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Review on Fowl Cholera

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ABSTRACT

Pasteurella multocida subspecies multocida is the most common cause of fowl cholera, although pasteurellamultocida subspecies septica and gallicida may also cause fowl cholera-like disease to some extent. However, the virulence properties of the different subspecies for various hosts have not been elucidated. The seventy and incidence of pasteurellamultocida infections may vary considerably depending on several factors associated with the host (including species and age of infected birds), the environment and the bacterial strain. No single virulence factor has been associated with the observed variation in virulence among strains. Possible virulence factors include the following: the capsule, endotoxin, outer membrane proteins, and iron binding systems, heat shock proteins, neuraminidase production and antibody cleaving enzymes. Carrier birds seem to play a major role in the transmission of cholera. The site of infection for asteurella multocida is generally believed to be the respiratory tract. The outcome of infections may range from peracute/acute infections to chronic infections. In the former type of infections, few clinical signs are observed before death and the lesions will be dominated by general septicemia lesions. In chronic forms of pasteurella multocida infections, suppurative lesions may be widely distributed, often involving the respiratory tract, the conjunctiva and adjacent tissues of the head. Diagnosis is always dependent the development of safe and efficient live vaccines still poses problems.

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Introduction

Fowl Cholera is a serious, highly contagious disease caused by the bacterium Pasteurella multocida in a range of avian species including chickens, turkeys, and water fowl, (increasing order of susceptibility). It is seen worldwide and was one of the first infectious diseases to be recognized, by Louis Pasteur in 1880 [1].

The bacterium has a worldwide distribution and produces septicemia and respiratory disease in a wide variety of domestic and wild birds. Acute illness is common; infection can result in mortality within six to 12hr after exposure, although one to two days is more typical [2].

The disease can range from acute septicemia to chronic and localized infections and the morbidity and mortality may be up to 100%. The route of infection is oral or nasal with transmission via nasal exudate, feces, contaminated soil, equipment, and people. The incubation period is usually 5-8 days [1].

Disease transmission among wild birds is believed to occur from bird-to-bird contact and by ingestion of bacteria or aerosol transmission within a contaminated environment. Discharge of pasteurellae from dead or diseased birds is considered an important source of wetland contamination and transmission to susceptible birds. Despite its occurrence in domestic fowl on most continents, avian cholera seems best described as having a limited distribution and significance for most wild bird populations around the world [3].

So far, no study has been conducted on fowl cholera in Ethiopia. It will be therefore to create awareness about the magnitude, spatial and temporal distribution and economic impact of the disease in poultry farm so as to design cost effective control strategies. Therefore the objective of this seminar paper is: To highlight the general characteristics of fowl cholera, public health significance of the disease and to review control and preventive strategies against the disease.

Literature Review on Fowl Cholera

Synonym

Avian pasteurellosis, avian hemorrhagic septicemia, chicken cholera (Rhoades and Rimler, 1990).

Etiology

Three subspecies of P. multocida (P. multocida subspeciesmultocida, septica and gallicida) are recognized.Pasteurella multocida subspecies multocida is the mostcommon cause of disease, but subspecies septica and gallicidamay also cause fowl cholera-like disease to some extent.Pasteurella multocida subspecies gallicida is mainly associatedwith web-footed birds but also reported in pigs [4].

Epidemiology

Although fowl cholera probably occurs world-wide and has been studied extensively for many years, the epidemiology of the disease remains controversial, and many aspects are not yet fully understood. Basic knowledge, such as the route of introduction of fowl cholera into a flock, is still lacking. Due to genotypic variation within serotypes, serotyping in many cases does not provide sufficient detailed information to determine the epidemiology of infections [5].

Pathogenesis

The site of infection for P. multocida is generally believed to be the respiratory tract. However, inoculation through ocular-nasal-oral routes may also generate typical lung lesions and a progressive bacteraemia, indicating that other mucosal membranes may multocida on the cloacal surface of carrier birds indicates that some organisms may survive passage [6]. The observation that some strains of P. multocida can be virulent and immunogenic following oral administration also suggests that intestinal invasion or interaction with the intestinal mucosae occurs to some degree. Localization of P. multocida in the bursa may occur following bacteraemia, since P. multocida has been detected in the bursa of intra-tracheally infected chickens. Pasteurella multocida may also enter the tissues through cutaneous lesions and result in septicaemia or localized cutaneous lesions. Following an upper respiratory tract infection, P. multocida may subsequently spread to the lungs and multiply before entering the bloodstream [7].

Once in the bloodstream, P. multocida either multiply rapidly or localize in the liver and spleen where initial multiplication occurs before a massive bacteraemia. Death is presumed to be due to the effects of endotoxin as signs of acute fowl cholera have been reproduced by injection of endotoxin from P. multocida [8].

Clinical sign

There are two forms of clinical signs depending on the nature of the disease.

Acute forms

Acute fowl cholera causes sudden death, sometimes without signs of infection. Signs of infection can be severe depression, cyanosis (dark-purple discoloration of skin) and mucus coming out of the beak (http// www.merkmanuals.com). Signs associated with fowl cholera also includes such as dejection, ruffled feathers, loss of appetite, diarrhea, coughing, nasal, ocular and oral discharge, swollen and cyanotic wattles and face, sudden death, swollen joints, lameness [1].

It is more common form as flock problem. So that loss of appetite, bluishness of combs and wattles, catarrhal discharge from nostrils, fever, and prostration, drooling of saliva and diarrhea, later becomes yellowish and greenish. Death occurs due to dehydration and septicaemia and varies between 15 to 90%, depending on the strain of the causal organism. In 3-6 week old boilers' the birds revealed 'getting down' on legs and dying. Death can be so rapid that birds may literally fall out of the sky or die while feeding, with no signs of illness (Scott et al., 1999).

Chronic Form

Chronic forms as sporadic cases, usually at the end of an outbreak. And most common form is oedema of wattles, combs and sometimes as otitis, arthritis and subcutaneous tissue of the head oviduct and the respiratory tract. Sever forms of dermal necrosis in turkeys have also been reported [8].

Diagnosis

The history of the disease, clinical signs and gross lesions may be helpful in diagnosis, but are insufficient to allow a definite diagnosis of the disease. The final diagnosis depends on isolation of the organism [9].

Public Health Implications

Disease in humans caused by P. multocida is not uncommon, and P. multocida may be considered a zoonotic organism. This is substantiated by the observation that the disease apparently occurs predominantly among the farming population [10]. No reports exist of direct transmission from poultry to man or vice versa, but the possibility for such infections cannot be excluded. The organism is a common cause of infection following animal bites or scratches which are mostly caused by dogs or cats (including large cats) [11].

Bite wound infections caused by pigs have also been reported. A severe cellulitis may develop which may progress to osteomyelitis and subsequently to septicaemia. In patients with dysfunction of the liver in particular P. multocida is known to cause bacteraemia which may localize in joints, respiratory tract or progress and cause sepsis. In addition to these principal types of infections, P. multocida has been isolated from a variety of infections, including peritonitis, puerperal sepsis, neonatal sepsis, brain abscesses and urinary tract infections. The significance of the different subspecies of P. multocida in relation to diseases reported has not yet been elucidated [10].

Treatment, Prevention and Control

Treatment

Antibacterial chemotherapy (gentamicin, entrofloxacin, doxycyclin, neomycin plus doxycyclin, sulphonamide&chloramphnicol) has been used extensively in the treatment of avian cholera in domestic birds and could be used in captive birds [12]. However, against the per acute forms of the disease, drugs may be more valuable as a prophylactic than as a therapeutic agent [13].

Prevention

Good management practices, including a high level of biosecurity, are essential to prevention. Rodents, wild birds, pets, and other animals that may be carriers of P multocida must be excluded from poultry houses [14].

Vaccination is commonly used in domestic fowl to reduce the potential for disease epizootics and related mortality. A vaccination strategy has also been employed for captive Canada Geese, but most vaccines have a limited duration of ≤ 1 year, require individual handling and immunization, and have varying degrees of efficacy among species. In either case, large-scale drug therapy or vaccination of wild populations is likely to be impractical, if not futile [15].

Control

Appropriate actions to control avian cholera epizootics depend on the severity and distribution of disease and the importance of the species or populations involved. More typically, the control strategy for wetlands with ongoing disease epizootics involves regular wetland surveillance, carcass removal, and disposal of carcasses. However, the benefits of a carcass collection program have not been rigorously tested [16].

Under extreme conditions, wetland disinfection, depopulation, or treatment measures may be warranted [2]. Depopulation appears

to be feasible only under a limited set of conditions involving a discrete and localized epizootic that presents a high risk to other susceptible species, when complete eradication of infected birds without substantial risk of disease spread is feasible, and when eradication measures are specific to the target species.

Conclusion and Recommendations

Fowl cholera is aserious, highly contagious bacterial disease of poultry birds caused by the bacterium pasteurellamultocida in arrange of avian species including chickens, turkyes and water fowls. The disease is seen worldwide and was one of the first infectious diseases to be recognized, by Louis Pasteure in 1880. The rout of infection is oral or nasal with transmission via nasal exudates, feces, contaminated soil, equipment, and vectors as well as peoples. It is characterized by sudden death, severe illness, drooling of saliva, diarrhea and sudden onset of per acute and acute clinical signs and in chronic form is oedema of wattles, combs, sometimes as otitis, arthritis and subcutaneous tissue of the head, oviduct, and respiratory tract. It is also examined by history, clinical sign and gross lesion, especially, for identification of the organism in laboratory practices (blood smear); in acute septicaemic form, blood smears should be stained with Leishmans, Wright's or Giemsa stain and examine for the presence of bipolar organism in large numbers. No country can be considered free of fowl cholera primarily because the causative agent. Pasteurella multocida has abroad habitat including mucosal surface of wide range of domestic and wild birds as well as mammals. Among this prevention and control strategies is more practical in it to reduce the transmission and distribution of the disease by under taking vaccination program and treatment administration when the disease can be outbreak to the flock of chicken production. Based on the above conclusion the following recommendations are forwarded :- 1) Peoples who are involved in the poultry production should be aware of the effect of fowl cholera on their farm and on their own health impact. 2) Adequate control and prevention methods should be taken in order to decrease the prevalence of this disease. 3) Studies to know the prevalence of the disease should conduct and eradication methods should adopt before the disease becomes wide spread and high in prevalence.

References

1. Paul McMullin (2004) Fowl cholera, pasteurellosis http// www.the poultrysite.com.
2. Friend M (1999) Avian cholera. In Field Manual or Wildlife Diseases. General Field Procedures and Diseases or Birds. (M. Friend and J. C. Fransoneds.) US Geological Survey Information and Technology Report 1999-001: 75-92.
3. Botzler R (1991) Epizootiology of avian cholera in wildfowl. Journal of Widlife Diseases 27: 367-395.
4. Christensen JP and Bisgaard M (2000) Fowl cholera. Revue Scientifiqueet Technique de Le Office International Des Epizooties 19: 62-637.
5. Christensen JP, Petersen KD, Hansen HC & Bisgaard M (1999) Occurrence of fowl cholera in Danish wild birds, and poultry production. Possible connections [in Danish, summary in English]. Dansk Vettidsskr 82: 342-346.
6. Muhairwa AP, Christensen JP and Bisgaard M (2000) Investigations on the carrier rate of Pasteurella multocida in healthy commercial poultry flocks and flocks affected by fowl cholera. Avian Pathol 29: 133-142.
7. Matsumuto M, Strain JG and Engel HN (1991) The fate of Pasteurellamultocida after intratracheal inoculation into turkeys. Poult Sci 70: 2259-2266.
8. Frame DD, Clark FD and Smart RA (1994) Recurrent outbreaks of a cutaneous form of Pasteurella multocida infection in turkeys. Avian Dis 38: 390-392.
9. Rimler RB, Sandhu TS and Glisson JR (1998) Pasteurellosis, Infectious Serositis, and Pseudotuberculosis. In: A Laboratory Manual for the Isolation and Identification of Avian Pathogens, 4th Edition, (Swayne D.E., Glisson J.R., Jackwood, M.W., Pearson, J.E. and Reed W.M., eds). American Association of Avian Pathologists, Kennett Square, Pennsylvania, USA 17-28.
10. Bisgaard M (1995) Salpingitis in web-footed birds: prevalence, aetiology and significance. Avian Pathol 24: 443-452.
11. August J (1990) Dog and cat bites. In zoonosis updates from journal of the American Veterinary Medical Association. American Veterinary Association 58-62.
12. Christensen JP, Dietz HH & Bisgaard M (1998) Phenotypic and genotypic characters of isolates of Pasteurella multocida obtained from back-yard poultry and two outbreaks of avian cholera in the avifauna in Denmark. Avian Pathol, 27: 373-381.
13. Ficken MD, Barnes HJ and Qureshi MA (1991) Acute airsacculitis in turkeys inoculated with cell-free culture filtrate of Pasteurella multocida. Vet Pathol 28: 46-54.
14. (2014) cholera overview of fowl cholera.html. November 13 http://www.merkmanuals.com/vet/poultry/fowl.
15. Lee MD, Glisson JR and Wooley RE (1992) Factors affecting endotoxin release from the cell surface of avian strains of Pasteurella multocida. Vet. Microbiol 31: 369-378.
16. Wilson C and Smit Th (1991) Pasteurellosis among breeding eiders Somateriamollissima in the Netherlands. Wildfowl 42: 94-97.
17. Frost A, Boyle D, Wilkie I and Townsend K (1999) Live metabolic drift vaccines for pasteurellosis in chickens and ducks. In Proc. Haemophilus, Actinobacillus and Pasteurella Conference, 13-17, September, Mabula, South Africa. University of the Orange Free State, Bloemfontein 42: 13-17.
18. Glisson JR and HsinNing Cheng I (1991) In vivo antigen expression by Pasteurella multocida. Avian Dis 35: 392-396.
19. Harmon BG, Glisson JR, Latimer KS, Steffens WL and Nunnally JC (1991) Resistance of Pasteurella multocida A: 3, 4 to phagocytosis by turkey macrophages and heterophils. Am.J vet Res 52: 1507-1511.
20. Kasten RW, Carpenter TE, Snipes KP and Hirsh DC (1997) Detection of Pasteurella multocida-specific DNA in turkey flocks by the use of the polymerase chain reaction. Avian Dis 41: 676-682.
21. Moore MK, Cicnjak-Chubbs L and Gates RJ (1994) A new selective enrichment procedure for isolating Pasteurella multocida from avian and environmental samples. Avian Dis 38: 317-324.
22. Office International des Epizooties (OIE) (1996) Fowl cholera (avian pasteurellosis), chapter 3.6.11. In Manual of standards for diagnostic tests and vaccines, 3rd Ed. OIE, Paris 572-577.
23. Ogunnariwo JA, Alcantara J and Schryvers AB (1991) Evidence for non-siderophore-mediated acquisition of transferrin-bound iron by Pasteurella multocida. Microb. Pathogen 11: 47-56.
24. Snipes KP, Hirsh DC, Kasten RW, Carpenter TE, Hird DW, et al. (1990) Homogeneity of characteristics of Pasteurella multocida isolated from turkeys and wildlife in California, 1985-88. Avian Dis 34: 315-320.
25. Wilson MA, Duncan RM, Nordholm GE and Berlowski BM (1995) Serotypes and DNA fingerprint profiles of Pasteurella multocida isolated from raptors. Avian Dis 39: 94-99.

26. Wilson MA, Duncan RM, Nordholm GE and Berlowski BM (1995) Pasteurella multocida isolated from wild birds of North America: a serotype and DNA fingerprint study of isolates from 1978 to 1993. Avian Dis 39: 587-593.