# Fowl cholera

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**Abstract:** Fowl cholera is a septicemic disease caused by *Pasteurellamultocida* which affects a variety of domesticated and wild birds. This highly contagious disease causes high morbidity and mortality resulting in great economic losses, especially in large industrial-type poultry complexes. It usually occurs as an acute disease, but chronic infections can also occur in some outbreaks. *Pasteurellamultocida* type A is the etiologic agent of fowl cholera, highly contagious and fatal disease of chickens. *Pasteurellamultocida* have five types of capsular serotype i.e. type A, B, D, E and F. Diagnosis of the disease is mainly based on the clinical signs and symptoms, post mortem findings. Confirmatory diagnosis is done by isolation and identification of causative agent. A variety of laboratory diagnostic techniques have been developed over the years for pasteurellosis and used routinely in the laboratory. Among these techniques molecular techniques of diagnosis is most important. Accurate and early diagnoses are considered as the effective tools to frame the strategy for controlling of any infectious disease like Fowl cholera. Vaccination is considered as one of the common preventive measures worldwide to reduce the prevalence and incidence of disease. Control of fowl cholera is primarily ensured by good management practices and treatment with antimicrobial agents.
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## Introduction

Fowl cholera (FC) is a highly contagious disease caused by *Pasteurellamultocida* (*P.multocida*) that affects a broad host range of birds and causes high mortality that incur significant economic losses in commercial and backyard poultry production **(Christensen and Bisgaard, 2003)**. The incidence of fowl cholera, along with other bacterial diseases, is on the increase, despite vaccination and proper medication and can be attributed to various incriminating factors **(Jonas et al., 2001)**. Fowl cholera, caused by infection with *P.multocida*, is a disease of many avian species. Chickens, turkeys, ducks, and quail are the most important domestic avian species involved and the disease is of economical significance. Although *P.multocida* may induce lesions in multiple organ systems, respiratory pathology is the most important facet of the disease. *P.multocida*is a common organism found in the oral cavity of a variety of animals, including dogs, cats, and rodents. Various animals, particularly cats and rodents, are a common source for the introduction of the organism into commercial poultry **(Rimler and Glisson, 1997)**. In domesticated birds, (FC) causes significant economic losses worldwide **(Christensen et al, 2008)**. Turkeys and waterfowl are most affected, and death from (FC) in chickens usually occurs in laying flocks, because birds of this age are more susceptible than younger chickens **(Gilsson et al, 2003)**. Acutely affected birds typically die from septicemia and exhibit hemorrhages in the heart and lungs. The liver usually contains multiple small necrotic foci. Chronic FC is characterized by localized infections in the wattle, infraorbital sinus, sternal bursa, hock and wing joints, and foot pads. Chronic FC may follow an acute stage of the disease or result from infection with organisms of low virulence **(Christensen et al, 2008)**. *P.multocida* was isolated from apparently healthy ducks (25.9%) and chickens (6.2%) **(Mbuthia et al., 2008)**. *P. multocida* is a Gram-negative, non-motile, cocco-bacillus, capsulated, non-spore forming bacterium occurring singly, in pairs or occasionally as chains or filaments belonging to the Pasteurellaceae family **(Akhtar, 2013)**. *P.multocida* strains are classified intoserogroups A, B, D, E and F based on capsular antigens, and further classified into 16 serotypes (1 to 16) primarily based on lipopolysaccharide antigens **(Kwaga et al., 2013)**.

## Etiology

*P.multocida*is a gram-negative, non-motile, small, facultative anaerobic rod from the family *Pasteurellaceae* which grows rapidly on standard media but not on selective media such as McConkey agar **(*Bergey's Manual of Determinative Bacteriology*, 1994)**. *Pasteurellamultocida* is a gram-negative rod with bipolar staining that affects cattle **(Frank, 1989)**, pigs **(Blackall et al, 2000)**, rabbits **(Langan et al, 2000)**, and humans **(Weber et al, 1984)**, in addition to fowl **(Christensen et al, 2008)**.

*P.multocida* includes capsulated and noncapsulated types, with the former being more virulent than the latter **(Chung et al,2001)**. *P.multocida* can be differentiated serologically by capsular antigens into serogroups A, B, D, E, and F 4 **(Rimler and Rhoades, 1987)**, and also by somatic antigens into somatic serotypes 1~16 **(Borgden et al, 1978)**. Among the five capsular serogroups, B and Ecausehemorrhagic septicemia in cattle and wild ruminants **(De Alwis, 1992)**, and serogroup D causes atrophicrhinitis in pigs **(Quinn et al, 1994)**. In poultry, capsular serogroup A causes (FC) **(Timoney et al, 1992)**. All somatic serotypes have been isolated from birds. Among sixteen somatic serotypes, 1, 3 and 4 are most frequently reported **(Adler et al, 1999)**. Fowl cholera is caused by *P.multocida* type A:1, A:3 and type D in Asian countries **(Ranjan et al., 2011)**. Among the different serogroups, serotype A:1 strains causes 80% mortality, in contrast 20% mortality caused by type D strains of (FC) in chickens **(Mohamed et al, 2012)**. Pathogenicity or virulence of *P.multocida*is variable and complex, depending on the host species, strain, variation within the strain or host, and conditions of contact between two birds **(Richard and Rimlen, 2001)**.

**Susceptibility**

All domestic and wild species of birds are susceptible to (FC). Most reported outbreaks involve chickens, turkeys and ducks, and occasionally species such as geese, pigeons, pheasant, quail, sparrows and finches. In turkeys, (FC) generally occurs between 10 to 13 weeks of age. It rarely occurs in birds less than 2 or 3 weeks of age **(Mbuthia et al.,2008)**.

**Source of infection**

The primary source of *P.multocida* infection can be excretions from the nostrils, mouth and eyes of sick birds or chronic carriers. The secondary sources are contaminated feed, water, crates, equipment and shoes. Wild birds, including sparrows and pigeons and many mammals (especially pigs, cats, and wild rodents) can disseminate *P.multocida*. The organism can persist for years in the oralcavity of rodents and carnivores. Birds bitten by such animals can become infected and disseminate the disease within the flock. Cannibalism of sick or dead birds is also as significant method of dissemination **(Rimler and Glisson, 1997)**.

**Pathogenesis**.

*P.multocida* usually enters the host through the mucous membranes of the upper respiratory tract and probably the digestive tract also. The ability of pasteurella to resist phagocytosis after invading tissues allows these bacteria to multiply very quickly, causing septicemia and severe endotoxemia; death often ensues within 24 hours. The role of endotoxemia, however, in (FC) is not clear. The pathogenesis of clinical signs and lesions depends upon various factors such as virulence of the infecting pasteurella strain, the infecting dose, the age and immunologic competency of the host, the route of infection, and other predisposing factors including poor nutrition, environmental temperature and concurrent viral infections that may induce immunosuppression **(Rimler and Glisson, 1997)**. *P.*multocida is a heterogeneous species in witch the pathogenicity of individual strains is highly variable and susceptibility of the host to these bacterial strains varies considerably among avian species **(Christensen and Bisgaard, 2000).**

**Economic Significance**

*P.multocida*is associated with hemorrhagic septicaemiain cattle and buffaloes, pneumonic pasteurellosis in sheep and goats, (FC) in poultry, atrophic rhinitis in pigs and snuffles in rabbits **(De Alwis, 1996)**. An annual economic loss in India due to P. Multocidais Rs. 225/- millions **(Singh et al., 2008)**. Fowl cholera has been recognized as an important disease in domestic poultry for more than 200 years that causes devastating economic losses to poultry industry worldwide **(Aye et al, 2001)**. Fowl Cholera (avian cholera, avian pasteurellosis or avian hemorrhagic septicemia) is a contagious disease affecting domesticated and wild birds **(Swapnil *et al.*, 2011)**. The incidence of fowl cholera caused by *P.multocida*is reported to be on the increase, **(Mbuthia *et al.*, 2008)** documented the occurrence of *P.multocida* among healthy-appearing family poultry in a tropical setting and concluded it to be the most common bacterial disease encountered in village chickens. Fowl cholera, occurs sporadically or enzootically in most countries of the world wherever intensive poultry production occurs, and is known as a bacterial disease with major economic importance due to its high mortality **(Glisson, *et al.*, 2013)**.

**Clinical signs**

*Pasteurellamultocida* subspecies *multocida* (*P. multocida*) is an important pathogen that causes fowl cholera (FC) in poultry and wild birds **(Xiao et al., 2015).** In poultry, *P.multocida* often associated with severe economic loss due to loss of cattle or poultry species **(Marza et al., 2015)**. The Fowl cholera, a septicemic disease, is associated with high morbidity and mortality in poultry especially chicken and ducks. Signs and symptoms of (FC) in acute cases are often present for only few hours before death; the signs in chicken include fever, ruffled feathers, mucus discharge from mouth, nose and ears, and cyanosis of comb and wattles **(Glisson et al., 2008)**. Five capsular serotypes (A, B, D, E, and F) are usually found in *P.multocida*, and each is generally associated with a specific host, for example, Serotype A causes (FC) in avian species **(Harper et al., 2006),** and Serotype B causes hemorrhagic septicemia in cattle **(Marza et al.,**

**2015)**. Virulence of *P. multocida* varies depending on the strain involved and factors host species **(Glisson et al. 2008)**. The Fowl cholera is mostly prevalent in fall, winter and late summer **(Heddleston and Rhoades, 1978)**. Laying flocks are mostly affected by (FC) because of their more susceptibility to the disease as compared to younger chickens (**Wang et al., 2009**). **Choudhury et al. (1985)** reported about 25-35% mortality of chickens due to FC in Bangladesh. *P.multocida* causes acute septicemia and chronic respiratory infection in birds, (**Furian, *et al.,* 2016)**. Fowl cholera is of historical importance because it is the first bacterial disease investigated for which a vaccine was developed **(Pasteur., 1881)**. The clinical picture of (FC) does often, but not always, include respiratory infections. Virulent strains cause acute (FC), which is featured by fever, anorexia, strong mucous discharche from the mouth, diarrhea and an increased respiratory rate. These signs occur within a day before death, which is caused by sepsis **(Park.1982)**. Chronic (FC), caused by less virulent strains, shows signs generally related to localized infections e.g. in the wattles, footpads, joints and the respiratory tract Free-flying birds and chronically infected birds are considered to be the source of infection (**Rhoades and Rimler, 1991 )**.

**Post Mortem Lesions**

The gross lesions were extensive congestion, enlarge and necrotic foci on spleen and liver, petechial hemorrhage in cardiac muscle, necrotic parenchymatoushepatitis, congestion and hemorrhages in the intestinal mucosa **(Mohamed et al., 2012)**. The histopathological signs of (FC) were hemorrhage, congestion and lymphoid cell infiltration in liver, heart and spleen. **(Shilpa et al., 2006)**. Experimental inoculation of *P.multocida* isolates in chickens produced characteristics changes in heart, liver and spleen. The chickens were died within 24 h after challenging with *P.multocida* Type A. In post-mortem examination, marked septicemiclesions consisting of white necrotic foci and hemorrhages in heart, liver and spleen were found as reported by **Zahoor and Siddique (2006).** Histopathological study confirmed the occurrence of ra (FC) in the experimental chickens. Huge lymphocytic infiltration in central vein of liver was found. Inflammatory cells were found in the pericardium of heart, and lymphocytic infiltration was noticed in the red areas of spleen, as described by **Shilpa et al. (2006)**.

**Diagnosis**

There are complexities associated with the diagnosis of fowl cholera by conventional methods, using capsular serotyping. Some avian strains of *P.*multocidaare non-encapsulated and cannot be classed into a serological group **(Wilson et al.,1993).** In addition, indirect methods could fail due to the difficulty in producing high titre antibodies against serogroups A,D and F; this is mainly due to the presence of inert capsule materials, such as hyaluronic acid and muco-polysaccharides concealed beneath the serogroup antigens **(Carter and Rundell,1975).** There is an urgent need to establish reliable and rapid methods to identify and type field isolates of *P.*multocida before an effective vaccine against (FC) can be developed in Egypt. In recent years, genotypic methods for bacterial identification have proved beneficial in overcoming limitations of traditional phenotypic procedures **(Arumugam et al., 2011)**. A polymerase chain reaction (PCR) assay has been developed for capsular typing of *P.* multocidastrains. This assay represents a rapid and reproducible alternative to serological methods **(Sellyei et al., 2008).**

**Treatment**

Antibacterial treatment is still commonly used to control (FC) but has been accompanied by the emergence of resistant strains. The resistance strains are a result of the widespread use of antimicrobials in feed, both for prophylaxis and for growth promotion. Subtherapeutic uses of antimicrobials in feed have caused the emergence of multi-antimicrobial resistance. Antimicrobial resistance can evolve in the strains by the molecular transmission of resistance mechanisms from other bacteria carried by mobile genetic elements **(Tang et al.,2009).** Florfenicol and fluoroquinolones (ciprofloxacin) were the most active drugs **(Huang et al.,2009).** The aminoglycoside antimicrobials usually showed poor activity against *P.*multocida **(Gutierrez and Rodriguez,1993).** The higher resistance among strains to doxycycline was considered to be due to cross-resistance between oxytetracycline and doxycycline **(Woo and Kim, 2006).**

**Control and Prevention Strategies**

Accurate and early diagnoses are considered as the effective tools to frame the strategy for controlling of any infectious disease like FC. Conventional diagnostic system is not effective in all cases since it is time consuming and less sensitive as compared to molecular technique, for example, polymerase chain reaction (PCR). Vaccination is considered as one of the common preventive measures worldwide to reduce the prevalence and incidence of disease **(Kardos and Kiss, 2005)**. Vaccination to prevent (FC) is an important aspect of controlling the disease, particularly in broiler breeders and turkeys. There are two broad categories of vaccines commercially available, live vaccines of low virulence and inactivated vaccines. There are a wide variety of vaccination programs utilized that minimize the impact of fowl cholera (FC) on a flock. Inactivated *P.*multocidabacterins are whole cell suspensions of

inactivated *P.*multocidaemulsified in an oil adjuvant. These products induce an immune response specific to the serotype of the organism in the bacterin **(Rimler and Glisson, 1997)**. Vaccination with bacterins induceserovar-dependent protection (unpublished observations). Live vaccines induce better cross-protection but often have residued pathogenicity. Acute (FC) is very hard to treat. Only high doses of antibiotics e.g. Streptomycin, given intramuscularly just before or at the time of inoculation of *P.multocida*in experimental challenge studies, can prevent death. The efficacy of the treatment of chronic (FC) is dependent on the sensitivity of the strain involved because *P.multocida* strains strongly vary in susceptibility to antibiotics (**Rhoades and Rimler, 1991)**. Broiler breeder pullets should be vaccinated twice at least 4 wk apart with either two inactivated vaccines, two live vaccines, or one live vaccine and one killed vaccine. Young meat turkeys are typically vaccinated with a live vaccine at about 6, 10, and 14 wk of age. Fowl cholera can be effectively treated with several different antibiotics **(Rimler and Glisson, 1997)**.

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