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Stara Zagora

Assoc. Prof. Dr. Ivan Dinev



ESCHERICHIA COLI INFECTIONS

Escherichia coli infections in poultry are a group of either local or systemic diseases, where *E. coli* is the primary or the secondary agent.

LOCAL FORMS OF E. COLI INFECTION



1. Omphalitis (navel infection).

It is characterized with reddening and tissue oedema in the umbilical region.



2. *Escherichia coli* infections are widely distributed among poultry of all ages and categories. They are primarily related to poor hygienic conditions, neglected technological requirements or to respiratory and immunosuppressive diseases. A common sequel of navel infections is local or diffuse peritonitis.



3. When the amount of egg white is bigger (in larger eggs), it impedes the absorption during hatching, resulting in subcutaneous jelly-like oedemas that are an excellent media for the development of *E. coli* infections.



BACTERIAL DISEASES

7. Salpingites are among the commonest causes for death in layer hens. *E. coli* penetrates from the cloaca via an ascendant route. Predisposing factors are the intense egg laying and the associated estrogen activity.



8. Salpingitis.

In older cases, the caseous masses in the oviduct have a lamellar structure. *E. coli* organisms are usually found in excreta because of their presence in avian and mammalian intestine, the birds are constantly at risk of infection through contaminated water, dust, faeces and environment.



9. Salpingitis.

Retained yolks among the caseous masses in the oviduct. In some cases, when the systemic resistance is lower, places, contaminated with *E. coli*, such as intestine, genital tract or nasal passages, could be latent sources of infection.

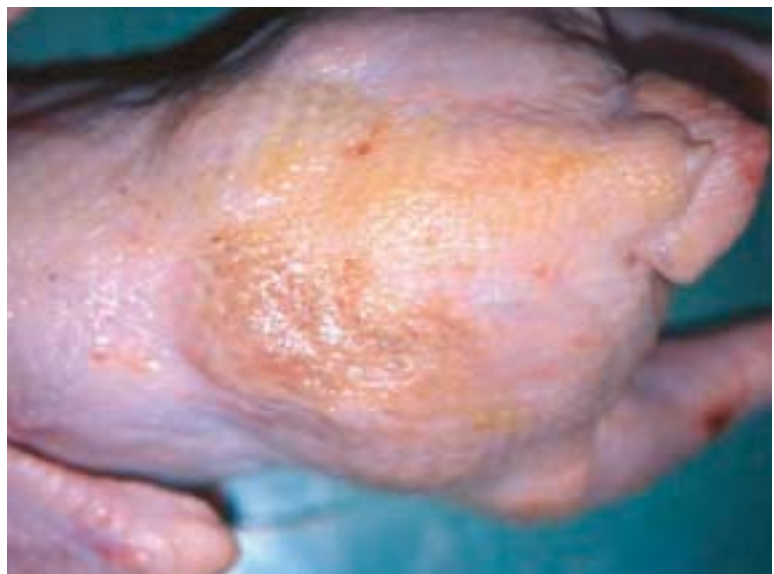


13. Oophoritis
(inflammation of the ovary)
consequently to a salpingitis
due to ascendant *E. coli*
infection

14. Cystic degeneration of ovarian follicles following an *E. coli* oophoritis.



15. Cellulitis (inflammation of the subcutaneous tissue that affects also the overlying skin). It predominates in broilers and is detected mainly in slaughterhouses. Macroscopically, the lesions are with a yellowish-brown colour.





19. In some cases with adult birds, in the region of the head, subcutaneous masses of thick serofibrinous exudate resulting from a local *E. coli* infection could be detected.

20. Enterocolitis.

Enterotoxigenic *E. coli* that produce toxins, cause the secretion and retention of fluids in some intestinal loops and especially in the caeca. Clinically, diarrhoea and dehydration are observed. The intestines are pale and distended, particularly the caeca that are overfilled with fluid containing many gas bubbles.



SYSTEMIC E. COLI INFECTIONS



21. Neonatal *E. coli* septicaemia. Chickens in the first 24 - 48 h after hatching are affected. The death rate during the first ten days is higher and could reach 5 - 6%. The yolk sac is unabsorbed. The spleen is enlarged. Some days later, the typical serofibrinous polyserositis lesions, affecting the peritoneum, the pericardium, the air sacs and the liver capsule are manifested.



26, 27. *E. coli* septicaemia secondary to enteritis.

It is most commonly encountered in turkeys. The intestinal mucosa, damaged by the haemorrhagic enteritis virus (see Adenovirus infections), is the entrance door of *E. coli* infection. The most typical lesions are the marked enlargement, hyperaemia, haemorrhages and necrosis of the liver and the spleen.



28. Panophthalmitis (inflammation of all tissues of the eyeball). Generally, it develops secondary to *E. coli* septicaemia and is usually unilateral.





32. The lesions that develop in the articular spaces of thoracolumbar vertebrae result in spondylitis (spondylosis) and after that, in progressive paresis and paralysis.



33. **Coligranuloma** (Hjarre's disease). It is characterized by multiple granulomas in the intestinal tract, the mesentery and the liver, but not in spleen. The lesions are similar to these observed in tuberculosis.

34, 35. **Bursitis sternalis** (inflammation of the sternal bursa). The bursa is enlarged in a various extent and filled with inflammatory exudate. **The diagnosis** of coli - infections is based on isolation and typization of pathogenic *E. coli* serotypes. Many other bacteria (salmonellae, pasteurellae, staphylococci etc.), viruses, chlamydiae and mycoplasmae should be excluded as possible aetiological agents. **The prevention** should aim at minimizing the probability of faecal contamination of eggs. This implies the maintenance of clean nests, discarding floor eggs and removal of eggs that are cracked or contaminated with faeces. Breeder eggs should be fumigated or disinfected in the farm prior to their transportation in the storage premise. **The treatment** is effective if initiated soon after testing the antibacterial sensitivity of isolates.





38. The oedema of tibiotarsal joints is a frequent associated sign. Pullorum disease is widely distributed among all age groups of chickens and turkeys. The highest losses are in birds under the age of 4 weeks.



39, 40. The aetiological agent is *S. pullorum*, a non-motile Gram-negative microorganism. *S. pullorum* is very resistant under moderate climatic conditions and could survive for months. It could be killed by fumigation with formaldehyde of breeder eggs in the hatchery. Typical for this form are the greyish-whitish nodes in one or some of the following places: heart (39), lungs, liver, gizzard walls (40) and intestines, the peritoneum.



FOWL TYPHOID

Fowl typhoid is an acute or chronic septicaemic disease that affects primarily adult hens and turkeys.



43. Acute fowl typhoid.

The outbreaks usually begin with a sharp decline in forage consumption and egg production. The fertilization and hatchability rates are considerably reduced. Diarrhoea appears. The death rate in acute fowl typhoid is high and varies between 10% and 90%. About 1/3 of chickens hatched from eggs from typhoid-infected flocks die. A characteristic lesion for acute fowl typhoid in adult birds is the enlarged and bronze greenish tint of liver.



44. Acute fowl typhoid.

In some instances, the enlarged liver is mottled with multiple millimetric necroses. The outbreaks are observed primarily in hens and turkeys, but the disease is sometimes encountered in other domestic or wild fowl.



45. Acute fowl typhoid.

In other cases, the size of liver necroses varies from millimetric to spots with a diameter of 1 - 2 cm. Unlike pullorum disease, fowl typhoid is lasting for months.



49. Acute fowl typhoid.

The lungs acquire a characteristic brown colour. Here, necroses and, following their organization, „sarcoma-like nodules” could be observed.

50. Chronic fowl typhoid.

The lesions are primarily in the gonads. The ovaries are affected by inflammatory and degenerative changes.



51. Chronic fowl typhoid.

Frequently, affected follicles are deformed and appear like thick pendulating masses. Fowl typhoid should be differentiated from other salmonellosis, *E. coli* infections, *Pasteurella spp.* infections etc. If breeder flocks are proved to be carriers of the infection, their eggs should not be used for breeding.



55. The inflammatory fibrinous exudate in caeca often forms casts with the shape of mucosal folds. The aetiological agents are about 10 - 15 *Salmonella* serotypes and the most common isolates are *S. Enteritidis* and *S. Typhimurium*. Most fowl paratyphoid organisms contain an endotoxin, responsible for their pathogenic effects.

56. Sometimes, necrotic foci in the liver are discovered. The infection of small chickens occurs by penetration of microorganisms into the egg after faecal contamination. The transmission of agents could be done also by a contaminated source of animal protein (meat and bone meal etc.). The rodents are a significant reservoir of paratyphoid microorganisms. The treatment inhibits but does not eradicate the infection. The appropriate treatment minimizes the death rate until the birds develop immunity.





60. A subcutaneous haemorrhagic oedema in the region of the neck about the site of MD vaccine application.

P. aeruginosa is widely distributed in the soil, water and the environment. The high humidity favours its development. Susceptible avian species are chickens, turkeys, pheasants, ducks, goose, ostriches and exotic birds.

61. Sometimes, subcutaneous haemorrhages in the muscles are present.



62. In the liver, hyperaemia, subcapsular haemorrhages and dystrophy are detected.

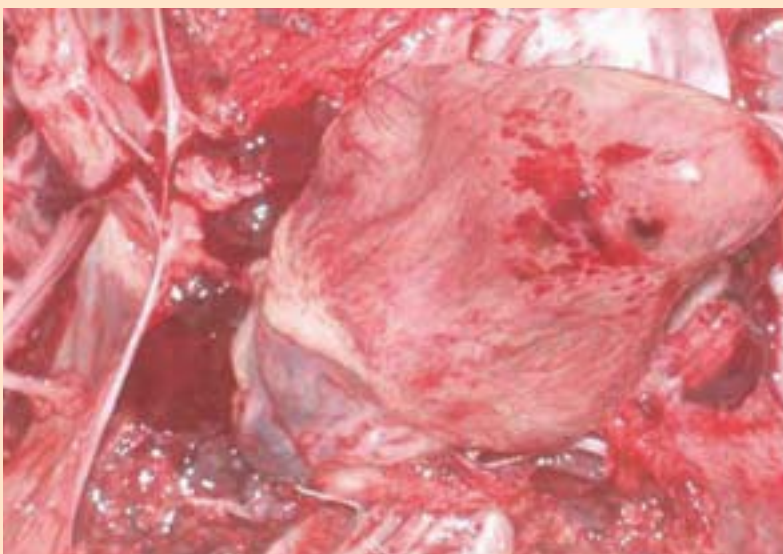




66, 67. Arthritis and periarthritides are encountered in broilers as well as in growing birds. Tibiotarsal joints are most commonly affected. The skin and the mucous coats are the entrance door of the infection. The prevention is based upon detection and elimination of the causative agent. The strict hygiene in hatcheries and throughout the injection of birds is essential for the prevention of *Pseudomonas* infection. The test of isolates' sensitivity is of most importance for the treatment, because the microorganism is resistant to a high number of antimicrobial drugs.



FOWL CHOLERA



68, 69. Fowl cholera is an infectious disease in domestic fowl, waterfowl and other avian species. It is manifested either in acute septicaemic form with a high morbidity and death rates or as chronic local forms (independently or secondary to acute ones). **Acute fowl cholera.** The sudden and unexpected death could be the first sign of the disease. In this form, the lesions are predominantly related to vascular injuries.



73. In layers (commercial or breeders), acute oophoritis with regressing follicles and consequently, diffuse peritonites are commonly observed.



74. Chronic fowl cholera.
It is characterized by local inflammations. The periorbital sinuses are frequently affected by a serofibrinous inflammation.





78. The fibrinous caseous exudate accumulated in wattles sometimes leads to gangrene of the covering skin.

79. In turkeys, a common finding is the unilateral or bilateral croupous pleuropneumonia.



80, 81. The inflammation could possibly be spread from sinuses to adjacent air-filled skull bones with subsequent necrosis and onset of neurological signs (opisthotonus and torticollis). The diagnosis is made on the basis of disease history, clinical signs, the lesions and the results of bacteriological studies.



STAPHYLOCOCCAL INFECTIONS



84, 85. *Staphylococcus aureus* infections are commonly seen in poultry. Usually, bones, tendon sheaths and joints, particularly coxofemoral and tibiotarsal joints are affected. The clinical signs include unilateral or bilateral lameness, reluctance to move and lying down. When the tibiotarsal joints are affected, swellings, fever and sometimes necroses of overlying tissues and purulent exudation are observed.



86, 87. As a sequel to septicaemia, osteomyelitis could occur. The lesions are usually detected in the region of the proximal femur, where inflammatory necrotic foci in the bone marrow and partial or complete fracture of the femoral head are observed.





91, 92. Other local forms of staphylococcosis are sternal bursitis. The sternal bursa is enlarged at a various extent because of gathering of purulent substance. Sometimes, the covering skin is necrotized. The microorganism is widely distributed in the environment and mainly on the skin. Most of *Staphylococcus aureus*-induced lesions are associated with skin injuries, debeaking, finger cuts. All categories of birds are affected. Toxicogenic strains are able to induce food intoxications.