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# Economically Important Infectious Causes of Enteritis in Poultry

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## Abstract

Enteric infections in poultry pose a threat to intestinal health and can contribute to poor feed efficiency and livability of a flock. A variety of enteric bacterial diseases are recognized in poultry. Necrotic enteritis (NE) produced by *Clostridium perfringens* (*C. perfringens*) is amongst the most prevalent enteric diseases of chickens and turkeys. However, several other bacterial, parasitic and viral agents can cause clinical signs, gross and microscopic lesions in poultry very similar to those of NE and the diseases produced by those agents need to be differentiated from NE. The main differential diagnoses for *C. perfringens* NE include bacterial (*C. colinum*, *C. sordellii*, *C. difficile, P. multocida*, *Brachyspira* spp.), parasitic (*Eimeria* spp., *H. meleagridis*) and viral (*Duck Herpes* virus type 1, *Avian Paramyxovirus* type 1) diseases. Confirmation of diagnosis of these diseases requires identification of the etiological agents by morphological, cultural and/or molecular methods. Prevention depends mainly on good diagnosis and differential diagnosis for choice of suitable disinfectant, medication, improve hygiene and application of preventive vaccination by commercial vaccine prepared from related strains or autogenous prepared vaccine in endemic farms or area.

This review aimed to collect available data about infectious causes of enteritis in poultry to be available to be available to students, researchers and veterinarian in poultry practical.

**Keywords:** Poultry; Birds; Necrotic Enteritis

## Introduction

Enteric disease can dramatically affect production in poultry, mainly by causing poor feed efficiency and loss of birds, both of which increase production costs [1]. Necrotic enteritis (NE) by *C. perfringens* is one of the most prevalent and severe enteric diseases of poultry [2]. NE is characterized primarily by distension of the small intestine with watery, mucous and/or fibrinous content, with necrosis of the mucosa and, occasionally, deeper layers of the intestine. The lesions can also involve caeca and colon [2]. NE is a toxic infection and is characterized by hemorrhagic enteritis, high morbidity, and mortality, resulting in annual billion-dollar losses [3]. NE can occur as primary infections as well as secondary infections in immuno-compromised birds [4]. Immunosuppression by viral diseases and intestinal erosions caused by coccidia increases the risk of NE [5]. NE has increased in occurrence and severity over the years. The re-emergence of NE has been the most significant threat for the poultry industry, which, in clinical form, causes high mortality and in subclinical forms, affects growth and feed conversion. It is one of the most common and economically devastating bacterial diseases in modern broiler flocks in terms of performance, welfare and mortality. According to estimation, NE losses increased to approximately US$6 billion in 2015 as compared to US$2 billion in 2000 [6]. NE

is a multi-factorial disease process, in which a number of co-factors are usually required to precipitate an outbreak of the disease. Although, *C. perfringens* has been identified as the etiological agent of the disease, the predisposing factors that lead to over-proliferation of *C. perfringens* and the subsequent progression to disease are poorly understood. The onset of NE is associated with a shift in the microbiota present within the gastrointestinal tract (GIT) [7]. However, NE-like disease in poultry can be caused by a wide variety of infectious agents, and differentiation between *C. perfringens* NE and other enteric diseases is critical to diagnose, differentiate, prevent and treat these diseases. This paper reviewed the most significant enteric diseases of poultry that need to be differentiated from *C. perfringens* NE.

## Viral Diseases 1

Duck viral enteritis (DVE) is caused by the alpha herpes virus anatid herpesvirus-1. DVE is an acute disease of ducks, geese, swans and other waterfowl [8]. Waterfowl of all ages and coots are susceptible. The disease is characterized by weakness, diarrhea and high mortality. Transmission of the virus is by the oral route either by contact with infected birds and/or contaminated environment. The main lesions are due to vascular damage and include hemorrhages in heart, pancreas, kidneys, thymus, coelomic cavity, gizzard, liver and intestine. The latter frequently shows typical annular bands of hemorrhage affecting the lymphoid tissue). In addition, fibrinonecrotic inflammation can be observed in the esophagus, caeca, colon, rectum, cloaca and bursa of Fabricius. Microscopically, eosinophilic inclusion bodies can be seen in the nucleus and/or cytoplasm of epithelial cells of the esophagus, small and large intestine, pancreas and kidneys, in hepatocytes, and in the mononuclear cells of thymus, spleen and bursa of Fabricius. DVE must be distinguished from AI, avian cholera, ND and NE. A presumptive diagnosis of DVE can be made based on histopathology, but confirmation of the diagnosis must be based on virus isolation and/or PCR.

### Avian Paramyxovirus Type 1

Virulent Newcastle Disease (VND) is an acute, highly contagious systemic disease of most species of birds, caused by virulent strains of avian paramyxovirus type 1 (APMV-1) [9]. Chickens, turkeys, quail and other gallinaceous birds are highly susceptible, but ducks and geese are relatively resistant. Diarrhea may occur, accompanied or not, by a variety of signs and high mortality. Lesions of VND include subcutaneous edema in the head, neck, and abdomen as well as hemorrhages and/or necrosis and inflammation in the conjunctiva, comb, wattles, oral cavity, pharynx, esophagus, larynx, trachea, lungs, crop, proventriculus, intestine, gonads, bursa of Fabricius, thymus, spleen and bone marrow. In the intestine the lesions range from necrosis of lymphocytes with hemorrhages and ulcerations in the Peyer's patches and caecal tonsils to severe hemorrhagic enteritis throughout the small and large intestine including rectum and cloaca. VND can be tentatively diagnosed based on clinical signs and lesions combined with serology in unvaccinated birds. Immunohistochemistry can be used to detect APMV-1, but does not discriminate highly virulent from low virulence forms. Confirmation of the diagnosis should be based on detection of highly virulent APMV-1 by virus isolation and/or PCR, Isolation of virus and serological diagnostics, such as HI Test, ELISA and molecular diagnostic tests like real time PCR confirmed the presence of VND virus [10].

## Bacterial Diseases

### Clostridial Species:

a. **Clostridium perfringens (C. perfringens):** Necrotic enteritis of poultry is caused by toxigenic *C. perfringens* Type A or C. Alpha toxin, produced by *C. perfringens* Types A and C, has been shown to produce the enteric lesions of necrotic enteritis when administered to broiler chicks [11]. *C. perfringens* also produces beta toxin that may play a role in necrotic enteritis [12]. Outbreaks of NE in poultry are most common in broiler chickens, but the disease has also been reported in commercial layers raised on the ground [13], cage reared replacement pullets [14], and turkeys [15]. *C. perfringens* may often be a normal inhabitant of the intestinal tract [16] and other factors promote microorganism overgrowth and toxin productionin the gut. For example, necrotic enteritis is often preceded by or associated with enteric coccidial infection [17]. Also, high levels of wheat or fishmeal in the ration may predispose birds to develop ND [18,19].

Clinical signs described with ND include depression, ruffled feathers, diarrhea, huddling, anorexia, sternal recumbency, and a sudden rise in flock mortality (Long, 1973) [20]. Gross lesions are usually restricted and ileum and less often the duodenum and cecum. The intestine is distended by gas and dark brown fluid. A discontinuous to diffuse layer of tan to grey, friable fibrinonecrotic material (pseudomembrane) is adhered to the mucosa. Rare focal lesions may be observed [17].

Histopathologic evaluation of affected intestine reveals the fibrinonecrotic material to consist of necrotic villi, fibrin, and necrotic inflammatory cells. Large Gram-positive bacterial rods with or without other bacteria are usually scattered throughout the areas of necrosis. A sharp line of demarcation occurs between the necrotic and viable tissue [21]. Electron microscopic evaluation of lesions indicated that *C. perfringens* was intimately associated with the inflammation, but the microorganism did not invade viable tissue. These findings

were consistent with enterotoxin release playing a primary role in lesion pathogenesis [22].Diagnosis of *C. perfringens* is challenging, because many clostridial species can be normal inhabitants of the gut. Diagnosis is based on clinical and pathological findings, negative culture and toxin detection [23]. Various PCR protocols including multiplex PCR assays have been established to genotype *C. perfringens* isolates with respect to cpa, cpb, etx, itx genes encoding the alpha, beta, epsilon and iota toxins, respectively [24].

**b. Clostridium colinum (C. colinum):** Ulcerative enteritis (UE) produced by *C. colinum*, also known as quail disease, has been described in many avian species but is most common in quail [2]. Bobwhite quail is the most susceptible species [2]. UE is a highly fatal enteric disease that primarily affects captive quail, but the disease has also been reported in other birds including chickens, turkeys, and pheasants [25,26] . Young quail, from 4 to 12-wk-old, are most susceptible. Quail may die in 1 to 3 d after experimental infection [27]. Experimental infections in susceptible chicken with *C. colinum* alone have been produced in quail [28]. UE is more frequently seen in young birds of many avian species [2]. It has not been possible to experimentally reproduce UE in chickens, which raises questions about what other factors (e.g., feed, crowding, other infectious agents) act to promote the disease in poultry [29]. Birds may die in good physical condition without premonitory signs, whereas others may appear weak and depressed with ruffled feathers and diarrhea. Intestinal lesions are widespread throughout the duodenum, jejunum, ileum, and cecum and consist of multifocal to coalescing 1 to 5 mm diameter white punctate mucosal ulcers that can usually be observed on the serosa). The ulcers often perforate the intestinal wall to produce peritonitis and intestinal adhesions [27]. Clinical signs include sudden death or watery to bloody Diarrhea, depression and listlessness. Emaciation is common in chronic cases. Often there is an underlying coccidial infection associated with UE. Enteric lesions in quail are located mainly in the duodenum and also in other parts of the intestine and consist of large, round, yellow ulcers surrounded by hemorrhages that can be seen from the serosa. The ulcers may coalesce to form larger, sometimes perforating ulcers that lead to coelomitis. Multifocal hepatic necrosis can also occur [2]. A presumptive diagnosis of UE can be based upon clinical signs, and gross and microscopic lesions. The presence of large Gram-positive rods with sub-terminal spores in liver, spleen, and/or intestinal smears or histological sections strengthens a presumptive diagnosis. Confirmation of the diagnosis must be based on isolation of *C. colinum* from liver, intestine, and/or spleen. Differentiation from NE, coccidiosis and histomoniasis may be difficult as there may be significant overlapping in lesions [2].

**c. Clostridium sordellii (C. sordellii):** In birds, *C. sordellii* has been reported to produce gangrenous dermatitis in chickens and turkeys [30], and enteritis and hepatitis in ostriches [31], as well as UE of quails [32]. A disease clinically and pathologically identical to NE in commercial broiler chickens was due to *C. sordellii* [33]. Birds affected with *C. sordellii a*ssociated NE were between 18 and 26 days of age and increased mortality, depressed birds and diarrhea were the main clinical abnormalities [34]. Gross and microscopic lesions were identical to those observed in NE [34,35]. The pathogenesis of NE-like lesions caused by *C. sordellii* is still unclear, but as with *C. perfringens* NE, coccidiosis was the main predisposing factor in the few cases was described by Rimoldi et al [34]. C. sordellii-associated NE was diagnosed based on gross and microscopic findings coupled with anaerobic culture and immunohistochemistry [34].

**d. Clostridium difficile (C. difficile):** NE associated with *C. difficile* has been described in newly hatched ostriches [36]. Clinical signs include acute onset of Diarrhea and death with mortality rates as high as 90%, while, lesions include fibrino-NE and typhlocolitis. Disseminated hemorrhagic lesions and multifocal liver necrosis are also seen [36]. The diagnosis can be confirmed by isolation of *C. difficile* and detection of toxin A and/ or B in intestinal content or fasces [2].

**e. Pasteurella multocida (P. multocida):** *P. multocida* causes an acute systemic disease called fowl cholera in poultry and avian cholera in wild waterfowl [37]. The disease is characterized by an acute onset with little or no clinical signs and high mortality, especially in turkeys and waterfowl. A chronic form of the disease also occurs. *P. multocida* is transmitted most commonly through the respiratory tract, and the disease is mediated by bacterial endotoxins. In waterfowl, *P. multocida* can cause intestinal lesions that range from mucous-distended intestine to severe fibrinonecrotizing enteritis. Diagnosis of *P. multocida* infection must be confirmed by culture from organs with lesions, including, amongst others, the intestine [38]. PCR assay has been developed for capsular typing of *P. multocida* strains. This assay represents a rapid and reproducible alternative to serological methods [38].

### *Salmonella* Spp

Salmonellosis is a group of acute or chronic diseases of fowl, turkey, ducks, pigeon and other birds caused by one or more members of the genus Salmonella. It is of economic importance to the poultry industry. Domestic fowl is the largest reservoirs of Salmonella as a risk to public health through consumption of contaminated eggs and meat. Four diseases caused by Salmonella are in poultry; pullorum

disease caused by *Salmonella enterica* serovar Pullorum, fowl typhoid caused by *S. Gallinarum*, paratyphoid caused by several motile serovars and subspecies of Salmonella ( *S. Enteritidis, S. Typhimurium and S. Heidelberg*) and arizonosis caused by *S. enterica* subsp. Arizona [39].

- **Pullorum disease (PD) and fowl typhoid (FT)**

*S. Pullorum* and *S. Gallinarum* are the aetiologic agents of PD and FT, respectively. These two salmonellas are now considered one single serovar, *Salmonella enterica* subspecies enterica serovar Pullorum–Gallinarum of the Enterobacteriaceae family. Both S*. Pullorum* and *S. Gallinarum* are in the serogroup D of salmonellas together with *S. Enteritidis*, and are very well adapted to chickens and turkeys. PD and FT occur worldwide [40,41], although they are currently uncommon in the developed world. PD and FT occur naturally in chickens, turkeys, quail, pheasants, guinea fowl, sparrows and parrots [42]. *S. Pullorum* and *S. Gallinarum* are transmitted both vertically and horizontally [40,41]. In PD, high mortality usually occurs mainly in birds during the first 2–3 weeks of age; outbreaks originating from vertical transmission can beoccasionally asymptomatic [40,41]. In FT, mortality occurs in young and mature birds, and some birds that survive the infection may become *S. Pullorum* asymptomatic carriers. The mechanism by which *S. Pullorum* and *S. Gallinarum* cause disease in susceptible birds is not completely understood, although it is thought to be related to the ability to produce endotoxin and survive in macrophages [43]. Chicks with peracute PD or FT may not have gross lesions. In acute cases, congestion, enlarged liver, spleen, and kidneys and retained yolk sacs with caseous content may be observed. Acute cases of PD and FT are characterized by multifocal liver necrosis, fibrinous splenitis, polyserositis, synovitis, necrosis of myofibers with pleocellular inflammation in heart and gizzard. In young birds fibrino-necrotic typhlitis is common.

- **Paratyphoid**

Infections caused by motile salmonellas in poultry are called paratyphoid infections. *S. enterica* subspecies enterica serovar Typhimurium (*S. Typhimurium*) is motile by peritrichous flagella and belongs tothe serogroup B. *S. Typhimurium* infection has worldwide distribution and is one of the most common causes of paratyphoid in poultry [44,45] . However, *S. Typhimurium* rarely causes systemic disease in chickens [46]. Mortality associated with natural *S. Typhimurium* infection is much higher when infection occurs at 1 day of age than at later age. Newly hatched poultry infected with *S. Typhimurium* may have septicaemic-type lesions, including unabsorbed yolk sacs with caseous material and necrotic duodenitis and typhlitis. Necrotizing hepatitis and splenitis, enlarged kidneys, perihepatitis, pericarditis, oophoritis, salpingitis, omphalitis, peritonitis, pneumonia, fibrino-necrotic typhlitis, hypopyon, synovitis and polyserositis can be found [47,48].

- **Avian arizonosis**

This is an acute or chronic septicaemic infection of poultry caused by *S. enterica* subspecies arizonae (*Salmonella Arizona*). Avian arizonosis is more common in turkey poults, but chickens, ducks, canaries and parrots are also susceptible [40,41,49] . The pathogenesis of Salmonella Arizona infection is unknown, but it may be similar to infection by other salmonellas [50]. Lesions produced by Salmonella Arizona in poults are similar to the lesions including fibrinonecrotic typhlitis caused by other motile salmonellas, although encephalitis and ophthalmitis are more common [40,41].

- **Colibacillosis**

Colibacillosis is an infectious disease affecting a wide variety of birds in which *E. coli* is a primary or secondary pathogen. *Escherichia coli (E. coli)* is a Gram-negative medium-sized (2 to 3 mm long) rod that is widespread in nature and is a normal inhabitant of the intestinal tract of poultry [51]. Pathogenic serotypes of *E. coli* can often be isolated from the intestinal tracts of healthy poultry, which supports the claim that *E. coli* is often a secondary or opportunistic pathogen. Feces and dust in the poultry house are an important source of pathogenic *E. coli* [51]. Both ingestion and inhalation of *E. coli* are potential routes of infection. A wide variety of pathogenic *E. coli* have been identified by somatic O antigen serotyping, but the most common are Serotypes O1, O2, and O78. Many pathogenic *E. coli* isolates are not among the established serotypes and are classified as untypeable [53]. The major disease syndromes in colibacillosis of poultry are yolk sac infection, respiratory disease complex (airsacculitis, perihepatitis, and pericarditis), acute septicemia, salpingitis, peritonitis, synovitis, osteomyelitis, cellulitis, and enteric coligranuloma. Young birds with little resistance to infection will acutely die from septicemia, but older chickens are often resistant and survive the initial septicemic lesions; however, *E. coli*-associated septicemic peritonitis in adult laying hens can cause significant mortality (personal observation).

Coligranuloma is a rare condition that may be observed in adult chickens that are found dead and in poor physical condition. Gross findings in coligranuloma consist of firm, white to yellow nodules in the mesentery and wall of the intestine [52]. The nodules are granulomas from which *E. coli* may sometimes beisolated. Coligranuloma can be differentiated from mycobacteriosis by microscopy and bacterial culture.

- ***Brachyspira* spp**

Brachyspira spp. can colonize caeca and less frequently the colon of chickens, pheasants, turkeys, partridges, ducks, geese and rheas [54,55]. The most common species of Brachyspira involved in enteric infection of these birds are

*Brachyspira pilosicoli (B pilosicoli)* and *B intermedia*; less commonly *B alvinpulli* and *B hyodysenteriae* are involved in these enteric infections. Brachyspira spp. are anaerobic, flagellated and helically shaped (spirochaete) bacteria. Brachyspira spp. have been associated with delayed onset and/or reduced egg production, and wet faeces in chickens, catarrhal typhlitis in turkeys, necrotizing typhlocolitis in ducks and geese and fibrino-necrotic typhlitis in rheas Silver stains and/or immunohistochemistry can be used to demonstrate the presence of spirochaetes in the intestine Brachyspira spp. can be isolated from caeca and colon and identified by PCR [55].

### Parasitic Diseases

- **Histomonas meleagridis (H. meleagridis)**

Histomoniasis, commonly called blackhead or enterohepatitis, is a disease of many species of gallinaceous birds caused by *H. meleagridis* [56] that has worldwide distribution [57]. Because of the ban of chemotherapeutic substances and an increase in free-range poultry production, histomonosis is considered a re-emerging disease [58], which is able to cause mortality up to 100% in turkey flocks and 20% or more in chicken flocks [59]. Diarrhea or sudden death is the most significant clinical alterations of histomoniasis. Lesions are characterized by fibrinonecrotizing and/or hemorrhagic typhlitis, which is usually bilateral perforation of the caeca leading to coelomitis may occur [59]. Multifocal areas of hepatic necrosis, with a depressed center circumscribed by a clear ring are present in the liver. Lesions in various other organs such as spleen, kidneys, lungs, pancreas and proventriculus may also be observed in some cases. Bursas of Fabricius affected may contain fibrinous exudate in their lumen [56].

Histomonads can be seen microscopically in areas of necrosis of all organs affected [60]. When typical histomoniasis lesions are present in the liver and in caeca, diagnosis can be made based on gross pathology. However, the lesions can be variable and need to be differentiated from NE, bacterial septicemia, lymphoma, salmonellosis and coccidiosis and NE in chickens. Final diagnosis can be made by histopathology to demonstrate the presence of histomonads in affected tissues [56,59,60] . The presence of live histomonads can also be determined by phase-contrast microscopy on wet mounts) or by culture [59,61,62] Molecular tests are also available [63,64].

- **Eimeria spp**

Avian coccidiosis produced by protozoa of the Eimeria genus is one of the most important enteric diseases of poultry. Coccidiosis affects chickens of all ages and breeds by impairing feed utilization and growth of infected birds. Maximum incidence, however, occurs in 3–6-week-old chicks. Chickens are mainly infected by seven species, which in order of virulence are: *E. tenella, E. necatrix, E. brunetti, E. maxima, E. acervulina, E. mitis and E. praecox.* Clinically, coccidiosis is characterized mostly by hemorrhagic diarrhea and high mortality. The disease spreads from bird to bird by contact with infected faeces via litter, water, feed and other fomites. Coccidiosis is also common in turkeys although lesions are not as prominentas in chickens. Different species of coccidia in chickens affect specific parts of the gut and the type and location of lesions can be used to suggest the species *of Eimeria* involved. *E. acervulina* affects mainly duodenum and upper jejunum, *E. brunetti* causes most damage in the lower ileum, colon and proximal portion of caeca. *E. maxima* and *E. necatrix* infect lower duodenum, the whole jejunum and proximal ileum. *E. tenella* infects primarily the caeca. *E. praecox* and *E. mitis* do not cause significant lesions.

Grossly, *E. acervulina* affected intestine is distended and shows red spots and white bands, E. maxima lesions are characterized by watery, bloody and mucoid content in the intestine. *E. tenella* infected birds show hemorrhagic lesions in the caeca which may be filled with blood and pseudomembranes. Histologically, with all species there might be variable degree of necrosis, villus blunting and coccidian forms invading epithelial and lamina propria cells. The presumptive diagnosis is based on clinical signs and gross lesions.

Confirmation of the diagnosis relies on observation of oocysts in fecal or intestinal wet mounts, or fecal float. Demonstration of oocysts in histological sections of intestine is diagnostic. The main differential diagnoses include NE, UE and histomoniasis. Differentiation of coccidiosis from NE can be challenging in cases of the latter in which coccidiosis was the predisposing factor, as there is some lesion overlap and coccidian oocysts would be present in both diseases.

## Conclusion

Enteritis can be considered as a threat for poultry production due to it causes losses due mortality, low growth rate and high cost of both prevention and control. Prevention depends mainly on good diagnosis and differential for ideal choice of suitable disinfectant, medication for bacterial and parasitic causes as well as good hygiene to prevent entrance and spread of infection. Application of preventive vaccination by using commercial vaccine prepared from antigenic immunogenic related strains or autogenous vaccine in endemic farms or area is of great value.

## Declarations

All data collected in this review are included in this published article.

## Competing Interests

The author declares that they have no competing interests.

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