A HERD OUTBREAK OF PASTEURELLA PNEUMONIA OF THE PIGS PATHOLOGICAL AND EPIDEMIOLOGICAL FIELD STUDIES

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It has been described that acute pneumonia of the fattening pigs is usually caused by the infection with Pasteurella(P) multocida, but this agent is generally considered to be a secondary invader in producing of a pneumonia (Bentley and Farrington, 1980; Little, 1975). Little and Harding indicated that Haemophilus parahaemoliticus was capable to initiate sufficent damage of the lungs to allow invading of P. multocida and produce a severe pneumonia in pigs. Pijoan and Ochoa demonstrated on interaction between live attenuated hog cholera virus and P. multocida producing a pneumonia in pigs.

P. multocida is a common pathogen of porcine respiratory disease in Korea and hog cholera is also widesspread in the country. The pigs are usually vaccinated with rapinized hog cholera live virus around 40 days old. There was a serious outbreak of the pneumonic disease in an intensive pig farm from September through November in 1980. The piglets of the farm were vaccinated with rapinized hog cholera live virus between 25 and 30 days old but the piglets began to show severe pneumonic symptoms and diarrhea between 35 and 60 days old. Onset of the disease appeared to be sudden and its course was lasted for 4 to 10 days with usually a fatal termination. The morbidity and mortality ranged from 20 to 30 per cent and from 50 to 70 per cent respectively. The total losses were more than 800 heads of piglets from the disease.

Twenty carcases which showed typical symptoms were necropsied and examined for histopathological and microbiological studies. Portions of pneumonic lungs and the other organs were taken and homogenized. Swabs from the emulsion were plated on blood agar or incoulated on the selective media for isolation of the several bacteria.

All of the carcases except one represented prominent pneumonic lesion and most of the pneumonic cases were complicated with other lesions such as necrotic typhlocolitis. Macroscopically the lungs of nine piglets were characterized by severe fibrinous pneumonia with pleuritis. In the acute cases of the pneumonia, constant lesions such as lobar consolidation characterized by fibrinous pleurisy were observed in the affected lung lobes. The cardiac and apical lobes were the most severly affected parts followed by anterioventral portion of the diaphragmatic lobes. Excess accumulation of serosanguinous fluid mixed with strands of fibrin was found in the pleural and pericardial cavities. There was a often well developed overlying pleurisy with fibrinous adhesion to thoracic wall and pericardium. On cut surface of the affected lobes, firm hepatized areas often with centrally located necrotic foci of various shape and size, and broad band of gelatinous septa were observed.

Histologically in these cases the broad areas of alveoli contained serofibrinous exudate and some other alveoli and bronchioles were filled with purulent exudate. Most of the bronchioles were dilated by significant amount of the copious exudate. Fibrinous thrombi were present in many vessels throughout the lungs. Severe edematous and fibrinous involvement of the interlobular septa was most striking feature of the histology. Most of the septal lymphatics were occluded by fibrinous and cellular clots which may provoke prognosis of pneumonia worse. There were areas of necrosis of varying size and shape outlined by zone of mononuclear cells and fibroblast layers oriented in a characteristic swirling pattern.

The remainings of the 19 pneumonic cases revealed the lesions of purlent bronchopneumonia. The pneumonic changes in these cases extended to lobular or sublobar distribtion. There were also seen bronchiolar obstruction, considerable involvement of the interlobular septa, hemorrhage into alveoli or thrombosis of small vessels.

Microbiologically heavy infection of P. multocida was seen on the lungs of all pneumonic cases in conbination with Staphylococcus aureus. Two infected carcases among the twenty were diagnosed as hog cholera by histopathological examination and fluorescent antibody technique.

P. multocida swine pneumonia models have required multiple agents such as other bacteria, viruses or enviromental stresses (Bentley and Farrington, 1980; Kielstein et al, 1977; Little and Harding, 1980; Raynaud et al, 1977; Smith, 1970). All of the affected piglets in the farm were farrowed in the poorly sanitated houses neglecting all in and all out system. Therefore most of the piglets which were farrowed in these houses might be affected heavily with the bacteria such as P. multocida. Moreover hog cholera vaccinal virus by which the piglets were injected between 25 to 30 days old might predispose pigs to infection by P. multocida to allow production of a severe pneumonia.

Conclusions:

A herd outbreak of pasteurella swine pneumonia followed by severe losses of the piglets has been recorded. The lung lesions of the affected piglets showed distinct fibrinous pleuropneumonia or purulent bronchopneumonia with striking involvement of the interlobular septa. From the point of pathological and epidemiological studies, the epizootics migh be discussed with interrelationship between pathogenicity of P. multocida and hog cholera vaccinal virus or enviromental stresses.

Selected references: Bentley, E. and Farrington, D.J: Am. J. Vet. Res. 1980, 41: 1870; Kielstein, P. et al: Arch. Exp. Veterinaermed. 1977, 31, 609; Little, T. W.A.: Vet. Rec. 1975, 96:540; Little, T.W.A. and Harding, D.J.: Br. Vet. J. 1980, 136, 371; Pijoan, C. and Ochoa, G.: Proc. Int. Pig. Vet. Soc. Congress, 1980, Copenhagen, Denmark, 195; Raynaud, J.P. et al: J. Rech. Porcine. 1977, 165; Smith, I.M.: Ph.D. Thesis, 1970, University of London.