CEVA HANDBOOK
OF POULTRY DISEASES

3 FOWL CHOLERA

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DEFINITION

Fowl Cholera (FC) is an infectious disease found in domestic fowl, waterfowl and other avian species. It is manifested either in acute septicaemic form with a high morbidity and death rates, or in various chronic local forms (independently or secondary to acute ones).

HISTORY AND SYNONYMS

FC was recognised as a disease in domestic fowl 230 years ago in France by Chabert (1782), although we do have reports of a cholera-like illness in Italy dating from the 17th century. The infectious nature of this disease was established in the 1850s (Hutyra et al. 1949). During the 19th century, the illness spread throughout many Northern European countries. Towards the end of that century it was assumed that the disease had been introduced into Germany via geese and other domestic birds imported from Eastern Europe, yet there are also reports from the same period about a spread of cholera among domestic ducks, swans and geese (Willach, 1895). The same author notes that sparrows and some other bird species were not affected during those epizootics. The cause was found in the blood of hens by Rivolta (1877) and Perroncito (1878), while Tousaint (1879) and Pasteur (1880) cultivated it on nutrient media. The etiological agent *Pasteurella* was named in honour of Louis Pasteur, who attenuated the bacterium and produced the first vaccine for the disease, with the principle of its creation serving as the foundation for producing vaccines against other infectious diseases (Samuel et al, 2008).

In the beginning of the 20th century, FC spread throughout Great Britain and a number of countries in Eastern and Southern Europe. At that time, reports of the disease's spread in South Africa, Australia and New Zealand emerged (Gray, 1913). During this period it was believed that the source of the disease could be wild animals, carriers of the disease through contamination of food at the farms, transmission via infected birds from infected farms to disease-free ones, etc. At the end of the 19th century, the disease was detected in North America (USA and Canada), predominantly during the winter months, whereas in Europe the epizootics occurred mostly between August and October (Hutyra et al., 1949).

The term fowl cholera was used for the first time by Mailet in 1836. Other synonyms include avian pasteurellosis, avian haemorrhagic septicaemia and chicken cholera.

CHARACTERISTICS AND CLASSIFICATION OF THE PATHOGEN

Pasteurella multocida is a non-spore-forming, immobile, Gram-negative bacterium, rod-shaped to coccobacillary. At different periods, the isolates have been named in accordance with their corrsesponding clinical expression and the animal species in which they were isolated, such as *P. avicida* or *P. aviseptica*, and *P. muricida or P. muriseptica* (Heddleston 1972). The name *P. multocida* (from Latin "multo" - many; "caed" - kill) was proposed by Rosenbusch and Merchant (1939). Up until the 1930s, when new variations were proposed, *P. multocida* was the only defined species of the *Pasteurella* genus (Samuel et al., 2008). Since this period, new species, subspecies and other unnamed members of the *Pasteurella* genus derived from birds have been described (Blackall & Mifflin, 2000).

Three subspecies of *P. multocida* have been identified (*multocida*, *septica* and *gallicida*) (Mutters et al., 1985). *P. multocida* subsp. multocida is the most common cause of disease, yet the *septica* and *gallicida* subspecies can also cause cholera-like illness in poultry to a certain extent (Hirsh et al., 1990).

The *gallicida* subspecies is mainly associated with waterfowl. The relation between the *P. multocida* subspecies and serovars, derived from the known serotyping systems, is not clear. Five capsular (A, B, D, E and F) and 16 somatic (1-16) serovars of *P. multocida* are currently known. All of them except serotypes 8 and 13 were isolated from bird hosts (OIE, 1996).

The subspecies *P. multocida* and serovar A, however, have proved to be the subspecies and serogroup most commonly isolated in the most severe cases of cholera (Rhoades & Rimler, 1987; Rhoades, 1989). Some of the 16 somatic serovars were demonstrated among the isolates of serovar A. Isolates, which have multiple somatic antigens, are commonly encountered and considered to be different serotypes. It has been demonstrated that different isolates of the common serovar A: 3,4 varied significantly in terms of virulence (Lee et al., 1988). The virulent properties of the various subtypes from different bird hosts are not yet clear.

The capsule is considered to be the primary virulence factor, yet other factors probably influence the severity of the infections.

Such factors are considered to be: endotoxin, outer membrane proteins, iron-bound systems, heat shock proteins, neuraminidase production (Christensen & Bisgaard, 2000). Apart from endotoxin, other toxins can also play a role in the pathogenesis of cholera. The production of RT X toxins (repeats in toxin), which are of great importance to the pathogenesis of infections caused by some members of the Pasteurellaceae family, have not been observed in *P. multocida*.

During the acute phase of the infection, *P. multocida* can be isolated from the blood and the internal organs (heart, liver, spleen, etc.). It has been experimentally established that the bacterial load in the blood progressively increases between the 4th and 20th hour after intravenous inoculation of turkeys, with concentration peak by the 16th–20th hour. These results make it reasonable to assume that the ability for extracellular replication and the occurrence of septicaemic lesions are dependent on the strain's virulence (Prantner et al., 1990).

Other factors can also affect the illness' development. Even though it is believed that most bird species are vulnerable to *P. multocida* infection (the organism has been isolated from more than 100 different bird species), they differ considerably in their susceptibility to the pathogen (Botzler, 1991). Among domestic fowl, turkeys are probably the most susceptible. Considering the high losses, waterfowl also seem to be very sensitive, whereas chickens appear more resistant. This was proven through experimental infection of several bird species using the original *P. multocida* isolate from eiders (Christensen et al., 1998).

Other factors with reported impact on the course of the illness include overpopulation, climate, nutritional stress, concurrent diseases, host age etc. Age definitely has an effect on the course of infection, especially in chickens, as birds under 16 weeks of age are relatively resistant. In natural conditions, the mortality rate can vary from a few per cent to close to 100%, depending on the factors listed above (Christensen & Bisgaard, 2000).

The pathogenicity of the different strains of *Pasteurella* varies within a wide range. The isolates from cases of acute cholera were strongly pathogenic for birds as well as for mice and rabbits. Strains with reduced pathogenicity can be isolated from chronic FC. The strains' virulence is related to their ability for encapsulation. After this ability is lost, the strains lose their virulent properties as well. Old laboratory strains lose their pathogenicity for

birds. *Pasteurella* isolated from other animal species are not pathogenic to birds (Obreshkov et al., 1978).

P. multocida is a microorganism with a size of 0.2-0.4 x 0.6-2.5 µm. On Wright - or Giemsa-stained smears, well - formed bipolar rods can be identified. The optimal cultivation temperature is 35–37°C. Pasteurellae are aerobes or facultative anaerobes. Even though the serotyping of the capsular and somatic antigens was proven to be very useful in determining and identifying this bacterium, there is limited potential for retrieving useful information in epidemiological studies regarding the distinction between different strains of a same serotype. Molecular subtyping techniques have also been used for the subtypification of *P. multocida,* including restriction endonuclease analysis, plasmid profiling and ribotyping (Amonsin et al., 2002).

According to this latter research team, restriction endonuclease analysis and ribotyping are of limited use because they are so time-consuming. Plasmid profiling is a relatively easy and cheap method, yet it has limited application in epidemiological studies because not all isolates can bear plasmids, or may lose them during cultivation in laboratory conditions (Swaminathan & Matar, 1994). With regard to these circumstances, two PCR-based access systems have been proposed for the differentiation of *P. multocida* at the subspecies level from isolates obtained from different bird species after an FC outbreak, namely repetitive sequence-based PCR (rep-PCR) and amplified fragment length polymorphism (AFLP). The results obtained with these techniques shows that they are useful for detection of DNA from *P. multocida* isolates, that they can definitely be used for gene typing, and that they allow quick and easy analysis. Moreover, the data present evidence for the host specificity of some *P. multocida* clones (Amonsin et al., 2002).

P. multocida usually infiltrates bird tissues through the mucous membranes of the pharynx and the upper respiratory tract, though it may also penetrate through the conjunctiva or broken skin. Experimentally, infection can be reproduced through the pharyngeal mucosa but not through the oesophagus, the crop and the proventriculus (Arsov, 1965).

The pasteurellae are fairly sensitive to environmental conditions and the effects of common disinfectants. During the winter, they can survive

in the soil outside buildings for up to 26 days, in litter for up to 72 days, in grain crops for up to 30-40 days, and in water for up to 25 days. During the summer the period of survival is shorter, up to 14 days in the soil, up to 17 days in litter, and up to 11 days in water. Disinfectants such as 3% sodium hydroxide, 1% formaldehyde, 1% phenol, 1% glutaraldehyde inactivate the organism. In light of this, after the infection has been eradicated, the environment cannot be considered to be a reservoir of infection for any longer than the periods listed above.

EPIDEMIOLOGY

FC is widespread, affecting birds from all poultry-producing regions around the world, as well as many wild species. Nevertheless, from an epidemiological perspective, certain question marks remain. Basic aspects, such as the path of the infection's infiltration into the flock, are not yet sufficiently clarified (Christensen & Bisgaard, 2000). Even in the early stages of research it was believed that FC had a contagious character and that infection had to be introduced from outside in a flock in order for an outbreak to occur. It was later postulated that carriership of pasteurellae exists among birds of all flocks, and under certain unfavourable conditions an outbreak of the disease can occur. This theory is supported by the fact that growing birds reared in isolation from adult infected flocks did not exhibit cholera, despite the effects of some detrimental factors on the overall condition of the birds. It has been established that there were no *Pasteurella* carriers in healthy flocks, yet the source of infection is very difficult to locate in some cases, especially with chronic local forms of the disease (Obreshkov et al., 1978).

The primary source of infection is the environment, contaminated by sick birds. It is assumed that the chronically ill act as reservoirs of the infection. Previoulsy affected flocks may remain carriers of *Pasteurella* for anywhere from a few months to a whole year. Infectious agent

emission occurs primarily through nasal secretions, rarely via the faeces, thus contaminating the environment, the food and the water. Cannibalism is another transmission route. Rodents at the farm also play a role in the spread. Various mammals may act as carriers, and the pathogen has been isolated from the mouths and tonsils of dogs and cats living on farms. However, not all types of *P. multocida* found in mammals will cause illness in birds. It is reported that only strains isolated from swine are pathogenic to birds, and thus pigs may play a major role in maintaining and spreading the infection (Iliev et al., 1963a; 1963b). The spread of the infection can occur through contact when personnel, equipment, feed, etc. are moved. Aerosol transmission is possible as well.

Transmission cannot occur through breeding eggs. Pasteurellae can be isolated from the surface of the eggshell, yet they perish quickly during storage, and especially during incubation, within 3–7 days. The potential contact between growing and formerly affected adult flocks, i.e. the presence of mixed-age flocks at the farm, is a contributing factor to the persistence and spread of the infection.

Free-flying birds that have contacts with infected farms can also be a source of FC organisms.

CLINICAL SIGNS AND PATHOLOGY

Depending on the virulence of the infective *P. multocida* strains, the host's susceptibility and the effect of environmental conditions, a wide variety of clinical signs can be observed. Pasteurellosis in fowl is exhibited in acute septicaemic form and chronic local forms.

The acute form is characterised by high morbidity and mortality rates. Sometimes death comes before the symptoms are fully developed. Signs that can be observed include: general malaise, diarrhoea, dyspnea, oral and/ or nasal discharges. Cyanosis of the comb and the wattles is also possible.

The chronic form can be the result of an infection with low-pathogenicity strains or a resurgence of historic acute cholera. It is expressed as

local infections in the area of the wattles, the joints, the sternal bursa and the sinuses of the skull. When the latter are affected, torticolis is possible. Sinus inflammation is also accompanied by catarrhal purulent conjunctivitis, nasal discharge and wheezing. These conditions can persist for a long time, with some of the affected birds dying, while others survive but remain carriers of the infection. There have also been reports of the oviduct being affected in waterfowl (Bisgaard et al., 1995) and dermal necroses in turkeys (Glass & Panigrahy, 1990; Frame et al., 1994). Post-mortem findings in acute cases are dominated by septicaemic lesions, mainly vascular disorders, manifested as haemorrhages, congestion and passive hyperaemia in different parts of the carcass. Petechial and ecchymotic haemorrhages are often found on the epicardium, mesenterium and the subserous surfaces of the thoracic and abdominal cavities. The lungs are often affected, especially in turkeys, where croupous pleuropneumonia with diffuse fibrinous deposits are commonly found. After protracted illness, the inflammation can become proliferative, leading to strand formation between the lungs and the costal pleura (adhesions and synechiae). In such cases, heterophilic infiltration isseen at the microscopic level.

The lesions of chronic cholera occur alone or as combination of pathomorphological expressions. They are detected as fibrinous caseous inflammation of the wattles and the ears, sinusites, arthrites, and dermal necroses in turkeys.

ACUTE FOWL CHOLERA

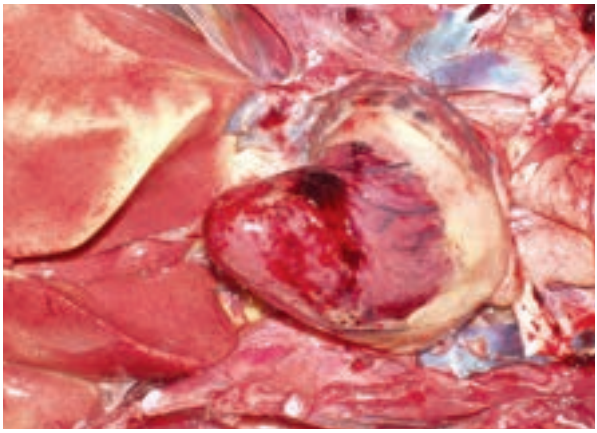

Fig.1
Multiple subepicardial petechial haemorrhages affecting the heart are a typical finding.

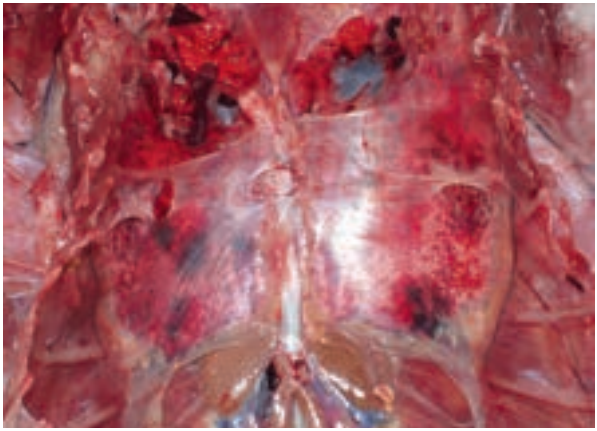

Fig.2
Another typical finding in acute fowl cholera is croupous pneumonia in the red hepatisation stage.

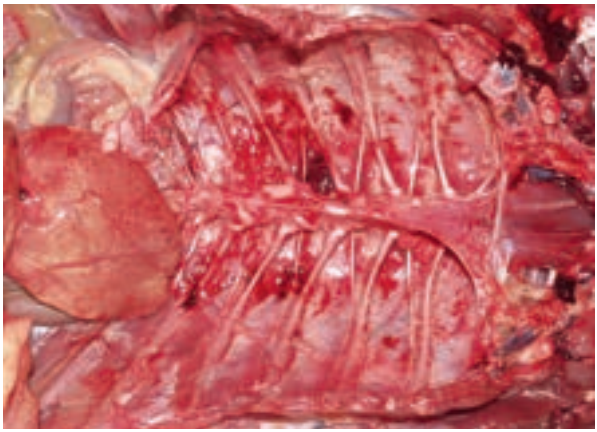

Fig.3

Frequently, subserous petechial or ecchymosed haemorrhages in the pleural cavity are discovered.

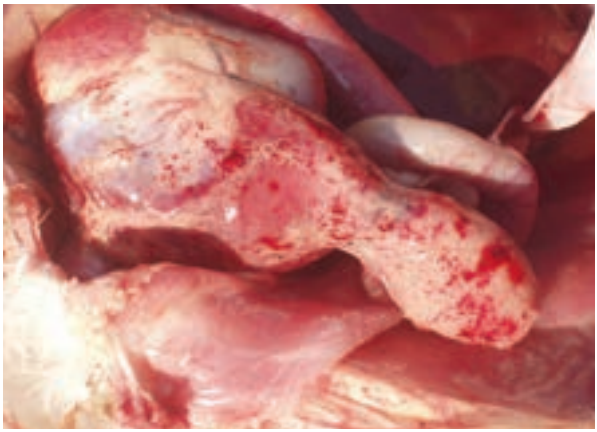

Fig.4

Subserous petechial and ecchymotic haemorrhages in the anterior part of the small intestine, the gizzard or the abdominal fat.

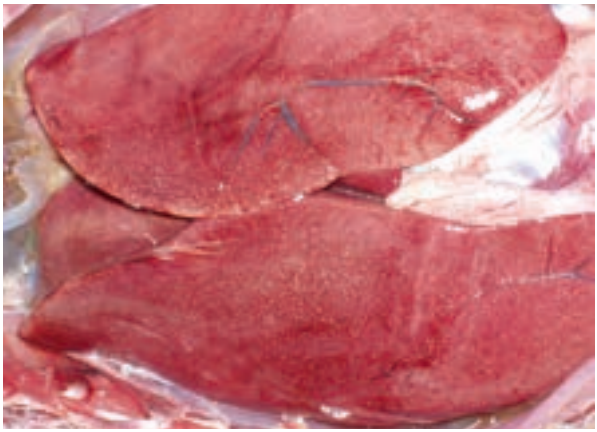

Fig.5
Important findings are the multiple milliary or submilliary necroses in the liver.

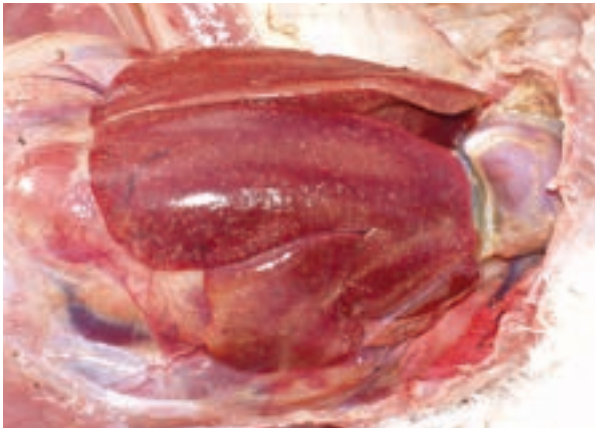

Fig.6
Multiple milliary necroses in the liver.

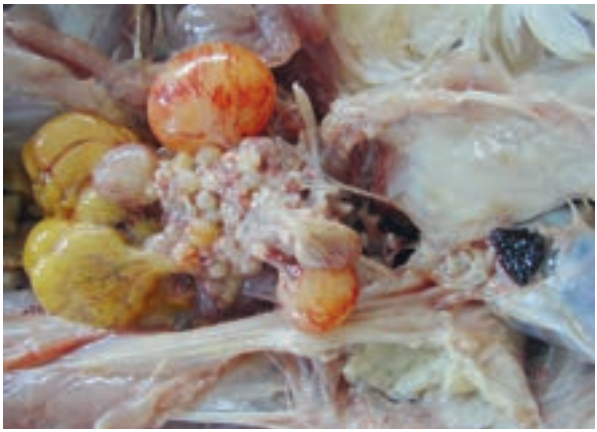

Fig.7

In layers (stock or breeders), acute oophorites with regressing follicles and consequently, diffuse peritonites are commonly observed.

Fig.8

The periorbital sinuses are frequently affected by a serofibrinous inflammation.

Fig.9
Another local form is the injury of wattles that are strongly distended because of their filling with fibrinouscaseous content.

Fig.10
Wattles filling with fibrinouscaseous content – cross section.

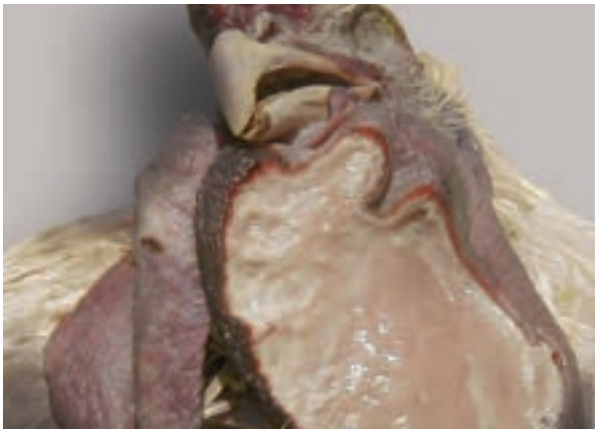

Fig.11
Wattles filling with fibrinouscaseous content – cross section.

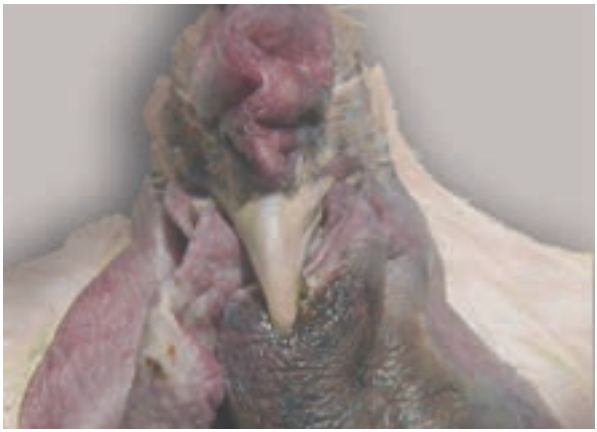

Fig.12
The fibrinouscaseous exudate accumulated in wattles sometimes leads to gangrene of the covering skin.

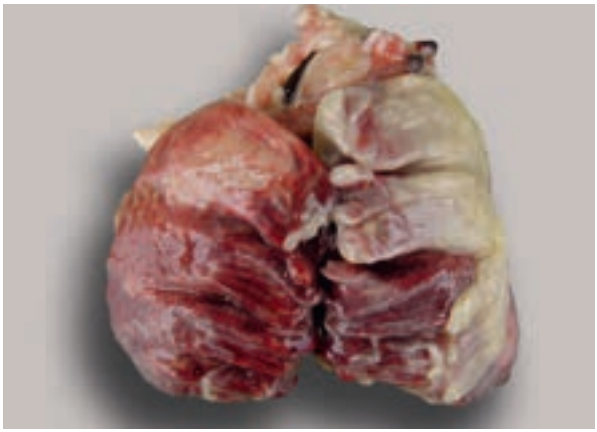

Fig.13

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

Fig.14

The inflammation may spread from sinuses to adjacent air-filled skull bones, with subsequent necrosis and onset of neurological signs (opisthotonus and torticolis).

DIAGNOSIS

A tentative diagnosis is made on the basis of disease history, observed clinical signs and any lesions observed. This initial diagnosis must be confirmed by isolating the organism. The nutrient media most commonly used for initial isolation are blood agar, dextrose starch agar or trypticase soy agar. *Pasteurella multocida* is easily isolated from the internal organs of birds killed by an acute form of cholera. Isolation of the organism in the chronic forms of the disease is more difficult. After 24 hours of incubation, the colonies vary in size from 1 to 3 mm. They are usually single, semi-transparent, bulging and round. In acute forms, bipolar organisms can be demonstrated in imprint liver preparations stained per Wright's or Giemsa (OIE, 1996). Immunofluorescence microscopy was used to identify *P. multocida* in exudates or tissues (Christensen & Bisgaard, 2000).

Identification is based primarily on the results of biochemical tests. Carbohydrate fermentation is of great significance. The carbo--hydrates metabolised are glucose, mannose, galactose, fructose, and sucrose, and the non-fermenting ones include rhamnose, cellobiose, raffinose, inulin, erythritol, adonitol, m-inositol, and salicin.

P. multocida rarely grows on MacConkey agar and does not cause haemolysis. It produces catalase, oxidase, and ornithine decarboxylase, but not urease, lysine decarboxylase, beta-galactosidase, or arginine dihydrolase.

Antigenic characteristics are based on capsular serogrouping and somatic serotyping. Capsular serogrouping is performed using the passive haemagglutination test. The serogroups determined by this test are A, B, D, E and F. With the exception of serogroup E, the others can be isolated from avian hosts. A disc diffusion test to differentiate serogroups A, D and F has been developed (Rimler R.B., 1994).

Somatic serotypes are usually established in the agar gel immunodiffusion (AGID) test (Heddleston et al., 1972). To determine the serotype, the prepared bacterial culture has to be tested as an antigen against all 16 serotype-specific antisera. The antigens present in an isolate can react with multiple serotype-specific antisera, yielding bi- or trinomial serotypes, such as strains 3,4 or 3,4,12 (Rimler et al., 1998). The most effective method involves profiling through both serogrouping and serotyping.

DIFFERENTIAL DIAGNOSIS

Fowl cholera should be differentiated from acute *E. coli* septicaemia, erysipeloid, fowl typhoid etc.

PREVENTION AND CONTROL

Biosecurity

Extensive management systems exist in all poultry farms in the world. However, the control of *P. multocida* infections is nearly impossible due to the natural reservoir. The increasing utilisation of open-type farms for reasons of animal welfare creates an increased risk of introducing the infection into commercial flocks. The prevention of FC can be effective through elimination of the reservoirs of *P. multocida* and restricting opportunities for the organism to enter poultry farms. Unlike many other bacterial infections, FC is not a hatchery illness. The infection occurs after the birds arrive at the farm. The management of procedures for non-specific prevention and control includes application of certain biosecurity measures which are standard practice in poultry production. As such, poultry production facilities should establish special isolation spaces between the sectors inhabited by growing and production age birds. Isolated housing of the different age groups should be maintained. Best practices for depopulation of barns and the all-in all-out principle should be respected. Cleaning, washing and disinfection of premises, equipment and vehicles should be conducted in accordance with the established biosecurity procedures. Moving out of the buildings and the treatment of the litter should follow the same rules as well.

Furthermore, other animals such as pigs, dogs and cats, should not be kept on the same farm. There should be an autonomous water source and adequate control over water safety. Another key priority is to take measures to limit the access of free-flying birds.

Vaccines

The control over FC in areas where the disease is prevalent or stationary generally depends on vaccination. Many live and inactivated (bacterins) vaccines against FC have been developed and tested in an attempt to control the illness (Glisson, 2008). Live attenuated vaccines are applied primarily in North America. Live vaccines induce protection against FC provoked by heterologous *P. multocida* serotypes. Because modified live vaccinal strains may revert to their pathogenic phenotypes and thus exhibit a tendency towards causing illness in immunosuppressed birds, most commercial vaccines are of the bacterin type. Bacterins usually contain whole cells from serotypes 1, 3 and 4 emulsified in oil adjuvant. Such vaccines provide protection against homologous serotypes, which are contained in the vaccinal bacteria, but not towards FC caused by other serotypes. Because of this, in some cases the chosen course of action is to prepare autogenous vaccines from whole bacterial cells from locally isolated strains. Another flaw of these vaccines is their administration by injection, and the potential for tissue reaction. In addition to oil adjuvant aluminum hydroxide may also be used, although it has been shown to be less effective at stimulating immune response, thus necessitating revaccination. When drawing up a suitable vaccination programme against FC, several circumstances such as the prevalent serotypes of *P. multocida* in the region and the age of the birds to be vaccinated should be taken into account. Immunisation with live vaccines or bacterins at 8-12 and reimmunisation at 18-20 weeks of age give promising results (Glisson, 2008).

TREATMENT

Certain antibiotics and sulfonamides reduce the death rate, but after discontinuation of the treatment the disease may recur. Sulfonamides are effective for treatment, but do inhibit egglaying.

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