Res. Bull. Obihiro Univ., **20** (1997) : 113～122

Pathology of Naturally Occurring Porcine Adenovirus Type 4 Infection in Japan

Dewi Ratih AGUNGPRIYONO*, Michio NAKAGAWA**, Tetsuo MOROZUMI[1)], Shinobu YOSHIHARA[2)], Hidefumi FURUOKA, Takane MATSUI

(Received : November 11, 1996)

ABSTRACT

Porcine adenovirus infection was accidentally detected in 12 of 19 pigs that were used for studies on experimental *Haemophilus parasuis* infection. Histopathologically, intranuclear inclusion bodies were found in the small intestines of 9 pigs, kidneys of 5 pigs, lymph nodes of 5 pigs, large intestines of 2 pigs and nasal mucosa of 1 pig. The inclusions were associated with interstitial nephritis, degeneration and desquamation of epithelial cells in the renal tubules and intestinal mucosa. Nonsuppurative encephalitis was found in 10 pigs, but they did not exhibit any neuronal signs or inclusion formation in their brains. Electron microscopy demonstrated numerous viral particles, typical of adenoviruses, in the intranuclear inclusions, and the virus was identified as serotype 4 by immunohistochemistry. Ten of 12 infected pigs were concurrently infected with cryptosporidium, which did not cause any morphological alteration or clinical disease. The pigs with the present adenovirus type 4 infection developed neither clinical signs before bacterial inoculation nor macroscopic lesions in the kidneys, intestinal mucosa and lymph nodes suggesting that the virus is of the asymptomatic type.

Key words : adenovirus, encephalitis, inclusion body, nephritis, swine.

Department of Veterinary Pathology, Obihiro University of Agriculture and Veterinary Medicine, Obihiro, Hokkaido 080, [1)] National Institute of Animal Health, 3-1-1 Kannondai, Tsukuba, Ibaraki 305, [2)] National Institute of Animal Industry, Ikenodai 2, Kukizakimachi Inashikigun Ibaraki 305, Japan

*Present address : Department of Veterinary Pathology, Faculty of Agriculture, Miyazaki University, Gakuenkihanadainishi1-1, Miyazaki city 889-21

**Correspondence to : M. NAKAGAWA, Department of Veterinary Pathology, Obihiro University of Agriculture and Veterinary Medicine

080 帯広市稲田町　帯広畜産大学家畜病理学教室，305 茨城県つくば市観音台3-1-1　家畜衛生試験場，305 茨城県稲敷郡茎崎町池の台２　畜産試験場

*現住所：889-21　宮崎市学園木花台西1-1　宮崎大学農学部家畜病理学教室

**連絡先：中川迪夫　帯広畜産大学家畜病理学教室

Porcine adenovirus (PAV) has been associated with encephalitis (Kasza, 1966), enteritis (Abid et al., 1984 ; Coussement et al., 1981 ; Fujiwara et al., 1968 ; Sanford and Hoover, 1983), pneumonia (Schiefer et al., 1974), interstitial nephritis (Nietfeld and Leslie-Steen, 1993), abortion (Kirkbride and McAdaragh, 1978), and transplacental fetal infection (Narita et al., 1985) in pigs. However, its significance as a causal agent of disease in the field remains controversial (Buller and Moxley, 1988 ; Kawamura et al., 1972 ; Nietfeld and Leslie-Steen, 1993 Sanford and Hoover, 1983).

In Japan, PAV was isolated from a normal piglet (Kawamura et al., 1972) and from pigs affected with toxoplasmosis (Shimizu et al., 1978). Based on serological surveys, PAV types 1 to 5 have been known to be prevalent among pigs in this country (Hirahara et al., 1990 ; Kawamura et al., 1972 ; Shimizu et al., 1978). There are a few reports dealing with the histopathology of naturally occurring PAV infection (Fujiwara et al., 1968 ; Narita et al., 1985).

In the present study, natural cases of PAV infection in pigs were examined by light microscopy, immunohistochemistry, and electron microscopy to shed some light on the pathology of adenovirus infection.

MATERIALS AND METHODS

Pigs : The pigs were obtained from a farm without a history of outbreak of PAV infection and were used for experimental infection with *H. parasuis* (Table 1). The first group consisted of ten 40-day-old pigs from two litters. The second group consisted of nine 61-day-old pigs from one litter. Before the experimental infection, all the pigs were isolated for 34 days for the first group and 5 days for the second group. No abnormal clinical signs were observed during these periods.

Light microscopy : At necropsy, tissue blocks from almost all organs were fixed in 10% buffered formalin and embedded in paraffin. Sections were stained with hematoxylin and eosin (HE) and some selected sections were tested by Feulgen reaction.

Immunohistochemistry : Sections were stained with an avidin biotin-peroxidase complex (ABC) Kit (Vector Lab., Burlingame, CA, U. S. A.). Rabbit anti-type 1, 2, 3 and 4 PAV sera (supplied by Dr. H. Hamada, Central Research Lab., Kyoritsushoji Co., Ltd.) were used as the primary antibodies at a dilution of 1 : 512. Sections were counterstained with methyl green. For the negative control test, nonimmunized rabbit serum was used in place of the primary antibody.

Electron microscopy : Small blocks of formalin-fixed tissues from the renal medullary region and Payer's patch region in the small intestine were postfixed in 1% Millonig's osmium tetroxide (pH 7.2), dehydrated with graded alcohols and embedded in Epon 812. Ultrathin sections were stained with lead citrate and uranyl acetate and examined with an electron microscope.

RESULTS

Clinical findings : First group ; pig no. 1 died at day 1 postinoculation (p.i.) after showing pyrexia and depression, while the other inoculated and control pigs showed no clinical signs.

Second group ; pig no. 1 died at 5 hours p.i., Pig no. 2 showed pyrexia, loss of appetite and depression at day 3 p.i., but recovered a few days later. The others remained without clinical signs. Pig no. 7 died 1 hour after intravenous injection with 4 ml of saline containing 4 mg of lipopolysaccharide of *H. parasuis* strain No. 4 at day 20 p.i..

Macroscopical findings : Macroscopic findings are summarized in Table 1.

Histological findings : As shown in Table 2, there were cerebral inflammataion, serositis, arthritis, pneumonia, and cryptosporidial infection in the small intestine.

Inclusion bodies ; Five pigs in the first group

Table 1. Experimental infection of pigs with *Haemophilus parasuis* and necropsy findings.

Group no.	Pig no.	Route of infection	Dose of inoculation (Strain no.)	Clinical observation period	Termination	Necropsy findings
I	1.	i. v.	1.3×10^9 (no. 4)	1 day	Died	Serositis, arthritis, meningitis.
	2.	i. v.	1.3×10^9 (no. 4)	6 days	Killed	Fibrinous exudates on serosa of small intestine and gall bladder; Pulmonary hepatization.
	3.	i. v.	1.3×10^8 (no. 4)	6 days	Killed	Fibrinous exudates on splenic capsule.
	4.	i. v.	1.3×10^8 (no. 4)	6 days	Killed	Fibrinous exudates on splenic capsule and synovia.
	5.	Control	-	7 days	Killed	No lesions.
	6.	i. p.	1.3×10^9 (no. 4)	6 days	Killed	No lesions.
	7.	i. p.	1.3×10^9 (no. 4)	6 days	Killed	No lesions.
	8.	i. p.	1.3×10^8 (no. 4)	6 days	Killed	Pulmonary hepatization.
	9.	i. p.	1.3×10^8 (no. 4)	6 days	Killed	No lesions.
	10.	Control	-	7 days	Killed	Pulmonary hepatization.
II	1.	i. t.	1.3×10^{10} (no. 4)	5 hours	Died	Pulmonary edema, subepicardial haemorrhage.
	2.	i. t.	1.3×10^{10} (no. 4)	9 days	Killed	Serositis and pulmonary hepatization.
	3.	i. t.	1.3×10^9 (no. 4)	9 das	Killed	No lesions.
	4.	i. t.	1.3×10^9 (no. 4)	9 days	Killed	Pulmonary hepatization.
	5.	i. t.	1.0×10^{10} (no. 140)	14 days	Killed	Pulmonary hepatization.
	6.	i. t.	1.0×10^{10} (no. 140)	6 days	Killed	Pulmonary hepatization.
	7.※	i. t.	1.0×10^9 (no. 140)	20 days	Died	Edema of lung and gallbladder, round heart.
	8.	i. t.	1.0×10^9 (no. 140)	21 days	Killed	No lesions.
	9.	Control	-	9 days	Killed	Pulmonary hepatization.

i. v.: intravenous
i. p.: intraperitoneal
i. t.: intratracheal
※ : Pig no. 7 died 1 hour after intraenous injection with 4ml of saline containing 4 mg of lipopolisaccharide of *H. parasuis* strain No. 4 at day 20 p. i.

showed intranuclear inclusion bodies in the medullary tubular epithelial cells associated with mild lympho-plasmacytic infiltration in the peritubular connective tissue of the kidney (Fig.1a). The inclusions were occasionally located in the swollen nuclei of the collecting tubular epithelium in the medulla, or in desquamated epithelial cells in the tubular lumen. Morphologically, 3 types of intranuclear inclusions, full, classical type A, and granular were distinguished (Figs. 1b and 1c). The full type inclusion consisted of a basophilic, homogeneous mass occupying the whole nuclear space; classical type A inclusion was an amphophilic mass, centrally located in the nucleus, and

Table 2. Distribution and frequency of the intranuclear inclusion bodies and other histopathologic findings

Group and Pig no.	Distribution and frequncy of the Inclusion bodies					Other histopathological findings						
						Brain lesion			Crypto-	Serositis	Arthritis	Pneumo
	Kidney	Small intestine	Large intestine	Lymph node	Nasal mucosa	Lymphocytic cuffing	Glial nodule	Fibrinous meningitis	sporidia			nia
I-1	+	−	−	+	−	−	−	○	−	○	○	−
I-2	+++	+	−	−	−	○	○	−	○	○	○	○
I-3	−	−	−	−	−	−	−	−	−	○	○	−
I-4	−	−	−	−	−	−	−	−	−	○	○	−
I-5	−	−	−	−	−	○	○	−	○	○	−	−
I-6	+	−	−	−	−	○	○	−	○	○	−	−
I-7	+++	+	−	+	−	○	○	−	−	−	−	−
I-8	−	−	−	−	−	○	−	−	−	−	−	○
I-9	+++	++	−	+	−	−	−	−	○	−	−	○
I-10	−	−	−	−	−	○	−	−	−	○	−	○
II-1	−	−	+	−	−	−	−	−	○	−	−	−
II-2	−	++	+	−	−	○	○	−	○	○	−	○
II-3	−	+	−	+	−	−	−	−	○	−	−	−
II-4	−	++	−	−	+	−	−	−	○	−	−	○
II-5	−	−	−	−	−	−	−	−	−	−	−	○
II-6	−	−	−	−	−	○	○	−	○	−	−	○
II-7	−	+	−	−	−	−	−	−	○	−	−	−
II-8	−	++	−	+	−	○	○	−	○	○	−	○
II-9	−	++	−	−	−	○	−	−	○	−	−	○

− : No inclusion body or no lesion
○ : Lesion appeared
+ : Mean number of inclusion bodies=1-10 in one section
++ : Mean number of inclusion bodies=11-20 in one section
+++ : Mean number of inclusion bodies=>20 in one section

separated from the prominent nuclear membrane by a clear halo and the granular inclusion was composed of a basophilic, granular mass, irregulerly dispersed in the nucleus. Only the full type was positive by Feulgen reaction.

Three pigs in the first group, that had intranuclear inclusions in their kidneys, and 6 pigs in the second group also had inclusions in the intestine. Almost all the inclusions appeared in the crypt epithelium (Fig. 2a), dome (Fig. 2b), or adjacent villous epithelium covering Peyer's patches of the jejunum and ileum. The epithelial cells containing inclusions were degenerative and tended to slough off, and inclusions were occasionally seen in the desquamated epithelial cells lying free in the lumen. A few intranuclear inclusions were also found in histiocytes in the lamina propria, or in reticular cells in the lymphoid follicles of Peyer's patches in 2 pigs each from the first and second groups. Only one inclusion each was seen in the crypt epithelium and histiocytes in the lamina propria of the large intestine in 2 pigs of the second group.

Five pigs (3 pigs in the first group and 2 in the second group) had classical type A inclusions (Fig. 3) in swollen nuclei of reticular cells in the lymphoid follicles of the inguinal, gastric, pancreatic, jejunal and colic lymph nodes. The same type of inclusions were also found in epithelial cells of the nasal gland in a pig from the second group.

Other findings ; Perivascular lymphocytic infil-

tration and glial nodules were scattered in the brains of 6 pigs in the first group and 4 in the second group. Only pig no. 1 of the first group developed severe fibrinous meningitis.

Small, round, basophilic organisms identified as cryptosporidia were found in the brush border of the villous and crypt epithelial cells covering Peyer's patches in 12 pigs (4 pigs in the first group and 8 in the second group). They frequently occupied the same region as the inclusion bodies (Fig. 2a).

Severe fibrinous polyserositis and polyarthritis were seen in pig no. 1 of the first group. Scattered focal fibrino-fibrous serositis was observed in visceral organs of 5 pigs of the first group and 2 pigs of the second group. Pig no. 7 in the second group, which received *H. parasuis* lipopolysaccharide, showed generalized circulatory disturbance.

Ten pigs (4 pigs of the first group and 6 of the second group) showed peribronchiolar and perivascular lymphocytic infiltration, lymphoid follicle hyperplasia and alveolar septitis in the lungs.

Immunohistochemical findings: Intranuclear inclusion bodies in the kidneys of the first group and in the small intestines of the first and second groups reacted positively to rabbit anti-type 4 PAV serum (Fig. 4a), but reacted negatively to rabbit anti-type 1, 2 and 3 PAV sera at a dilution 1 : 512 (Figs. 4b-d).

Electron microscopic findings: The epithelial cell nuclei containing full intranuclear inclusions showed chromatin margination. Microvilli in the enterocytes were occasionally lost. The nuclei contained granular electron-dense material intermixed with various numbers of viral particles (Fig. 5a). Virions were oval to hexagonal in shape, measured 82 to 92 nm in diameter and had an electron-dense or electron-lucent core surrounded by a capsid (Fig. 5b). They were scattered throughout the nucleus and occasionally packed in crystalline arrays (Fig. 5c).

DISCUSSION

In the present cases, intranuclear inclusion bodies were most commonly found in the epithelial cells of the jejunum and ileum, as reported in naturally infected pigs (Abid et al., 1984; Buller and Moxley, 1988; Coussement et al., 1981 Fujiwara et al., 1968; Sanford and Hoover, 1983). The three types of intranuclear inclusions described here were similar to those observed in cultured cells infected with canine hepatitis virus (Tajima et al., 1961) and may represent different stages of inclusion development. It has been reported that shortening of villi, cell infiltration in the lamina propria and hyperplasia of lymphoid follicles occur in the small intestine in pigs with diarrhea due to natural and experimental PAV infection (Abid et al., 1984; Coussement et al., 1981; Fujiwara et al., 1968). In the present pigs, however, only slight degeneration and desquamation of intestinal epithelial cells containing intranuclear inclusions were detected. Such changes may be a predisposing factor for secondary infection by other organisms, and also a possible source of transmission of the disease. It has been reported that the dome epithelial complex may function as the portal entry for PAV infection as well as play a role in the intestinal immune mechanism (Buller and Moxley, 1988; Chu et al.1982). There was no relation between frequency of the inclusion bodies and severity of serositis, meningitis and arthritis caused by *H. parasuis* infection.

In 5 pigs of the first group, interstitial nephritis accompanied the formation of intranuclear inclusions in the uriniferous and collecting tubules of the kidneys. A few intranuclear inclusions were also detected in epithelial cells of the large intestine and nasal mucosa as well as reticular cells in the lymph nodes of a small number of pigs. Judging from distribution and frequency of intranuclear inclusions, the intestine seemed to be

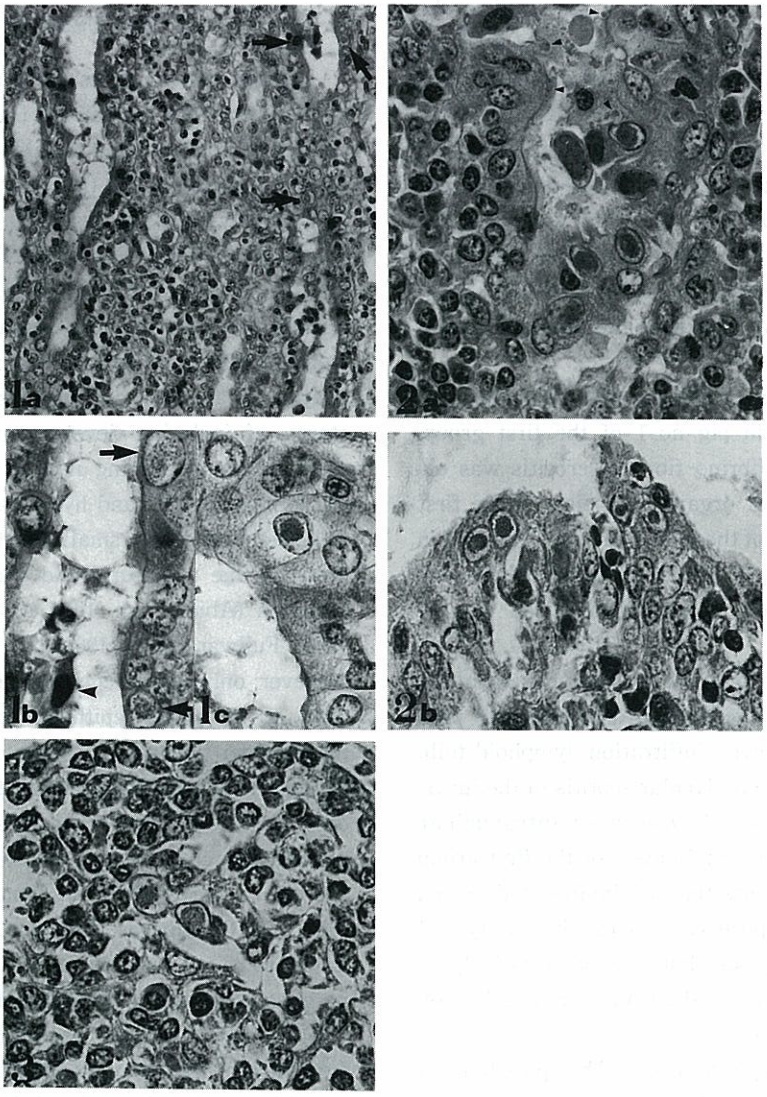

Fig. 1 Medulla of the kidney of pig no. 1 in group 1. (a) Intranuclear in clusions (arrows) are seen in occasional tubular epithelial cells along with desquamated cells in the tubular lumen and lympho-plasmacytic infiltration in intertubular connective tissue. HE, ×200 ; (b) Full type (arrowheads) and granular type (arrows) inclusions. HE, ×600 ; (c) Classical type A inclusion. HE, ×600.

Fig. 2 Intestinal epithelium. HE, ×600. (a) Intranuclear inclusions in the epithelium and desquamated epithelial cells accompanied by cryptosporidia (arrowheads) in the crypt above Peyer's patches in pig no. 9 of group 1 ; (b) Inclusions in the dome of pig no. 2, group 2.

Fig. 3 A lymphoid follicle in the jejunal lymph node of pig no. 3 in group 2. There are 2 inclusions in the swollen nuclei of the reticular cells. HE, ×600.

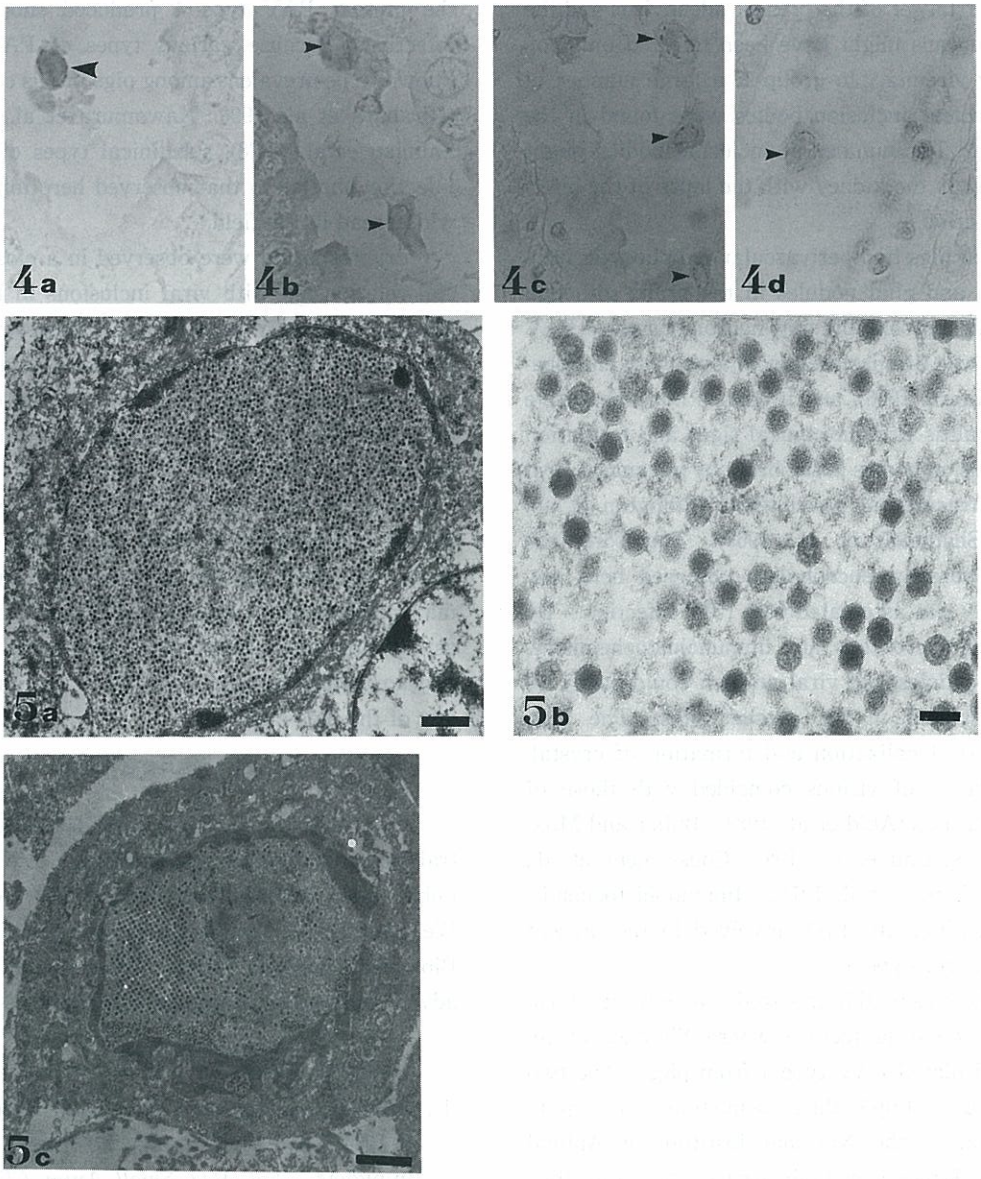

Fig. 4 Inclusion bodies as observed by immunohistochemical staining. ×600. Pig no. 7 in group 1. Inclusions (arrowheads) in desquamated renal tubular epithelial cells with chromatin margination react positively to PAV type 4 antiserum (a), but react negatively to PAV types 1 to 3 antisera (b-d) at a dilution of 1 : 512.

Fig. 5 Electron micrographs of full type intranuclear inclusion bodies. (a) In clusion body in a renal tubular epithelial cell of pig no. 7, group 1, contains numerous viral particles. Bar=1 μm ; (b) higher magnification of (a), virions are oval to hexagonal in shape. Bar=100 nm ; (c) Inclusion in a des quamated epithelial cell in the small intestine of pig no. 2, group 2. Viral particles in crystalline array. Bar=1 μm.

a major target of the present adenovirus and the other organs might have been infected only following viremia. In group 1, a large number of intranuclear inclusion bodies were found in the kidney. The number of inclusion bodies might increase in the kidney with the lapse of the infectious period.

Ten pigs had perivascular lymphocytic infiltration and glial nodules in the brain, although they did not exhibit any neuronal signs. PAV type 4 was first isolated from a pig with encephalitis (Kasza, 1966), and it caused nonsuppurative encephalitis without clinical signs and formation of inclusion bodies in the brain by intranasal, oral and intracerebral inoculations (Edington et al., 1972 ; Shadduck et al., 1967). Therefore, the nonsuppurative encephalitis observed here presumably was associated with PAV infection. By electron microscopy and immunohistochemistry, viral particles and viral antigen could easily be demonstrated in the present pigs. The size, structure, localization and formation of crystalline arrays of virions coincided with those of adenoviruses (Abid et al., 1984 ; Buller and Moxley, 1988 ; Chu et al., 1982 ; Coussement et al., 1981 ; Shimizu et al., 1978). Immunohistochemistry identified the PAV involved in the present cases as serotype 4.

The pigs used in this study came from a farm in the same prefecture where Shimizu et al. (1978) isolated PAV type 4 from pigs. The two pig groups set up in the present study were kept in isolation at the National Institute of Animal Health, Japan after their arrival ; therefore they had no chance to be infected with PAV at the site. However, the two pig groups might have already been infected with PAV on the farm before their departure for the Institute. They did not develop any clinical signs before bacterial inoculation or macroscopical lesions in the kidneys, intestines or lymph nodes. Derbyshire et al. (1975) suggested that only PAV type 4 is a clear-cut pathogen, but the present PAV type 4 produced subclinical infection. Because various types of PAV are known to be prevalent among pigs in this country (Hirahara et al., 1990 ; Kawamura et al., 1972 ; Shimizu et al., 1978), subclinical types of PAV infection similar to that observed here might be widespread in the field.

Cryptosporida were observed in a close special relationship with viral inclusions in the jejunum and ileum in 10 of 12 pigs. However, they could not be related to any morphological lesions and did not cause diarrhea. Therefore, cryptosporidial infection was interpreted as having nothing to do with PAV infection.

Experimental infection with PAV type 4 has caused interstitial pneumonia in the pig (Shadduck et al. 1967). In the present study, pneumonia was observed in 10 pigs, but it was diagnosed as mycoplasmal pneumonia (Dungworth, 1992) and no inclusions were detected in the lungs of any of the pigs examined.

ACKNOWLEDGEMENTS

We would like to thank Dr. H. Hamada, Central Research Lab., Kyoritsushoji Co., Ltd., Tsukuba, Japan, for supplying the anti-PAV sera.
We thank Dr. M. Tajima, the Nippon Institute for Biological Science, Oume, Japan,for valuable advice and review of this manuscript.

REFERENCES

1 . Abid, H. N., Holscher, M. A., and Byerly, C. S. 1984. An outbreak of adeno virus enteritis in piglets. *Vet. Med. Small Anim. Clin.* **79** : 105-109.
2 . Buller, C. R. and Moxley, R. A. 1988. Natural infection of porcine ileal dome M cells with rotavirus and enteric adenovirus. *Vet. Pathol.* **25** : 516-517.
3 . Chu, R. M., Glock, R. D., and Ross, R. F. 1982. Changes in gut-associated lymphoid tissues of the small intestine of eight-week-old pigs

infected with transmissible gastroenteritis virus. *Am. J. Vet. Res.* **43**, 67-76.

4. Coussement, W., Ducatelle, R., Charlier, G., and Hoorens, J. 1981. Adenovirus enteritis in pigs. *Am. J. Vet. Res.* **42**: 1905-1911.
5. Derbyshire, J. B., Clarke, M. C., and Collins, A. P. 1975. Serological and pathogenicity studies with some unclassified porcine adenoviruses. *J. Comp. Pathol.* **35**: 437-443.
6. Dungworth, D. L. 1992. Respiratory mycoplasmosis of swine. pp. 661-663. In: Pathology of Domestic Animals, 4th ed. Vol. 2 (Jubb, K. V. F., Kennedy, P. C., and Palmer, N. eds.), Academic Press Inc., California.
7. Edington, N., Kasza, L., and Christofinis, G. J. 1972. Meningo-encephalitis in gnotobiotic pigs inoculated intranasally and orally with porcine adeno virus 4. *Res. Vet. Sci.* **13**: 289-291.
8. Fujiwara, H., Minamimoto, S., and Namioka, S. 1968. Enteric lesions with intraranuclear inclusion bodies in piglets. *Nat. Inst. Anim. Hlth Quart.* 8: 53-54.
9. Hirahara, T., Yasuhara, H., Matsui, O., Izumida, A., Yoshiki, K., Ota, S., Miyata, Y., Yamanaka, M., Kodama, K., Nakai, M., and Sasaki, N. 1990. Serological survey on porcine adenovirus infection of pigs in Japan. *J. Jpn. Vet. Med. Assoc.* **43**: 779 - 783 (in Japanese with English summary).
10. Kasza, L. 1966. Isolation of an adenovirus from the brain of a pig. *Am. J. Vet. Res.* **27**: 751-758.
11. Kawamura, H., Hatano, Y., and Ito, Y. 1972. An adenovirus isolated from kidney cell culture of apparently normal piglet. Nat. *Inst. Anim. Hlth Quart.* **12**: 173-182.
12. Kirkbride, C. A. and McAdaragh, J. P. 1978. Infectious agents associated with fetal and early neonatal death and abortion in swine. *J. Am. Vet. Med. Assoc.* **172**: 480-483.
13. Narita, M., Imada, T., and Fukusho, A. 1985. Pathologic changes caused by transplacental infection with an adenovirus-like agent in pigs. *Am. J. Vet. Res.* **46**: 1126-1129.
14. Nietfeld, J. C. and Leslie-Steen, P. 1993. Interstitial nephritis in pigs with adenovirus infection. *J. Vet. Diagn. Invest.* **5**: 269-273.
15. Sanford, S. E. and Hoover, D. M. 1983. Enteric adenovirus infection in pigs. *Can. J. Comp. Med.* **47**: 396-400.
16. Schiefer, B., Moffatt, R. E., Greenfield, J., Agar, J. L., and Majka, J. A. 1974. Porcine *Hemophilus parahemolyticus* pneumonia in Saskatchewan 1. Natural occurrence and findings. *Can. J. Comp. Med.* **38**: 99-104.
17. Shadduck, J. A., Koestner, A., and Kasza, L. 1967. The lesions of porcine adenoviral infection in germfree and pathogen-free pigs. *Pathol. Vet.* **4**: 537-552.
18. Shimizu, M., Shimizu, Y., Ito, Y., and Hamada, H. 1978. Porcine adenovirus isolated from pigs with toxoplasmosis. *Nat. Inst. Anim. Hlth Quart.* **18**: 176-177.
19. Tajima, M., Motohashi, T. and Samejima, T. 1961. Electron microscopy of in fectious canine hepatitis virus grown in culture of canine kidney cells. *Amer. J. Vet. Res.* **22**: 236-249.

日本における4型豚アデノウイルス感染症の病理

デヴィ ラテイ アグリプリヨノ*・中川迪夫**
両角徹雄[1)]・吉原 忍[2)]・古岡秀文・松井高峯

帯広畜産大学畜産学部獣医学科
(北海道帯広市稲田町)

摘 要

Haemophilus parasuis 感染実験に供された19頭の豚が組織学的に検査され，うち12頭に4型豚アデノウイルス抗血清に免疫組織化学的に陽性に反応する核内封入体が認められた。封入体は9頭の小腸，5頭の腎臓，5頭のリンパ節，2頭の大腸，1頭の鼻粘膜にそれぞれみられた。間質性腎炎，非化膿性脳炎，及び封入体保有上皮細胞の変性ならびに腎集合管及び腸粘膜上皮からの剝離が豚アデノウイルス感染に関連して認められた。豚アデノウイルス感染豚12頭のうち10頭がクリプトスポリジウムに重複感染していた。実験豚は菌接種以前には無症状であり，剖検時に，腸粘膜，腎臓，及びリンパ節に異常は認められなかった。今回の4型豚アデノウイルス感染症は無症状で，偶発的に認められた疾病であった。

帯大研報 20(1997)：113-122