Pasteurellosis in Rabbits

M. H. Al-Haddawi
Dept. of Vet. Path., College of Veterinary Medicine
Chonnam National University
Kwangju 500-757, South Korea

Introduction

In domestic rabbits, pasteurellosis is a primary respiratory disease entity caused by *Pasteurella multocida*. Respiratory pasteurellsos in rabbits is highly contagious due to dissemination of the organism in the nasal discharge (Benirschke, *et al.*, 1978). The disease reflects a varied and complex host-parasite relationship. Acute infection with high mortality occurs sporadically especially in stressed rabbits less than 10 weeks old, in the form either an enzootic pneumonia, infection of the upper respiratory tract or septicemia. In addition to respiratory form, infection with *P. multocida* results in a variety of disease processes in rabbits including rhinitis, otitis media, otitis interna, abscesses, metritis, orchitis and meningoencephalaitis (Manning *et al.*, 1989). The deleterious effects of pasteurellosis are common and pose serious problems for rabbit breeders and researchers (Flatt, 1974).

Pasteurella multocida occurs whenever mammalian and fowl are found (Soltys, 1979). It has been recognized as an important primary or secondary animal pathogen for a century and also an important human pathogen.

The organism is also a communal in the naso-pharyngeal region of apparently animals. The organism usually acts as secondary invaders in animals with concurrent diseases or suffering from debilitating stressful condition (Benirschke, *et al.*, 1978). It may acquire virulence and produce severe primary disease outbreak in the absence of other diseases or predisposing factors (Carter, 1967).

Pasteurella multocida

It is a non-motile, non-sporeforming, coccobacillus or short rod which stains Gram-negative. Based on the antigenic properties there are five capsular types (A, B, D, E, and F) and 16 serotyps (1 through 16). Two capsular types of *P. multocida* are associated with pasteurellosis in rabbits, namely A and D (Lu *et al.*, 1983; Kawamoto *et al.*, 1990b; Al-Haddawi *et al.*, 1998a). Serotypes 1, 3 and 12 were reported to be the predominant serotypes (DiGiacomo *et al.*, 1983; Jasni *et al.*, 1999). The prevalence of *P. multocida* in rabbits breeding colonies and in laboratory animal facilities varies from about 20% to 70% (DiGiacomo *et al.*, 1983; Kawamoto *et al.*, 1990a).

The variation in the virulence of *P. multocida* isolates in animals has been well recognized. Several bacterial products including endotoxin, neuraminidase, an adhesin, antiphagocytic capsule, exotoxin and iron regulated proteins, haemagglutinins and

fimbriae have been reported to be virulence factors (Scharmann *et al.*, 1970; Glorioso *et al.*, 1982; Fortin and Jacques, 1987; Choi-Kim *et al.*, 1991; Al-Haddawi *et al.*, 2000a).

Pathogenesis

Pasteurella multocida occurs widely in the upper respiratory and digestive tract of a wide range of birds and mammals. It is either a frequent secondary invader or opportunist in a number of pathological processes. When the state of equilibrium is disturbed by a reduction in the hosts resistance and the organism established in the nasal passage, the infection spreads to the adjacent tissues. These tissues are paranasal sinuses, nasolacrimal duct and conjunctiva, Eustachian tube and middle ears, trachea, bronchi and lungs (Flatt, 1974).

The incubation period is difficult to define because many rabbits are subclinical carrier of infection. The transmission of *P. multocida* in rabbits is poorly understood, although it is assumed that the spread occurs both by direct contact and airborne means.

The clinical and Pathological Manifestations

There are many clinical manifestations of *P. multocida* infection in rabbits, which include upper respiratory diseases (rhinitis, sinusitis, conjunctivitis), otitis, pleuropneumonia, bacteremia and abscesses of many organs (Flatt, 1974).

Rhinitis and sinusitis are the common forms of pasteurellosis in rabbits and are called snuffles. The clinical signs of snuffles are characterized by serous, mucous or mucopurulent nasal discharge. Grossly, there is an exudate in the nasal cavity and paranasal sinuses. The nature of exudates change from serous to mucous and to mucopurlulent depends on the stage of the disease. Histologically, there is congestion of blood vessels, edema, focal mucosal necrosis and an inflammatory cells infiltration of mainly heterophils in acute cases. In subacute and chronic stages, hyperplasia of goblet cells, sloughing of focal areas of the mucosal epithelium and infiltration of degenerated heterophils (Al-Haddawi *et al.*, 1999). Ultrastructurally, changes consisted of ciliary deformation and deciliation of the ciliated epithelial cells, cellular swelling, goblet cell hyperplasia, excessive secretion of mucus and attachment of *P. multocida* to the degenerated cilia, microvilli and mucus.

Otitis media is usually asymptomatic unless the inflammatory process extends to the inner ear causing torticollis, nystagmus and ataxia.

The clinical signs of conjunctivitis include a moderately swollen eyelid, ocular discharge, chemosis and hyperemia.

In acute cases, enzootic pneumonia is a lethal form of pasteurellosis of rabbits. The clinical signs of pneumonia are undetected in natural infections. There are non-specific clinical signs such as anorexia, weight loss, depression and rapid fatigue. It is likely to take the form of pluropneumonia and pericarditis, with abscess

developing in or around the lung and heart. The antero-ventral areas of lungs are most common sites of lung lesions. Microscopically, fibrinopurulent exudate is in the airways, adjacent alveoli and on the serosal surfaces. The principle inflammatory cell is neutrophils besides macrophages and free red blood cells. In chronic cases, there is focal necrosis of the bronchial and alveolar epithelium. Under electron microscope, lungs showed degenerative changes in pneumocytes and endothelial cells and infiltration of inflammatory cells. It has been reported the presence of intracytoplamic organism in the epithelial cells of nasal mucosa and lungs of rabbits infected with serotype A:3 which indicates the ability of this serotype to invade the epithelial cells and acts as a virulence mechanism (Al-Haddawi *et al.*, 2000b). Fibrinous pericarditis and perihepatitis with liver necrosis may accompany this form.

Septicemic form is recognized as a primary or a sequalea to other forms of pasteurellosis and potentially fatal. Clinical sings of septicemia are not usually recognized because of its peracute form and rapid death of the animals (Manning *et al.*, 1989, Al-Haddawi *et al.*, 1999). Pathologically, the gross and microscopic lesions may comprise congestion of visceral organs and hemorrhages beneath serous membrane and subcutaneous tissues. Surviving rabbits showed ultrastructurally, the nasal and tracheal mucosa had changes varying from clumping of cilia to complete sloughing of epithelial cells. Bacterial cells attached to endothelial cells of the subepithelial capillaries of the trachea. Degenerative changes were also seen in the endothelial cells of blood capillaries. These cells displayed an irregular luminal surface. Some cells were swollen and contained vacuoles with large osmiophilic materials in their cytoplasm.

Generally, the nature of the exudate in *P. multocida* infections is suppurative. It is known that *P. multocida* and /or its products have a chemotactic ability for the neutrophils by generation of C5a (Latimer *et al.*, 1990). It has also been found that macrophages might play a role in the pathogenesis of pasteurellosis in rabbits (Al-Haddawi *et al.*, 1998b). Purified toxin produced by toxigenic *P. multocida* induces pneumonia, pleuritis, lymphoid atrophy and possible osteoclastic bone resorption in rabbits (Chrisp and Foged, 1991).

Control and Treatment

Since therapy is only marginally effective, several strategies have been attempted to control pasteurellosis. These include, barrier housing to prevent exposure, resistant breeds, vaccination and prophylaxis with antimicrobial.

Early weaning of rabbits with strict hygienic regimen is recommended to select *P. multocida*-free stocks even without using antimicrobial therapy (DiGiacomo, *et al.*, 1983). Some rabbits breeds are resistant to *P. multocida* infection than others. Thus choosing and breeding a resistant

will help in eradication of pasteurellosis. One promising approach is the development of immunization procedures, which over long period will protect animal exposed to *P. multocida*. Many trails have been conducted to prepare an effective vaccine including killed whole bacteria, live streptomycin-dependent strain and potassium thiocyanate extracts. So far, there are no effective vaccines commercially available against pasteurellosis in rabbits. Information on the pathogenesis and epizootiology of various serotypes is important in developing effective treatment, prevention and control methods (Manning *et al.*, 1989).

References

Al-Haddawi, M.H., Jasni, S., Mutalib, A.R, Zamri-Saad, M., and Sheikh-Omar, A.R. (1998a). *J. Vet. Malaysia*, (1998) 10: 41-45

Al-Haddawi, M.H., Jasni, S., Zamri-Saad, M., Mutalib, A.R., and Sheikh-Omar, A.R. (1999). *Res. Vet. Sci.*, 67: 163-170.

Al-Haddawi, M.H., Jasni, S., Zamri-Saad, M., Mutalib, A.R., Son, R. and Sheikh-Omar, A.R.(1998b). *Proceedings of the 7th Scientific Conference of the Electron Microscopy Society Malaysia* (pp 145-152).

Al-Haddawi, M.H., Jasni, S., Zamri-Saad, M., Mutalib, A.R., Son, R., Zulkifli, I. and Sheikh-Omar, A.R. (2000a). *Vet. J.* 159:274-281

Al-Haddawi, M.H., Jasni, S., Zamri-Saad, M., Mutalib, A.R., Son, R. and Sheikh-Omar, A.R. (2000b). *Vet. Res. Comm.*, 24 (3):153-167

Benirschke, K., Garner, F.M. and Joes, T.C. (1978). *Pathology of Laboratory Animals*, II. (ppl433-1434). New York: Springer-Verlag.

Carter,G.R. (1967). Adv.Vet.Sci.Comp.Med., 11:321-329.

Choi-Kim, K., Mahesuaran, S.K., Felice, L.J., and Molitor, T.W (1991). *Vet. Microbiol.*, 28:75-92.

Chrisp, C.E., and Foged, N.T. (1991). Am.J.Vet.Res., 52:56-61.

DiGiacomo, R.F., Garlinghouse, E.L., and Van Hoosier, G.L. (1983). *J. Am. Vet. Med Assoc.*, 183:1172-1175.

Flatt, R.E. (1974). *The Biology of the Laboratory Rabbits.* (pp.194-205).New York Academic Press.

Fortin, M., and Jacques, M. (1987). *J. Clin. Microbiol.*, 25:938-939.

Glorioso, J.C., Jones, G.W., Rush, H.G, Pentler, L.J., DaRif, C.A and Coward, J.E. (1982). *Infect. Immun.* 35:1103-1109.

Jasni, S., Al-Haddawi, M. H., Zamri-Saad, M., Mutalib, A.R and Sheikh-Omar, A. R. (1999). *J. Vet. Malaysia* 11 (1):41-44.

Kawamoto, E. Sawada, T., and Maruyama, T. (1990a). *Jpn. J. Vet. Sci.*, 52:915-921.

Kawamoto, E. Sawada, T., Suzuki K., and Maruyama, T. (1990b). *Jpn. J. Vet. Sci.*, 52:1277-1279.

Latimer, K.S., Harmon, B.G., Glisson, J.R., Kircher, I.M. and Brown, J. (1990). *Avian Dis.*, 34,137-140.

Lu, Y.S., Pakes, S. P., and Stefanu C. (1983). *J. Clin. Microbiol.*, 18:292-295.

Manning, J.P., DiGiacomo, R. F., and Delong, D. (1989). *Pasteurella and Pasteurellosis.* (pp 263-302). Academic Press.

Scharmann, W., Drzeniek, R. and Blobel, H. (1970). *Infect. Immun.*, 1:319-320.

Soltys, M.A. (1979). *Introduction to Veterinary Microbiology*, pp. 158-167