub genus are often referred to as, “Paratyphoid (PT) salmonellae”.
- ♣ These organisms can infect a wide variety of hosts, including humans
- ♣ In some cases asymptomatic intestinal carriers and produce clinical diseases.
- ♣ The most commonly isolated serotypes from chickens (i.e. PT salmonellae) are:
 - ‖ *S.typhimurium*
 - ‖ *S. enteritidis, S. hadar*
 - ‖ *S. montevideo, S.kentucky and, S. heidelberg.*

Transmission: Vertical transmission

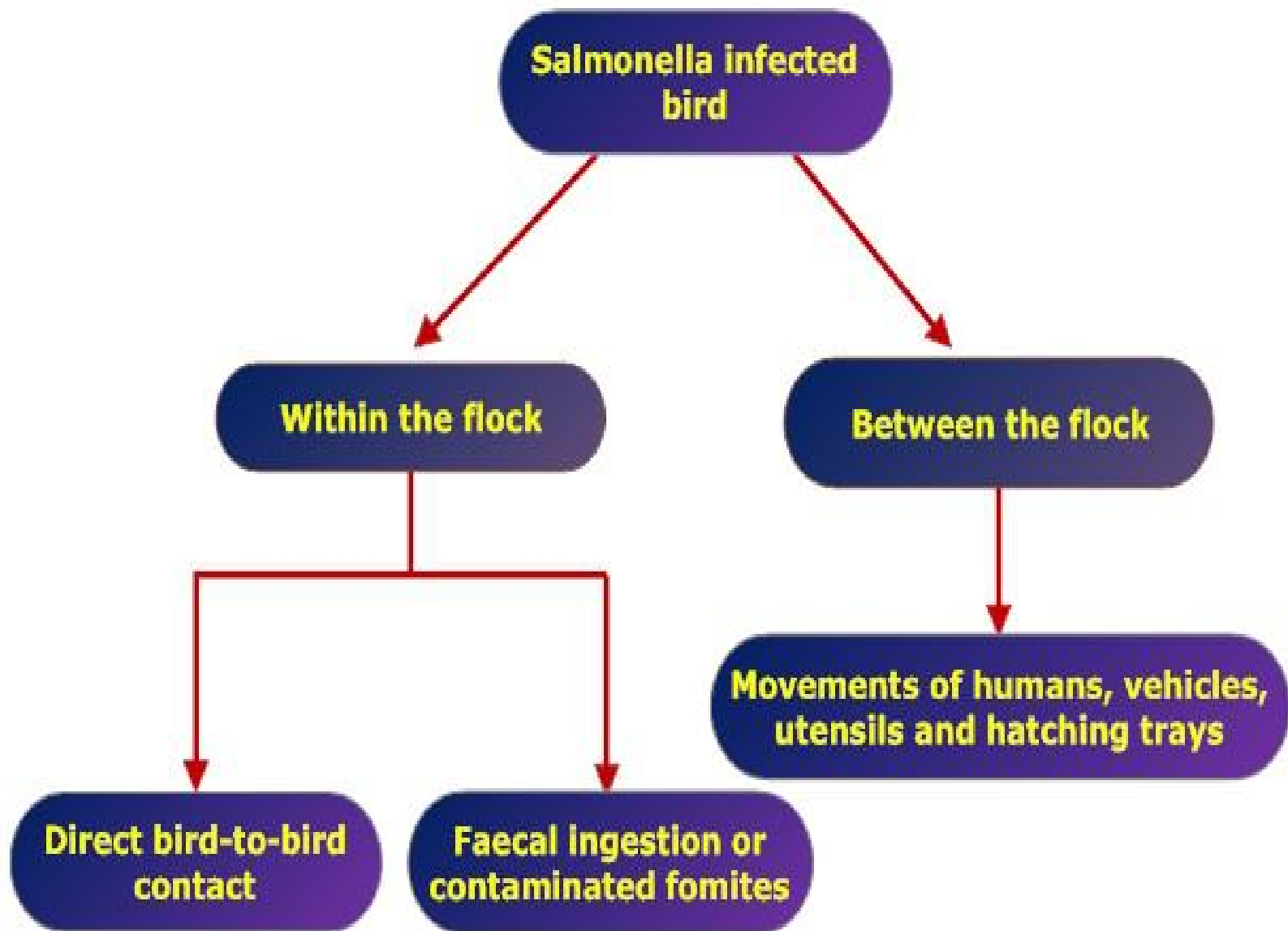
Internal deposition



External deposition



Horizontal transmission



Clinical signs

- Young chicks, poults or ducklings are commonly affected
- Mortality is usually less than 20%, exceptional case 100%.
- Ruffled feathers and drooping wings, and grouped near the sources of heat.
- Depression, closure of eyes and reluctance to move
- Diarrhoea with pasting of feathers around the (pasty) vent is common

Paratyphoid infection in chicken



Post mortem lesion

Acute:

- Enlargement (spleen and liver), enteritis, peritonitis.
- omphalitis.
- A septicaemic carcass with the congestion of lung, and swollen kidney.
- In chicks unabsorbed yolk sac
- Discrete (separate) **necrotic lesions** in the lungs, liver and heart.
- **Hemorrhagic enteritis.**
- The most characteristic lesion is **typhilitis** (inflammation of caeca) with the caeca distended by hard white necrotic cores.



Arthritis and peri-arthritis are encountered in broilers



Conjunctivitis and consequent keratitis and panophthalmitis

Fibrinous exudate in caeca
forms casts with the shape of
mucosal folds



Subcutaneous
haemorrhages in the
muscles



Diagnosis

- isolation and identification of *Salmonella* spp from liver, spleen, intestine, or heart blood.
- Routine microbiological screening of liver/spleen/intestinal pools from post mortem submissions to laboratories is strongly recommended.
- Specific ELISA-based test kits are available for assaying for *S. enteritidis* antibody.

Public health significance

- food-borne illness throughout the world.
- The paratyphoid infection in birds acquire in human illness caused by the consumption of contaminated poultry products

Treatment

- ⌘ Several antibacterial agents help prevent mortality but cannot eliminate flock infection.
- ⌘ Furazolidone if allowed will suppress mortality but will not eliminate infection.

Prevention

- implementing intensive programs of microbiological screening and biosecurity procedures.
- Strict sanitation in the hatchery
- fumigation of hatching eggs
- cleaning and disinfection of poultry house
- rodent control
- using live, modified *S. typhimurium* vaccines alone or in combination with inactivated *S. enteritidis* emulsion boosters



Avian colibacillosis (*Escherichia coli* infections (*e. Coli*)

- Synonym: Colibacillosis (*Escherichia coli* infection, avian colibacillosis, coliform infection)
- Economic losses can be due to decreased hatching rates, mortality, lowered production, carcass condemnation at processing and treatment costs.
- Affect all avian species, at all ages.
- Widespread distribution, it is an intestinal inhabitant.

The most common **co-infections** to occur with E. coli:-

- ⌘ Chronic respiratory disease (CRD)
- ⌘ Mycoplasma gallisepticum (MG) infection

→ Colibacillosis in neonatal chicks can also be a consequence of:

- └ Poor chick quality
- └ Sanitation in the hatchery, **leading to early chick mortality.**

Localised or systemic infections and syndromes caused by avian pathogenic E. coli

Localised infections	✚ Coliform omphalitis / yolk sac infection ✚ Coliform cellulitis (inflammatory process) Swollen head syndrome ✚ Diarrhoeal disease ✚ Venereal colibacillosis (acute vaginitis Coliform salpingitis / peritonitis Coliform orchitis / epididymitis
Systemic infections	→ Colisepticaemia → Haemorrhagic septicaemia → Coligranuloma (Hjarre's disease)
Colisepticaemia sequelae	} Meningitis } Encephalitis } ...

Etiology

The etiology of colibacillosis can be:

- Either due to **primary infection** with avian pathogenic *Escherichia coli* (APEC) or
- **Secondary (opportunistic) infection** after a primary insult has occurred.
- The etiologic agent is ***E. coli*** family ***Enterobacteriaceae*** meaning it is found in the **intestine**.
- This organism is **coliform**, gram negative, and motile.
- Since *E. coli* is a common inhabitant of the **intestine**, it is widely disseminated in **faecal material and litter**.
- Additionally, **contaminated** feed, feed ingredients, drinking water, and **rodent**

- While most strains are considered to be non-pathogenic, certain strains have the ability to cause clinical disease.
 - The O (somatic) antigen serotypes most commonly associated with disease outbreaks are **O1, O2, O35**, and **O78**.
 - The K (capsular) antigens most commonly associated with virulence are K1 and K80.
 - In the **intestinal tract** of normal poultry, nonpathogenic serotypes far outnumber

Factors pre-disposing to colibacillosis

Predisposing factors during peaking period	‣ Multi-age complexes ‣ Exposure to endemic mycoplasmas (M. gallisepticum or M. synoviae) and/or infectious bronchitis virus (IBV) ‣ Poor ventilation with high levels of dust and/or ammonia ‣ Stress of production in a young developing bird ‣ High levels of circulating endogenous hormones (especially oestrogen)
Predisposing factors	⌘ Vent trauma, non-lethal vent cannibalism,

Routes of Transmission

E. coli can enter the body by various routes, all of which can lead to colibacillosis.

A. Respiratory tract

- **Inhalation** of contaminated dust is the most likely source of E. coli infection (colibacillosis) for poultry.
- Damage to the respiratory tract from an infection (e.g. Newcastle disease virus, infectious bronchitis virus, M. gallisepticum, infectious laryngotracheitis, etc.)
- Breakdown of the mucosal lining in the trachea has the potential to allow pathogenic bacteria to enter the blood stream which can lead to **septicaemia**.

B. Gastrointestinal tract

- ┌ Coccidiosis
- ┌ enteritis
- ┌ Mycotoxins
- ┌ Antibiotics
- ┌ poor water quality
- ┌ abrupt feed

ability to disrupt
the normal
bacterial flora
of the intestine.

Pathogenic
E. coli can
invade the gut

→ When the mucosal barrier is disturbed,
pathogenic **ingestion of contaminated water,
feed, and litter** can serve as sources of *E. coli*.

C. Skin

- ⌘ Wounds and other breaks in the skin from scratches (due to overcrowding or old cages)
- ⌘ Rough handling by crews, ectoparasites or unhealed navels in chicks provide opportunity for pathogenic bacteria to enter the body.

D. Reproductive tract

- ⌘ **Ascending infections** travelling up the oviduct lead directly into the hen's body cavity.
- ⌘ **Vent pecking and prolapse** can lead to peritonitis.
- ⌘ **Oviduct infection**, respiratory disease and handling birds during late transfer (after the onset of egg production) can all result in yolks (or ova) laid outside the oviduct with the potentially developing into egg yolk

E. Omphalitis (yolk sac infection, navel ill, “mushy chick” disease)

- **Omphalitis**, or inflammation of the navel (umbilicus), is one of the **most common causes of mortality in chicks during the first week**.
- **Faecal contamination of eggs** is considered to be the most important source of infection; however, bacteria can translocate from the chick's gut or from the blood stream.
- **Infection with E. coli** follows contamination of an **unhealed navel and may also involve the yolk sac**.
- **Clinical signs of omphalitis** include swelling, oedema, redness and scabbing of the navel area and/or yolk sac.
- **In severe cases**, the body wall and skin undergo lysis, causing the chicks to appear wet

Pathogenesis

- Ingested *E. coli* → adhere to microvilli of enterocytes → colonize, proliferate, and elaborate enterotoxins → excessive secretion of fluid and electrolytes by crypt epithelial cells → markedly exceeds absorptive capacity → flow of tissue fluids into the lumen → enterotoxins, endotoxin → damage the microvilli and enterocytes → reduces the absorption of electrolytes, water from the lumen → diarrhea results.

- Damage to epithelial ~~cells~~ → septicemia.
- Diarrhea usually continues until death results from dehydration and metabolic acidosis or from terminal septicemia.
- Poor air quality and other environmental stresses predispose to E coli infections.
- Systemic infection occurs when large numbers of pathogenic E coli gain access to the bloodstream

Clinical Findings and Lesions

- Signs are **nonspecific** and vary with **age, organs involved, and concurrent disease**.
 - Respiratory signs, coughing, sneezing/Reduced appetite/Poor growth.
 - Omphalitis (yolk sac infection)
- Young birds dying of **acute septicemia** have few (no) lesions except for an **enlarged, hyperemic liver and spleen with increased fluid in body cavities**.
- Birds that **survive septicemia** develop **subacute** fibrinopurulent *airsacculitis*, *pericarditis*, *perihepatitis*, *Salphingitis*, Omphalitis (yolk sac infection)

- **Airsacculitis:** is a *classic lesion of colibacillosis*. occurs chiefly in 3-7-week-old broilers.
- Pericarditis, Arthritis
- **Sporadic lesions:** *pneumonia, arthritis, osteomyelitis/synovitis, peritonitis, and salpingitis* the **oviduct is distended with exudates that may be cheesy and has a foul odor.**
- The intestines are **pale and distended, particularly the caeca** that are **overfilled with fluid containing many gas bubbles**
- Cystic degeneration of **ovarian follicles** (E. 54

Egg peritonitis: yolk mixed with exudates. It is the inflammation of the hen's peritoneum

Omphalitis (navel infection): It is characterized with reddening and tissue oedema in the umbilical region, common condition affecting the navel of newly hatched chicks.

Salpingitis: inflammation of the oviduct. The oviduct is dilated and filled with caseous exudates.

Cellulitis: inflammation of the subcutaneous tissue.

- Affected areas are mostly in the region of the

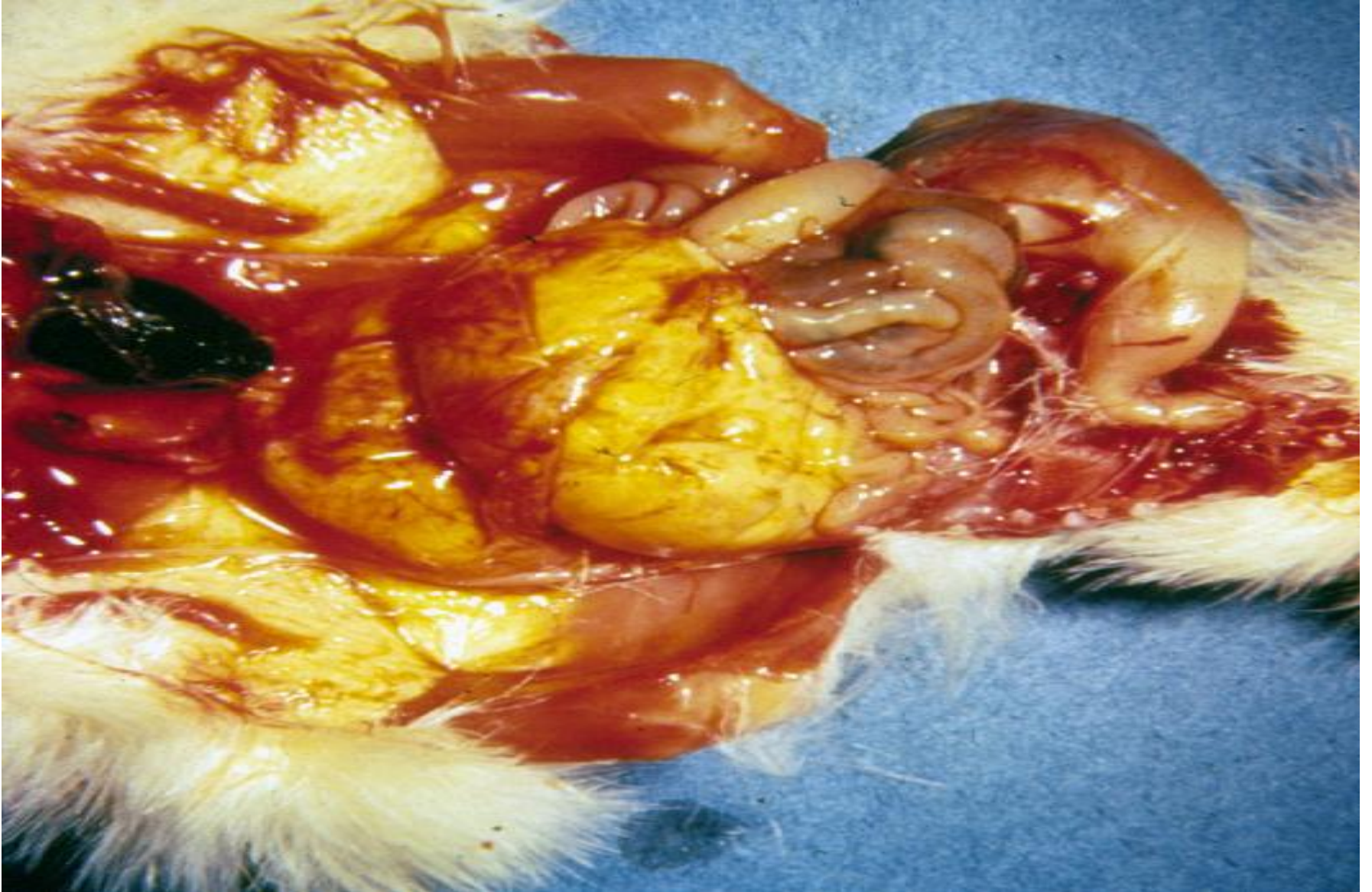
Coligranuloma (Hjärre's disease): Nodules (granulomas) occur along the intestinal tract, mesentery, and Granuloma on liver, duodenum, mesentery and ceca.



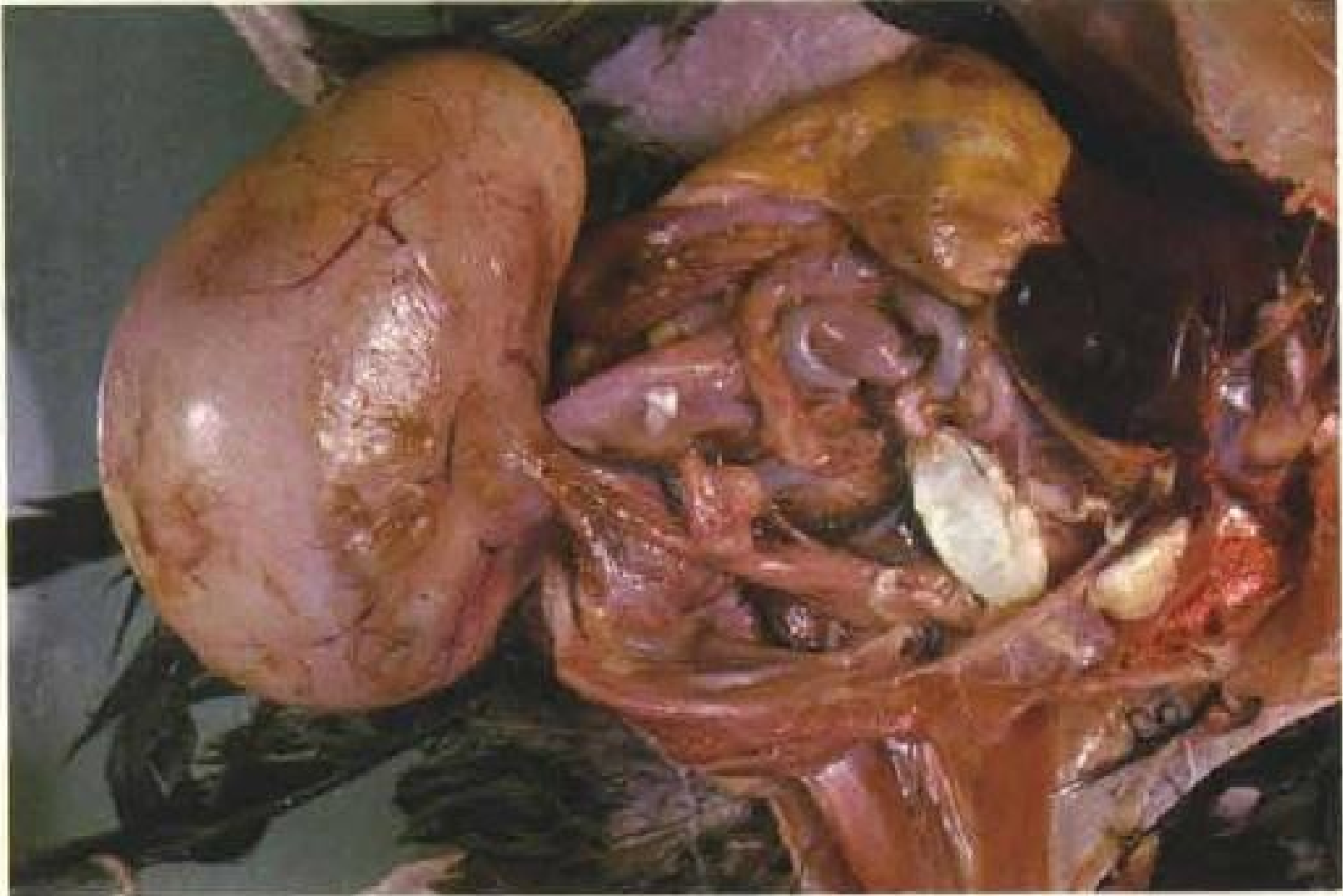
Egg peritonitis, yolk mixed with exudate



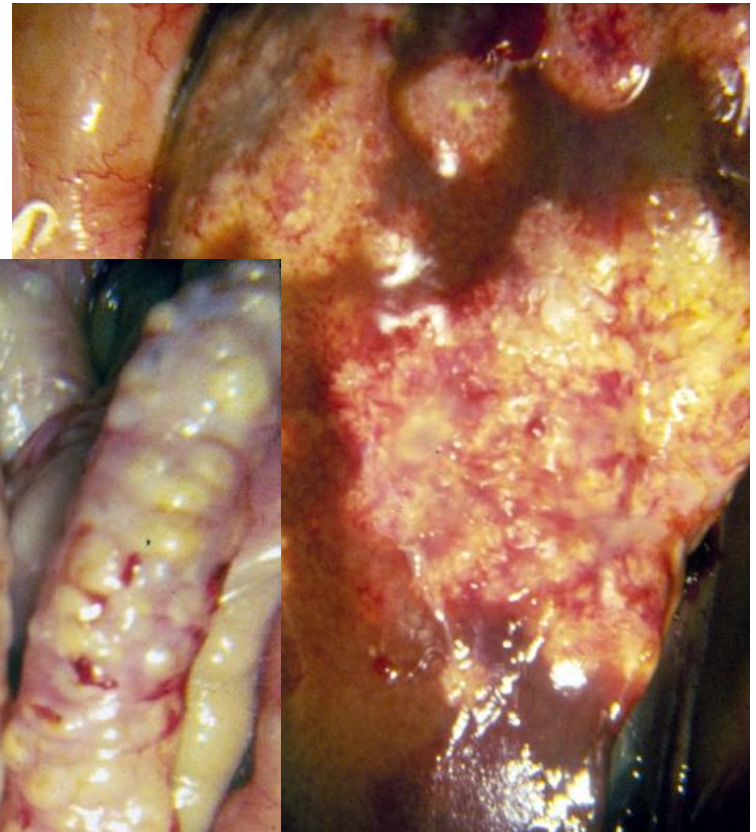
Omphalitis



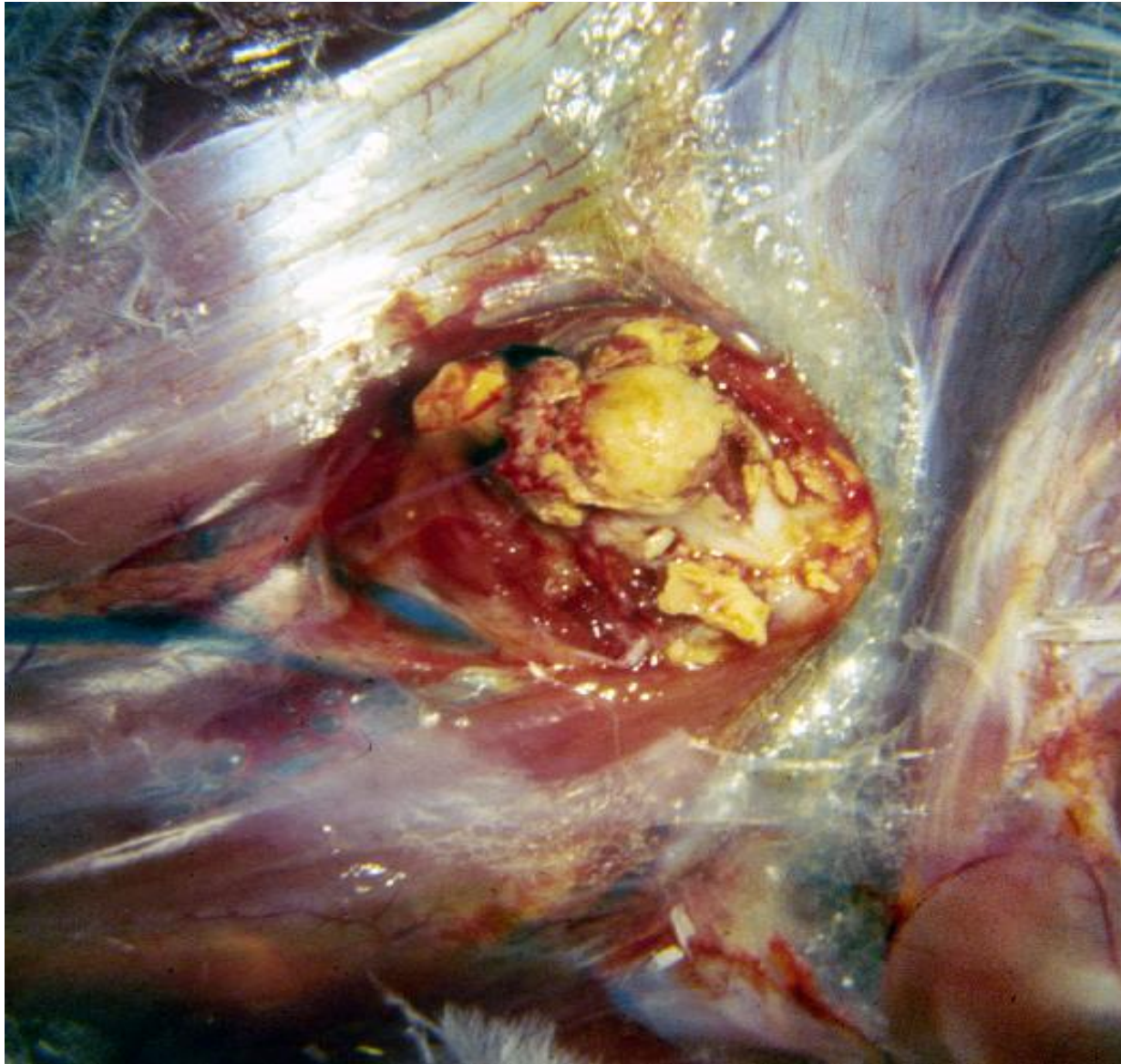
The affected oviduct is extremely enlarged



Coligranuloma



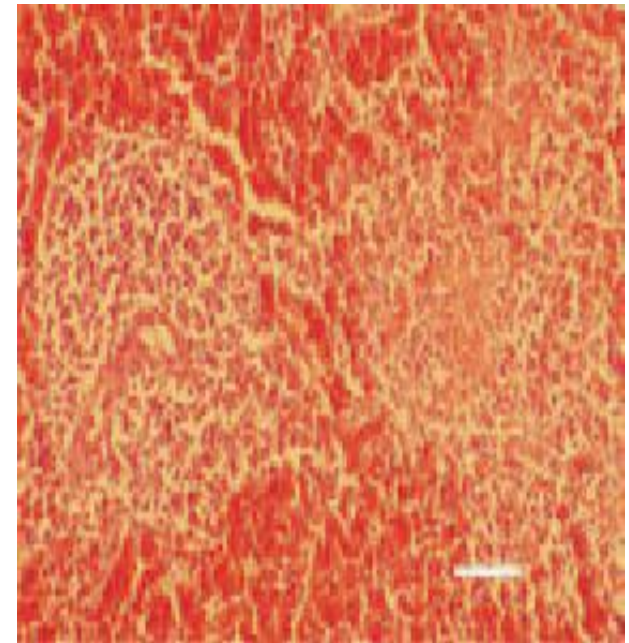
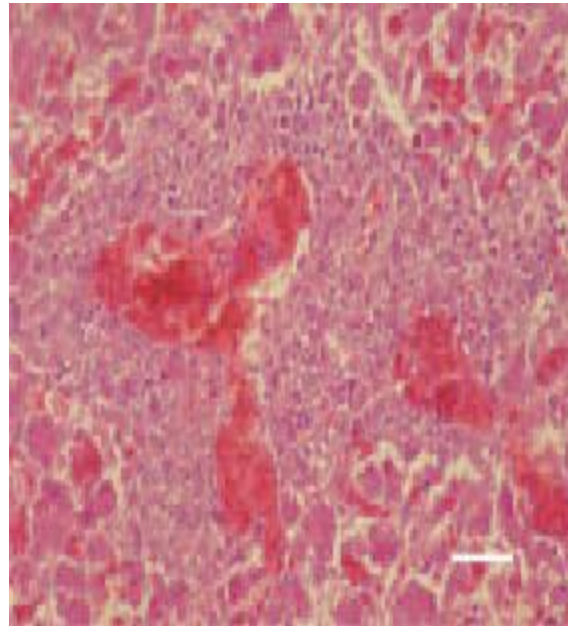
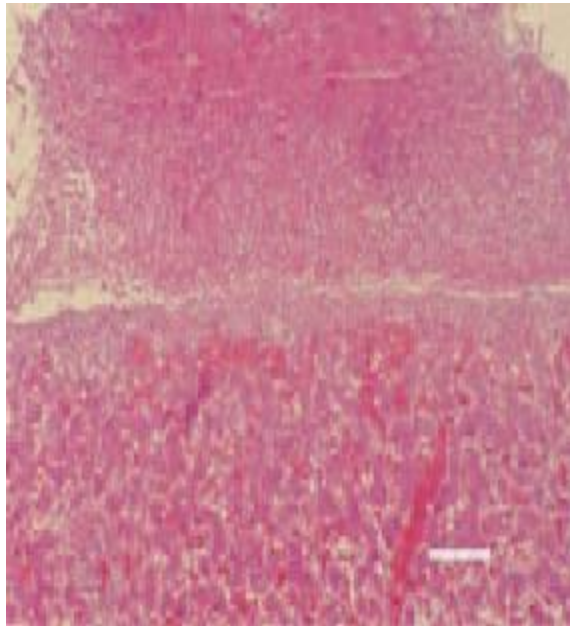
Arthritis



Microscopic lesion:

- ‖ Fibrinoid Necrosis and Congestion of the Spleen in *E. Coli Septicaemia*(C).
- ‖ Massive perivascular liver necrosis.
- ‖ Coligranuloma (Hjarre's Disease).
- ‖ Granulomatous Nodes against the Intestinal Wall.
- ‖ Central necrotic and marked acidophilia.

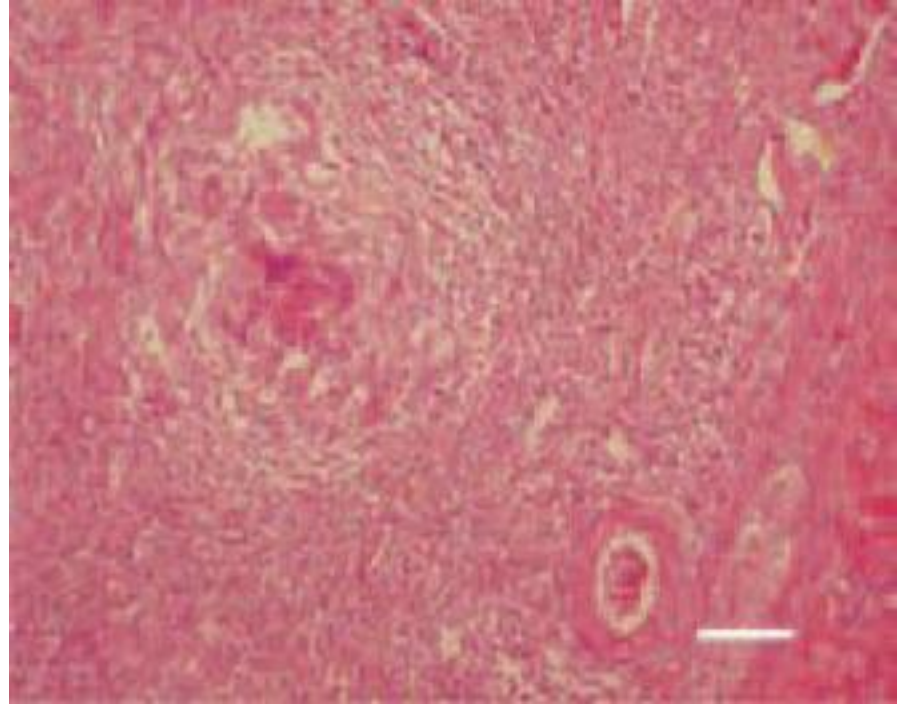
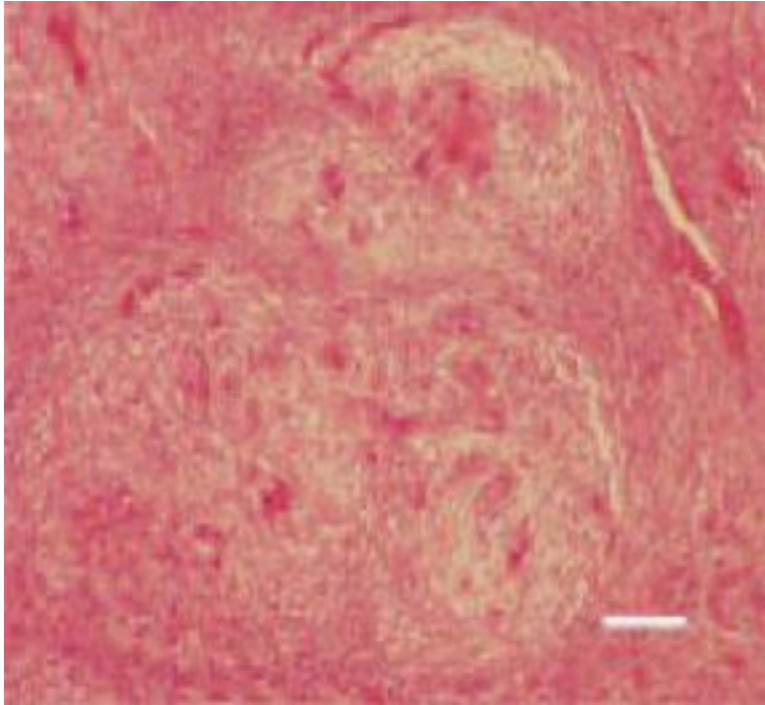
Periarteriolar Reaction, Fibrinoid Necrosis And Congestion Of The Spleen in *E. Coli* Septicaemia.



**Serofibrinous
Perihepatitis.
Organization Of
Pseudomembranous
Deposits.**

***E. Coli*Septicaemia.
Massive Perivascular
Liver Necrosis.**

Disease). Coligranuloma, Liver.



**Coligranuloma (Hjarre's Disease).
Conglomerates Of Granulomatous Nodes
Against The Intestinal Wall. Central Necrotic
Detritus And Marked Acidophilia. Single
Foreign Body-Type Giant Cells.**

Diagnosis:

- Based on history, Clinical signs, Bacterial culture, Necropsy
- Isolation of a pure culture of E. coli from heart blood, liver, or typical visceral lesions in a fresh carcass indicates primary or secondary colibacillosis.

Differential Diagnosis

- ▶ Mycoplasma
- ▶ Respiratory virus –NCD, IB
- ▶ Staph Infection
- ▶ Fowl Cholera
- ▶ Salmonella

Treatment

- Many different antibiotics and drugs including tetracyclines, neomycin, sulfa drugs and others.
- Antibiotic ***sensitivity*** testing is advisable
- Treatment is usually effective if given early.

Prevention

- ♣ minimize egg shell contamination of newly laid eggs (disinfection on the farm prior to storage and stored under ideal conditions).
- ♣ hatchery sanitation, disinfection, and/or fumigation procedures should be practiced.
- ♣ A vigorous sanitation program should be followed in raising poultry.
- ♣ feeds free of fecal contaminations should be fed to poultry.

CLOSTRIDIAL INFECTIONS

- Organisms are present in soil.
- Clostridium organisms are involved in conditions like:
 - A. Botulism
 - B. Necrotic enteritis
 - C. Gangrenous dermatitis

Botulism (limberneck/western duck sickness)

Etiology

- ‖ *Clostridium botulinum* (powerful exotoxin toxin producing organism).
- ‖ The disease affects poultry worldwide.
- ‖ There are 8 antigenic ally different toxigenic groupings (A, B, C-alpha, C-beta, D, E, F and G).
- ‖ Botulism toxins are among the most known potent toxins
- ‖ Type A and C are producing toxins affecting fowls.

Route of spread:

- (a) Eating of decomposed food/putrified carcase of fowl (In such fowls, if *Cl.* organisms are present in intestine → produce toxin).
- (b) Wild fowl develop it after eating dead maggots which have ingested toxin

Spread

- Type C organisms grow in the gastrointestinal tract of normal birds.
- Presence of organisms in the gastrointestinal tract, and resistance of spores to inactivation, favour spread of this organism.
- Outbreaks occur in chickens when the toxin is consumed, usually in decaying poultry carcasses, or toxin-containing maggots or beetles.
- The toxin produced in, and absorbed from, the caeca of chickens

Pathogenesis

- Type C botulism can be caused by ingestion of preformed toxin.
- Birds' scavenging (i.e., searching for decaying flesh as food) such carcasses can readily obtain enough toxin to become affected.
- The pathogenesis of botulism was once thought exclusively to be ingestion of preformed toxin.
- *C. botulinum* type C produces toxin in the gut to cause disease.
- Experimental evidence suggests caecum as the site of toxin production

Clinical Signs

- **In chickens**, flaccid paralysis (i.e. lacking firmness of muscles) of legs, wings, neck and eyelids are main features of the disease.
- Initially, affected birds are found sitting and reluctant to move.
- If forced to walk, they appear lame. Wings droop when paralysed.
- ruffled feathers.
- Limberneck, the original and common name for botulism, precisely describes paralysis of the neck.
- Death results from cardiac and respiratory failure.

Lesions: There are no gross or microscopic lesions.

Diagnosis

- The **presumptive diagnosis** of botulism is based on **clinical signs and lack of gross or microscopic lesions**.

Definitive diagnosis requires detection of in serum, crop, or gastrointestinal washings from sick birds.

- The presence of toxin is confirmed by the injection of serum, or extracts of crop, or intestinal contents, into mice. it → unheated toxin → death occurs. If heated/immunized mice → No death.
- Since **toxin** can be produced in **decaying**

Control: Avoid feeding decomposed feed and vegetables.

Treatment: Mildly affected birds' → antitoxin.

Ulcerative enteritis

- It is a bacterial infection of young chickens and turkeys
- characterised by sudden onset and rapidly increasing mortality.
- The disease was first seen in enzootic proportions in quail, and was therefore called "quail disease"
- many avian species other than quail are susceptible. Therefore, the earliest name has been replaced by "ulcerative enteritis".
- It is an acute, highly contagious disease of

- ‖ In chickens, it linked to stress, coccidiosis, infectious bursal disease, and other predisposing factors.
- ‖ It is characterized by ulcers of the intestines and caecae

Etiology

- *Clostridium colinum* is the etiologic agent.
- It is an anaerobic, fastidious /difficult to culture, gram-positive, spore-forming.
- The spores result in persistent contamination of premises after an outbreak.

Clinical Findings

- ♣ Sudden death without signs, with up to 100% mortality in just 2–3 days (in quails especially).
- ♣ In chickens, the course of the disease is less severe and is accompanied by anorexia.
- ♣ Signs are similar to those seen in coccidiosis
 - Depressed/ listless birds with humped backs/ ruffled feathers/ diarrhea, and sometimes bloody or watery white droppings, especially in quail.
- ♣ Chickens recover within 2–3 wk and mortality rarely exceeds 10%.

Transmission

- ♣ The route of infection is **oral** and transmission is from **faeces of sick or carrier birds or via flies**.
- ♣ Predisposing factors include Coccidiosis (especially *E. necatrix*, *E. tenella*, and *E. brunetti*), IBDV and overcrowding.
- **Birds that develop chronic ulcerative enteritis or that have recovered from the disease remain carriers.**

Pathogenesis

Administre viable *C. colinum* cells orally.
↓
the bacterium adheres to the intestinal villi
↓
inflammation and ulcers in the small intestine
↓
organism migrate to the liver via portal
circulation
↓
small necrotic foci and hepatic necrosis
↓
in situ-produced toxin.
↓
recovered birds remain carriers

active infection, shed the bacterium

- Flies feeding on contaminated fecal material can also introduce infection

Lesions

- ♣ After ingestion of the infected material, the bacteria **inhabit the intestinal villi of the bird** → enteritis → ulcers (small and large intestines).
- ♣ In early disease → small, round ulcers surrounded by hemorrhages in the small intestine, ceca, and upper large intestine.
- ♣ Small ulcers later coalesce/ band together to form larger, sometimes perforating ulcers, producing local or diffuse peritonitis.
- ⌘ The presence of blood in the gut resembles coccidiosis.
- ⌘ Characteristic **yellow to gray necrotic foci** are the predominant **lesions in the hepatic parenchyma.**

Microscopical lesions

- Intestinal ulcers consist of **small haemorrhagic and necrotic areas**, often with **clumps of Gram-positive bacteria**.
- The ulcers involve **villi and excreted into the submucosa**.
- The ulcers sometimes reach as deep as the muscular coat and serosa.
- **Liver lesions** consist of multifocal foci of **coagulative necrosis that** are often poorly demarcated and with minimal inflammatory reaction.
- **Gram-positive bacteria are occasionally found within the necrotic foci**

Diagnosis

- intestinal ulcerations and yellow to gray necrotizing lesions in the liver assist in diagnosis.
- *C. colinum* can be seen in gram-stained smears of the liver and intestinal lesions.
- In bacteremic birds, the microorganism can also be found in blood and spleen smears.
- In chickens, differentiating ulcerative enteritis from coccidiosis may be difficult as both diseases may be present simultaneously.
- Necrotic enteritis and histomoniasis may also present a diagnostic problem, but the hepatic lesions of ulcerative enteritis help differentiate it from these diseases.

- ♣ A fluorescent antibody test also has been used to accurately diagnose ulcerative enteritis.

Prevention, Treatment, and Control

- Streptomycin (0.006%) and furazolidone (0.02%) in the feed are effective for treating the disease.
- Prevention must start with good **management practices** (eg, avoiding the introduction of new birds into existing flocks).
- High population density is a predisposing factor.