Effect of *Mycoplasma hyopneumoniae* Infection on the Development of *Haemophilus pleuropneumoniae* Pneumonia in Pigs

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(Received 2 February 1984/Accepted 4 July 1984)

ABSTRACT. The exacerbating effect of *Mycoplasma hyopneumoniae* (Mh) infection on the development of pneumonia caused by subsequent infection with *Haemophilus pleuropneumoniae* (Hp) was investigated in pigs. Pigs were inoculated intranasally with 10^7 cells of Mh, followed 16 days later by an intranasal inoculation of 10^5 cells of Hp. Other pigs were given either organism alone by the identical manner. One pig infected with both agents died 3 days after Hp inoculation, and had necrotizing, hemorrhagic fibrinous pleuropneumonia. All other pigs were killed 35 days after Mh inoculation. Pigs infected with both organisms showed more severe clinical signs, more extensive and widespread pneumonic lesions characterized by chronic form with necrotic nodules and pleurisy when compared with pigs infected with either organism alone. Extrapulmonary dissemination of both organisms occurred more frequently in doubly infected pigs than in pigs infected with either agent alone. These results indicate that previous infection of Mh acts as one of predisposed factors exacerbating porcine haemophilus pleuropneumonia. There were no correlations between antibody titers to Hp and intensity of lung lesions. —**Key words:** *Haemophilus pleuropneumoniae*, mixed infection, *Mycoplasma hyopneumoniae*, swine pneumonia.

Jpn. J. Vet. Sci. 46(5): 705–713, 1984

INTRODUCTION

Porcine haemophilus pleuropneumonia (PHP) induced by *Haemophilus pleuropneumoniae* (Hp) has been recognized in many countries and causes great economic loss in the swine industry [20]. The disease is characterized by either peracute to acute fibrinohemorrhagic pneumonia or chronic and necrotizing pneumonia with pleurisy. It was found in survey at abattoirs that Hp could be isolated frequently from chronic pneumonic lesions with nodular formation [4, 9, 14]. However, factors involved in the difference linking acute and chronic forms of the disease have not been fully elucidated.

Mycoplasma hyopneumoniae (Mh) is the etiological agent of swine enzootic pneumonia which has frequent occurrence and worldwide distribution [17]. Pneumonic lesions which appeared to be caused by a

combined action of both Hp and mycoplasma have often been found [4, 9]. However, exacerbating effect of mycoplasma on the development of PHP has not been demonstrated. The present study was undertaken to answer this question.

MATERIALS AND METHODS

Animals: Thirteen specific-pathogen-free miniature pigs of the Pitman-Moore strain, 50-day-old, known to be free of mycoplasma and haemophilus, were obtained from the Nippon Institute for Biological Science, Laboratory Animal Research Station, Kobuchizawa, Yamanashi Prefecture. The animals were derived from 2 litters.

Inocula: The MI-3 strain of Mh [23] was isolated at our laboratory from the lungs of a pig with swine enzootic pneumonia and maintained by serial passage in pigs. It was used at passage 7. The inoculum containing 10^7

color-changing units (CCU)/ml was prepared by the method described elsewhere [23]. The suspension was bacteriologically sterile when cultured on blood agar medium, and viruses were not demonstrated in primary swine testis and PK-15 cell cultures or by chicken embryo inoculation test.

The SHP-1 strain of Hp belonging to serovar 2, which is widely distributed among pigs in Japan [4], was used. This strain was isolated at our laboratory from the lungs of a pig with pneumonic lesions, and was pathogenic for pigs when inoculated intranasally with 10^7 organisms [12]. The organism was cultured overnight on heart infusion agar (Nissui Seiyaku Co., Ltd., Tokyo, Japan) plates with 5% fresh yeast extract. A suspension containing 3×10^5 colony-forming units (CFU)/ml was prepared in phosphate-buffered saline (pH 7.2) and used as an inoculum.

Tryptose phosphate broth (TPB, Difco Laboratories, Detroit, U.S.A.), which was used for preparing the mycoplasma inoculum, was used as an inoculum for uninfected control pigs.

Experimental design: Thirteen pigs were divided into 4 groups consisting of 5, 2, 4 and 2 pigs. Pigs (Nos. 1 to 5) in group 1 and pigs (Nos. 6 and 7) in group 2 were inoculated intranasally with 5 ml of the Mh inoculum. Sixteen days after inoculation of Mh, the pigs in group 1 and pigs (Nos. 8 to 11) in group 3 were inoculated intranasally with 1.5 ml of the Hp inoculum. Uninfected control pigs (Nos. 12 and 13) in group 4 were given intranasally 5 ml of TPB at the time when the pigs in groups 1 and 2 received the Mh inoculum. Each group of pigs was housed separately under conventional conditions and special care was taken to avoid cross-contamination among the groups. The animals had free access to drinking water and pelleted feed without antibiotics. They were observed daily for clinical signs, and the group mean body-weights were determined at the time of inoculation and 19 days after inoculation with

Hp. Pig No. 4 died on day 3 post-inoculation (PI) with Hp and the remainders killed at day 35 PI with Mh or TPB were subjected to pathological examinations and recovery of the organisms.

Pathological examinations: After lung lesions were observed grossly, specimens for light microscopy were collected and fixed in 10% formalin solution. They were embedded in paraffin, sectioned and stained with hematoxylin and eosin(H-E).

Recovery of organisms: LGM-5 liquid medium [26] consisting of Hanks' balanced salt solution (45 ml), Hartley broth (30 ml), inactivated swine serum (20 ml), lactalbumin hydrolysate (0.5 g, Nutritional Biochemicals Corporation, Cleveland, U.S.A.), 25% fresh yeast extract (5 ml), 2.5% thallium acetate (1 ml), 100,000 U of penicillin G potassium and 0.5% phenol-red (0.4 ml) was used for Mh isolation. Tissues from consolidated areas of the lung, bronchial lymph node and spleen were homogenized in TPB to make a 10% suspension. In cases without gross pneumonia, tissue was collected from the apical lobe of the right lung, and processed similarly. CCU present in the suspensions were determined by the method described by Denny *et al.* [6]. Swabbed samples of tracheal mucus were also cultured. The cultures were incubated at 37°C for 21 days, during which time they were observed for growth of mycoplasma as indicated by change in color of the medium. Isolates were identified as Mh by the disk growth-inhibition test [25].

For Hp isolation, chocolate agar plates containing 10% horse blood were used. A 10% suspension of lung tissue described above was quantitatively cultivated by plating serial 10-fold dilutions. Portions of the nasal turbinate, tracheal mucus, bronchial lymph node, liver and spleen were streaked on the agar plates. The cultures were incubated at 37°C for 2 days in an atmosphere containing 10% CO₂ in the air. Except plates inoculated with lung samples, 1 to 50, 51 to 100 and 101

Fig. 1. Diagrammatic sketch of lungs, showing anatomical distribution of lesions in pigs inoculated with *M. hyopneumoniae* and/or *H. pleuropneumoniae* or tryptose phosphate broth.

or more of colonies grown per plate were expressed as +, ++ and +++, respectively. Isolates were identified as Hp by observing colony appearance and a Gram-stained preparation, and by the slide agglutination test with specific antiserum prepared in rabbits.

Serological test: Blood samples were taken from the jugular vein of pigs just before the inoculation with Hp and at the time of euthanasia. Sera were separated and stored at -20°C . The latex agglutination test [12] was used for detection of Hp antibody.

RESULTS

Clinical signs

Clinical signs appeared in pigs in groups 1 and 3 after they were inoculated with Hp. The animals were reluctant to move, showing inappetence, coughing, nasal discharge and a rectal temperature of $41-42^{\circ}\text{C}$. These signs were more severe in pigs in group 1 than those in group 3 and continuously observed for 4–10 days, while clinical signs in pigs in

group 3 were improved by day 2 to day 5 PI with Hp. Pig No. 4 in group 1 died on day 3 PI with Hp, showing severe acute signs. No pigs in groups 2 and 4 had clinical signs throughout the observation period. The group mean body-weight was apparently affected by mixed infection. That in group 1 was deprived 0.15 kg, whereas those in groups 2, 3 and 4 gained 2, 0.5 and 1.7 kg, respectively.

Gross lesions

The anatomical distribution of gross lung lesions in pigs in the 4 groups is shown in Fig. 1. In pigs in group 2, which were inoculated with Mh alone, the lungs showed characteristic, well-defined plum-red to gray atelectatic foci with frequent occurrence of lesions in cardiac and apical lobes.

Pigs in group 3, which were inoculated with Hp alone, developed multiple, reddish nodular lesions demarcated clearly from the surrounding tissues. The lesions varied in size and were randomly distributed in lobes of each lung with occasional fibrous adhesion to

Fig. 2. A section from the lung of a pig infected with *M. hyopneumoniae* alone, showing peribronchial lymphoid cell infiltration and atelectasis of the surrounding alveoli due to exudation. H-E stain. $\times 12$.

Fig. 3. A section from the lung of a pig infected with *H. pleuropneumoniae* alone, showing a part of necrotic focus of parenchyma surrounded by capsule of proliferated connective tissue and apparently intact nearby tissues. H-E stain. $\times 12$.

the parietal pleura.

In pigs in group 1 infected with both organisms, the most extensive changes were observed in pig No. 4 that died on day 3 PI. All surfaces of both lungs and the pericardium were covered with a grayish-yellow exudate, and a large amount of blood-stained fluid was present in the pleural and pericardial cavities. On the cut surface, lesions were multiple with extensive congestion and hemorrhage. All surviving pigs in the same group revealed well-demarcated necrotic nodules similar to those seen in Hp-infected pigs and consolidated areas appearing to be caused by mycoplasma in their lungs. But the intensity and frequency of these lesions were higher than those in pigs infected with either agent alone.

Two uninfected control pigs had no gross

lesions.

Histopathological lesions

One of conspicuous features in the lungs of pigs in group 2 was perivascular, peribronchiolar and peribronchial cuffings by lymphocytes and plasma cells, and the other was pneumonic foci which developed in close proximity to lesions of the respiratory tract (Fig. 2). These changes were similar to those previously reported [23].

The lungs of pigs in group 3 were characterized by the formation of multiple abscess-like nodules which were distributed throughout the entire lobes, sometimes causing local adhesive pleurisy (Fig. 3). The center of most nodules consisting of necrotic lung tissues was surrounded by abundant neutrophils and mononuclear cells with pyknotic nuclei.

Fig. 4. A section from the lung of a pig infected with *M. hyopneumoniae* and *H. pleuropneumoniae* and died 3 days after inoculation with *H. pleuropneumoniae*. Irregular areas of coagulative necrosis with severe edema of the interlobular septa and hemorrhage in the surrounding parenchyma. H-E stain. $\times 12$.

They were well-demarcated from the surrounding lung tissue by extensively proliferated connective tissue, which was infiltrated with numerous macrophages, plasma cells and a few neutrophils.

Pigs infected with both organisms showed most extensive and widespread lesions as compared with those in other 2 infected groups. Pig No. 4 which died on day 3 PI had changes in the lungs which appeared as a necrotizing, hemorrhagic, fibrinous pleuropneumonia (Fig. 4). The lobules were separated into distinct compartments due to severe inflammatory edema caused by lymphangitis and lymphangiectasis in the interlobular and peribronchial connective tissues. Blood vessels were greatly dilated and frequently ac-

Fig. 5. A section from the lung of a pig infected with *M. hyopneumoniae* and *H. pleuropneumoniae* and killed 19 days after inoculation with *H. pleuropneumoniae*, showing abscess-like nodules similar to those induced by single infection with *H. pleuropneumoniae* and severe pneumonic changes in the surrounding tissues. Single arrow points to desquamation of bronchial epithelial cells and double arrows point to purulent exudation in another bronchus. H-E stain. $\times 12$.

companied by intraalveolar hemorrhage. There were marked serofibrinous and purulent exudation and desquamation of the epithelial cells into the alveoli. The lungs of the other 4 pigs in this group which were killed at day 19 PI with Hp were characterized by the formation of multiple abscess-like nodules accompanied by purulent bronchopneumonia (Fig. 5). These nodular lesions were similar fundamentally to those seen in the lungs of pigs infected with Hp alone, but their occurrence and intensity were more remarkable. Most of the bronchi and bronchioles had marked deciliation and exfoliation of epi-

Table 1. Bacteriological and serological examinations in pigs inoculated with *M. hyopneumoniae* and/or *H. pleuropneumoniae* or tryptose phosphate broth.

Group (Exposure)	Pig No.	Isolation of										Antibody ^{a)} titer to Hp
		Mh				Hp						
		T	L	Bl	S	N	T	L	Bl	Li	S	
1 (Mh and Hp)	1	Pos	7 ^{b)}	3 ^{b)}	Neg	+++ ^{c)}	+++	7 ^{b)}	++	++	Neg	1:64
	2	Pos	6	4	Neg	+++	+++	8	++	++	+	1:64
	3	Pos	3	6	Neg	ND	Neg	8	+++	Neg	Neg	1:64
	4	Pos	5	ND ^{d)}	ND	+	+++	8	ND	+++	+	ND
	5	Pos	7	1	Neg	++	++	8	+++	+	+	1:32
2 (Mh)	6	Neg	7	Neg	1	Neg	Neg	Neg	ND	ND	ND	<1:8
	7	Neg	7	1	Neg	Neg	Neg	Neg	ND	ND	ND	<1:8
3 (Hp)	8	Neg	Neg	ND	ND	+	Neg	8	Neg	Neg	Neg	1:32
	9	Neg	Neg	ND	ND	ND	Neg	8	Neg	Neg	Neg	1:128
	10	Neg	Neg	ND	ND	+	Neg	8	+++	Neg	Neg	1:8
	11	Neg	Neg	ND	ND	+	Neg	7	+++	+	+	1:64
4 (TPB)	12	Neg	Neg	ND	ND	Neg	Neg	Neg	ND	ND	ND	<1:8
	13	Neg	Neg	ND	ND	Neg	Neg	Neg	ND	ND	ND	<1:8

a) Latex agglutination test.

b) Log₁₀ CCU of Mh/10 mg or log₁₀ CFU of Hp/10 mg of tissue.

c) Number of Hp colonies/agar plate (see text).

d) not determined.

Mh=*M. hyopneumoniae*; Hp=*H. pleuropneumoniae*; TPB=tryptose phosphate broth;

T=trachea; L=lung; Bl=bronchial lymph node; S=spleen; N=nasal turbinate; L=liver.

lial cells with severe exudation of neutrophils into their lumina.

Pigs in group 4 had no histological changes in their lungs.

Recovery of organisms

Mh was isolated from lung lesions of all pigs in groups 1 and 2, attaining viable titers from 10³ to 10⁷ CCU/10 mg of tissue. The organisms were isolated from the trachea of all animals in group 1, whereas no mycoplasmas were recovered from this site of pigs in group 2. The bronchial lymph nodes of 4 pigs in group 1 contained mycoplasmas ranging from 10¹ to 10⁶ CCU/10 mg of tissue, while only 10¹ CCU/10 mg were recovered from the node of 1 of 2 pigs in group 2 (Table 1).

Hp was recovered from lung lesions of all inoculated pigs and titers reached 10⁷ to 10⁸ CFU/10 mg of tissue. There were no differ-

ences in the titers of the organism between pigs in groups 1 and 3. Recoveries of the organism from the upper respiratory tract, bronchial lymph node, liver and spleen of pigs in group 1 were higher in the frequency of isolation and in the number of organisms than those from pigs in group 3 (Table 1).

Neither Mh nor Hp was isolated from uninfected control pigs in group 4.

Detection of serum antibody

Antibody titers to Hp as determined by the latex agglutination test were less than 1:8 in all serum samples collected before inoculation. The titers determined at day 19 PI with Hp ranged from 1:8 to 1:128 (Table 1). There was no correlation between the antibody level and severity of pneumonic lesions.

DISCUSSION

It has been reported that the occurrence and

severity of PHP might be affected by such unfavorable conditions as transportation of animals to slaughterhouse [14], management factors with overcrowding and poor ventilation [13, 16], extremely cold conditions and sudden changes of weather [16, 18]. The concomitant infection with various microorganisms including mycoplasma has also been shown to exacerbate the condition [4, 9, 10, 15, 19]. In the present study, pigs inoculated with both Mh and Hp were more severely affected than could be accounted for by the additive effect of each organism, indicating synergism between both organisms to produce a more severe disease. In doubly infected pigs, clinical signs and gross lung lesions developed more severely and both organisms multiplied to greater extent than in pigs infected with either organism alone.

The synergistic effect between Mh and Hp was also demonstrated by histologic examinations. Lung lesions in pigs infected with Hp alone were distributed in most of the lobes and all lesions were localized and clearly demarcated from the surrounding intact tissues, whereas in doubly infected pigs, severe purulent bronchopneumonia developed in association with necrotic foci. These findings suggested that lesions observed in pigs with mixed infection were not only overlapping of those lesions provoked by each agent but reflected synergism between the two.

There was no apparent difference in the number of Hp isolated from the lungs between pigs in groups 1 and 3 (Table 1). It was presumed, however, that a total number of the organisms grown in the whole lungs of a pig in group 1 may be much greater than the corresponding number in group 3, since lung lesions in pigs in group 1 were more severe and widespread than in those in group 3. This may be one of factors to cause the extrapulmonary dissemination of Hp in pigs infected with both organisms.

The synergistic effects have been investigated by many workers between various my-

coplasmas and bacteria in different hosts. It has been demonstrated in most of these studies [2, 3, 7, 11] that concomitant mycoplasmal and bacterial infections have tended to result in exacerbation of diseases. On the contrary, Howard *et al.* [8] reported that pneumonia induced by *M. pulmonis* in mice did not decrease resistance to subsequent *Staphylococcus aureus* infection, suggesting that outcomes of infections may be influenced by many factors associated with different hosts and organisms.

The exact mechanism by which a previous infection with Mh may affect the host's resistance and render the lung more susceptible to bacterial invasion remains uncertain. Mh infection might enable Hp to colonize efficiently the mucosal surface of the respiratory system by impairment of the mucociliary clearance mechanism. It has been well documented that cilia were injured functionally and structurally by the attachment of mycoplasmas to, and their colonization on, the mucosal surface of the respiratory and genital tracts [5, 22, 24]. It has also been reported that some mycoplasmas impaired phagocytosis of bacteria by neutrophils [21], and that they exerted an immunosuppressive effect on the host [1]. It is likely from these observations that dysfunction of the pulmonary antibacterial defence induced by mycoplasmal infection accelerated the multiplication of Hp in the respiratory system, resulting in extrapulmonary dissemination of the organism.

ACKNOWLEDGEMENTS. The authors thank Y. Shimada for his skilful technical assistance. This work was supported in part by a Grant-in-Aid for Scientific Research (no. 58480085) from the Ministry of Education, Science and Culture of Japan.

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要 約

豚の *Haemophilus pleuropneumoniae* による肺炎における *Mycoplasma hyopneumoniae* 感染の影響: 八木橋武・布谷鉄夫・三井正朗・田島正典 (財・日本生物科学研究所)——豚の *Haemophilus pleuropneumoniae* による肺炎における *Mycoplasma hyopneumoniae* 感染の影響を解明するため, *M. hyopneumoniae* (10^7) を鼻腔内接種後16日に *H. pleuropneumoniae* (10^5) を同経路から接種した豚 (MH豚) と, それぞれの菌を単独接種した豚 (MおよびH豚) との病態を比較した。MH豚の1頭がヘモフィルス接種後3日に死亡し, 壊死性, 出血性, 繊維素性胸膜肺炎を呈した。MH豚はH豚に比べ強い症状を示し, マイコプラズマ接種後35日の剖検で壊死性結節を含む慢性型肺病変と胸膜炎が認められ, その程度はH豚に比べより強かった。MH豚の肺以外の部位における両菌の増殖は, H豚あるいはM豚のそれに比べ高度であった。これらの結果から先行した *M. hyopneumoniae* の感染によって, 豚のヘモフィルス胸膜肺炎が増悪されることが示唆された。肺病変の強さと *H. pleuropneumoniae* に対する抗体力価との間には相関は認められなかった。