NOTE

Proliferative Hemorrhagic Enteropathy Detected at an Abattoir in Kagoshima

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Histopathological study was done on 303 pigs with proliferative hemorrhagic enteropathy, which were detected at the abattoir of Kagoshima City from June, 1981 to July, 1983. All the cases had the lesions of proliferative hemorrhagic enteropathy in the ileum, and 134 (44%) cases had the lesions in the cecum, 44 (15%) in the colon, and 5 (0.2%) in the jejunum.—Key words: Proliferative hemorrhagic enteropathy, *Campylobacter sputorum* subspecies *mucosalis*.

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Proliferative hemorrhagic enteropathy (PHE) is a naturally occurring enteric disease of swine characterized by proliferation of immature crypt epithelial cells, loss of the villi, and inflammation of the lamina propria. In this disorder *Campylobacter* sp.-like organisms were consistent within the cytoplasm of proliferative crypt epithelial cells [1–3, 5–8, 10–13].

In Japan, the disease was detected first in Okayama Prefecture in 1978 [2, 10], and since then it has occurred in all over the country [1, 3]. *Campylobacter sputorum* subspecies *mucosalis* (C. m.) was isolated from one pig of the outbreaks [9]. However too little is known about the epizootiological aspects and histopathology of this condition other than the ileum lesions.

The purpose of this paper is to describe the epizootiological features and histopathologic findings of PHE, which was detected by meat inspectors at the abattoir in Kagoshima City, where 12,000 pigs were slaughtered day by day.

From the pigs diagnosed as PHE specimens of the stomach, duodenum, jejunum, ileum, cecum, colon, rectum, mesenteric lymph

Fig. 1. Monthly incidence of PHE

Fig. 2. Incidence of PHE on different farms (A, B, C, D and others) during epizootic period (1982/83)

node, uterus and ovary were collected and fixed in 10% phosphate buffered formalin

Table 1. Morbidity of PHE

Year	Month	Farm A			Farm B			Farm D		
		Affected No.	Slaughtered No.	Morbidity (%)	Affected No.	Slaughtered No.	Morbidity (%)	Affected No.	Slaughtered No.	Morbidity (%)
1982	6	24	1162	2.07	3	489	0.61	0	1340	0.00
	7	13	1070	1.21	3	587	0.51	0	1206	0.00
	8	8	1080	0.74	4	612	0.65	5	1732	0.29
	9	16	1024	1.56	1	341	0.29	4	1210	0.33
	10	7	1215	0.58	0	594	0.00	5	1308	0.38
	11	15	1145	1.31	2	565	0.35	1	1392	0.07
	12	21	1192	1.76	0	441	0.00	0	1292	0.00
1983	1	1	996	0.10	3	473	0.63	1	1041	0.10
	2	3	1119	0.27	0	490	0.00	0	1451	0.00
	3	11	1330	0.83	0	520	0.00	0	1442	0.00
	4	11	754	1.46	2	527	0.38	0	1550	0.00
	5	10	1147	0.87	2	515	0.39	0	1278	0.00
	6	21	1457	1.44	7	511	1.37	0	1745	0.00
	7	13	811	1.60	3	550	0.55	0	1351	0.00
Total		174	15506	1.12	30	7215	0.42	16	19338	0.08

solution. Tissues were embedded in paraffin, cut at 5 μ m, and stained with hematoxylin and eosin for microscopy. Selected sections of the intestine were stained with Warthin-Starry silver method.

PHE was first detected on June 10, 1981 at the abattoir and found sporadically until May, 1982. From June, 1982 to July, 1983, the incidence of PHE was constantly high as shown in Fig. 1, being lowest in January and February and highest in June.

From June, 1982, the pigs with PHE were classified as breeding farms and the results are shown in Fig. 2. Out of 303 pigs affected with PHE, 174 were from farm A. About 80% of the affected pigs were those bred on farms A, B, C, and D. The number of pigs with PHE in all the slaughtered pigs and the rate of PHE detection per month are shown for farms A, B, and D in Table 1. The morbidity of PHE during the observation period was 1.12, 0.42 or 0.08% on farms A, B or D, respectively. The affected 303 cases were 144 females and 159 castrated males, and there was no sexual difference in PHE-occurrence.

Grossly, PHE lesions were principally found in the distal ileum with their length varying from 0.2 to 5 m. The mucosa was thickened and had accentuated mucosal folds. Blood was frequently observed in the ileal

lumen and the cecum and colon contained black and tarry feces. In some cases cheese-like materials were found on the surface of the mucosa or in the lumen of the ileum, and sometimes down into the lumen of the cecum and colon. In such instances the mucosa looked rather thin. The affected ileum was dilated reaching 5 cm in diameter. The serosa of the ileum in some cases was edematous, thickened and purple colored. The muscular layers were occasionally thickened. The mucosa of the jejunum, cecum, colon and rectum was normal.

Histopathologically, PHE-lesions were present in the ileum of all the cases (303 pigs), in the cecum of 134 (44%), in the colon of 44 (15%), and in the jejunum of 5 (0.2%). The lesions consisted of adenomatous proliferation of the crypt epithelium and decrease or disappearance of goblet cells (Fig. 4), which were similar to those reported previously [1-3, 5-8, 10-13]. In 168 cases, the surface of the hyperplastic mucosa revealed necrosis followed by infiltration of neutrophils in the mucosa as well as into the lumen. The cheese-like materials grossly observed in the intestines were composed of indigestible food substance, sloughed epithelium, infiltrating cells, and bacteria (Fig. 4). In those lesions associated with the cheese-like materials, the hyperplastic crypts decreased in number and

Fig. 3. Ileum of a pig, autopsy No.2990. Large and irregular crypts are lined with hyperplastic epithelial cells, and goblet cells lost. Hematoxylin and eosin (HE) staining, X125.

the mucosa was somewhat thin. Granulation tissue was found in the lamina propria and submucosa of the affected ileum. In 140 cases, macrophages or epithelioid cells were proliferated in the lamina propria, in which a few numbers of *Campylobacter* sp.-like organisms were observed (Fig. 5). Proliferation of these cells had no relationship with the severity of the PHE lesions. Intestinal ulcers were seen in some cases.

In the cecum and colon, the principal lesions were similar to those seen in the ileum. However, the hyperplastic crypts were rather few, found isolated among the normal crypts or in clusters, and located in the surface, middle, or deep layer of the mucosa. In some cases, *Treponema*-like organisms were found in the lumen of the colonic crypt (Fig. 6).

In this study, about 80% of pigs with PHE came from 4 breeding farms and the affected

Fig. 4. Ileum of a pig, autopsy No.3144. Cheese-like materials consist of indigestible food, infiltrating cells, desquamated epithelial cells, and bacteria. Hyperplastic mucosa becomes rather thin. HE staining, X32.

pigs were very few as compared to the total numbers slaughtered at the abattoir (Table 1). PHE occurred on restricted farms in Okayama Prefecture [10] and in Australia [7]. However, Lawson *et al.* [4] reported that in a limited examination of sera derived from pigs at slaughter almost all animals had high levels of antibody to C.m. Even in the present outbreaks it was possible that some pigs infected with C.m. were slipped from meat inspection. Further studies are needed to clarify routes or mechanisms through which C.m. is transferred and pathogenesis of PHE.

Surface necrosis of the hyperplastic mucosa was seen in 168 cases. This might be caused by secondary infection due to disappearance of goblet cells as seen in reginal ileitis or necrotizing enteritis which were considered to be one of different lesions of

Fig. 5. Ileum of a pig, autopsy No. 3189. Macrophages or epithelioid cells are proliferate in the lamina propria. Cellular infiltration is seen in the lumen of the hyperplastic crypt. HE staining, $\times 400$.

Fig. 6. Colon of a pig, autopsy No. 3123. *Campylobacter* sp.-like organisms are seen in the tip of hyperplastic epithelium and *Treponema*-like organisms in the lumen of the crypt. Warthin-Starry staining, $\times 1,250$.

PHE [11]. Macrophages or epithelioid cells proliferated in the lamina propria of 140 cases (46%), but these cells only occasionally phagocytized *Campylobacter* sp.-like organisms. The relationship between these cells in the lamina propria and cryptic hyperplasia was not clarified.

Treponema-like organisms found in the lumen of the colonic crypt were morphologically indistinguishable from *Treponema hyodysenteriae*. It might produce microenvironment in which *C. m.* could penetrate the epithelium of the cecum or colon.

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要 約

鹿児島県の一屠畜場において観察された豚の増殖性出血性腸炎について（短報）：久保正法・大宅辰夫・渡瀬 弘（家畜衛生試験場九州支場）——鹿児島市の1屠畜場において，1981年6月から1983年7月までに摘発された豚の増殖性出血性腸炎303例について病理組織的に観察した。303例中約80%は4養豚場由来で，1981年6月から1982年5月までは散発的であったが，1982年6月からは毎月摘発された。病変のみられた部位は，回腸303例（100%），盲腸134例（44%），結腸44例（15%），空腸5例（0.2%）であった。組織学的には陰窩の過形成が主で，168例には粘膜の壊死があり，140例では類上皮細胞の増殖がみられた。