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An Outbreak of Swine Dysentery in Korea: Clinical Aspects and Pathology

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Introduction

Swine dysentery has been described as an acute or chronic disease of young pigs characterized by bloody mucoid diarrhea and severe inflammation of the large intestine. Since the first case report was recorded in 1921[21], many reports on the disease have been succeeded in the veterinary literature of various countries.[2,3,5~7,9,12~16,18,20] However any report concerning occurrence of the disease was not yet recorded in Korea.

Until recently the etiologic factor of swine dysentery was generally accepted to be *Vibrio coli*.[4,17] Not only this organism has been isolated from affected pigs but also found commonly in normal pigs.[1,3,7~10] In several investigations pure culture of *Vibrio coli* did not produced swine dysentery.[1,7,10] In recent time some investigators confirmed involvement of large spirochetes, *Treponema hyodysenteriae* as a causative agent in association with swine dysentery.[10,11,19] Hughes et al.[11] induced severe disease characteristic of swine dysentery by oral inoculation of pure culture of *Treponema hyodysenteriae* into pigs.

Authors investigated an outbreak of a highly infective diarrhea of pigs in a piggery at Gimhae county, the southern district of Korea, confirming involvement of large spirochetes and Vibric-like organism with the colonic lesion.

Materials and Methods

Swine Observed: In April and May, 1975 the owner of a large piggery feeding a total of 1,200 Yorkshire or Landrace swine at Gimhae county consulted authors regarding a persistant muocus diarrhea. Of them 350 pigs showed of mucohemorrhagic diarrhea, and so clinical and epidemiological observation for patients was followed.

Necropsy and Histologic Examination: Five pigs with typical symptoms from 10 to 15 weeks of age were necropsied to get diagnostic aids. Specimens of tissues from the alimentary tract or other organs were fixed in 10% buffered formalin, prepared into paraffin sections and stained with hematoxylin and eosin for microscopic observation. Selected sections of the large intestine were stained with the Gram's or Warthin-Starry staining for spirochetes.

Results

Clinical Signs and Epidemiology: Most of the cases coursed as the acute form. In the acute form the pigs were usually submitted with signs of diarrhea, depression, inappetence, tucked-in flanks, dehydration and loss of weight. The diarrhea developed early in the course of the disease and feces varied in color depending on whether or not they contained blood. At onset of diarrhea the feces were usually yellowish or greyish brown in color and soft to watery in consistency. Shreds of white and inspissated mucous material as sloughed necrotic epithelium were mixed with liquid fecal material. With progression of the diarrhea the feces became muddy grey to dark brown or dark red in color.

The hind legs and tail were soiled with fecal discharges probably due to tenesmus or to anal

irritation. Affected pigs were usually diminished appetite, but frequently animals continued to eat rather well inspite of the severe diarrhea. Pigs became gaunt, emaciated, dehydrated and weak. Finally some animals became recumbent too weak to move and eventual death might follow.

In chronic infection which was not so frequent they remained stunted, dehydrated and showed watery diarrhea with the characteristic rice water appearance. They were accompanied by progressive emaciation and dehydration inspite of continued food intake. Rising in body temparature was not constant and it was generally within normal range, but variation from a low of 36°C in terminated cases to high of 40.8°C were noticed.

The course of the disease varied from 3 or 5 days to 2 or 3 weeks or longer in the chronic cases. The disease spread rapidly through a herd and morbidity reached around 70% in young pigs. The age incidence ranged from 3 weeks to 28 weeks but the majority of cases were observed in pigs between 8 and 14 weeks of age and was uncommon in suckling pigs and in the breeding stocks(Fig.1).

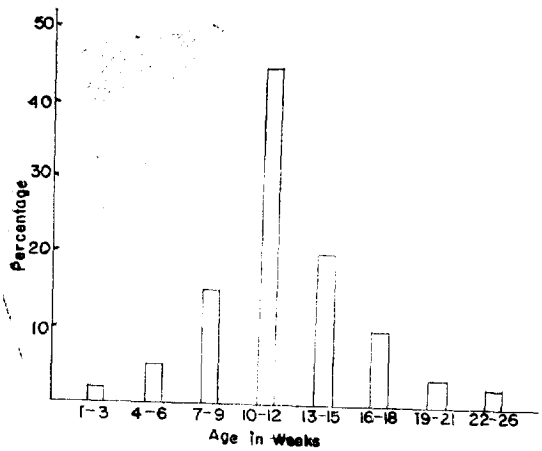


Fig. 9. Age distribution of 350 swine affected with dysentery.

Gross Lesions: All of necropsied pigs were acute cases with duration from one to two weeks in clinical course. At post-mortem examination the carcase showed signs of emaciation and dehydration. The skin was dry and wrinkled and showed a bluish red discoloration at ventral part of the body and extremities. The superficial lymph nodes appeared almost normal in size, color and consistency.

Some of necropsied pigs showed prominent lesion of the stomach, viz the thickend fundic mucosa was hyperemic, bright red or dark red in color and with sharp demarkation. In these cases the mucosa was usually covered with an excess of tenacious mucous exudate. The small intestine did not showed distinct changes except for a mild congestion or increased mucous exudate. The mesenteric lymph nodes of the some cases were slightly swelled and hyperemic.

The obvious inflammatory lesions were limited to the large intestine. Upon opening the abdominal cavity the serosal surface was congestive covered with fibrin and gave dull and rough appearance. The mucous membrane was irregularly reddened and covered by a varying amount of mucus or was more uniformly and severely reddened. The intestinal wall was swollen and edematous.

Three of the necropsied pigs had more advanced fibrinonecrotic membrane formation over the cecal and colonic mucosa. It was characterized by varying size of brown or gray necrotic membrane with underlying foci of reddened or hemorrhagic lesion. The necrotic patches coalesced to form a diffuse necrotic membrane affecting a large portion of the cecum, colon and even the rectum. Extent of necrosis of the mucosa was always superficial.

Throughout of thickened mucosa of the colon or cecum there were scattered numerous tubercles. They were one to 3mm in diameter, embeded in intestinal mucosa and convexed slightly over the mucosal surface. The larger one contained the core of necrotic debris at the cut surface.

The fecal material was dirty reddish brown in color and fluid in consistency and mixed with conspiquous mucous exudate or sloughed necrotic epithelium. Edema or mucoid degeneration were promient in the mesentery of the spiral colon. The associated colonic lymph nodes were enlarged and bright red

in sinusoidal areas. The liver, spleen and kidney did not show other constant or noteworthy changes.

Histopathology of the Lesion: The principal histologic changes were found in the wall of the large intestine. The histologic changes of the colonic mucosa consisted of acute or chronic catarrhal or severe coagulative necrosis of the superficial mucosa. In acute catarrhal inflammation the different portion of the affected intestine did not show same extent of reaction. The goblet cells of intestinal glands were more prominant and hyperplastic to get deeper crypt than that in the normal state (Fig. 1). The lumen of the crypts was enlarged with mucus, fibrin or cellular debris, occasionally showing distended condition.

The embeded minute tubercles in the colonic mucosa recognized macroscopically were conformed microscopically as the greatly enlarged crypts. The crypts were prominently distended by accumulation of mucous exudate and cellular debris in the lumen of the crypts while the neck of the gland was constricted. The covering epithelium of the distended crypts showed dysplastic changes manifested by converting from columnar cells to cuboidal or squamous cells with pyknotic nuclei (Fig. 4).

The epithelial desquamation of the intestinal mucosa was either focal or extensive. But the extent of epithelial necrosis was superficial; usually there was destruction of the upper one-third of the lining epithelium and of the lamina propria. The eroded surface of the mucosa was covered with exudate containing mucus, fibrin, erythrocytes and cellular debris. The lamina propria of mucosa and less frequently the submucosa showed definite congestion and were infiltrated with leukocytes predominantly of the lymphocytes and macrophages. The colonic lymph nodes, were edematous and hyperplastic appearing focal areas of hemorrhage and necrosis of lymphoid tissue.

By Gram stain of tissue sections numerous Vibrio-like bacteria were found in the degenerative colonic mucosa (Fig. 8). Warthy-Starry technique applied in the tissue specimens of the colon demonstrated involvement of spirochetes with the lesion (Fig. 6). The serpentine large spirochetes appeared in patchy foci in the surface exudate and necrotic debris. randomly. Numerous Vibrio-like organisms were mixed in the foci of spirochetes but generally the latter was distributed frequently in deeper position of the mucosa even at the level of the upper half of the mucosa than the former. Spirochetes were seen in close association with the apical surface of colonic crypt epithelium (Fig. 5). The distended crypts that was the cyst like tubercles contained masses of numerous spirochetes mixed in the necrotic debris (Fig. 7).

Discussion

The diagnosis of swine dysentery was based on the clinical, epidemioligical, pathological findings. From these points of view, the clinical and pathological features of the disease outlined here resamble closely those described by Doyle[5], Loveday[12], Lussier[13], Walz[20] or Davis[2] except the embeded tubercles in the wall of the colon.

The authors' report was the first presentation on the outbreak of swine dysentery in Korea. Diarrhea in these cases was characterized by presence of mucus and at times by the presence of sloughed necrotic materials mixed with fluid discharge or hemorrhagic exudates. The prominent lesions were limited to the large intestine and associated lymph nodes. The typical type of inflammation was of acute catarrhal or necrotic type accompanied with mucohemorrhagic exudates. Though extent of necrosis of the mucosa was extensive, the necrosis was appeared superficially and this type of inflammation was differentiated from that found in advanced cases of hog cholera and salmonella enteritis.

The minute tubercles embeded in colonic wall were characteristic lesion in these cases of swine dysentery. Regarding on the histologic structure of the tubercle, it is inferable that formation of the lesion is atributed to dilation of colonic crypts. Dilation of the crypts was occurred by accumulation of mucus, fibrin or cellular debris in the crypts showing dysplasia of covering epithelium of the tubercle

while the neck of crypt was constricted.

Pathogenesis of the tubercle formation would be based upon dilation of the crypt with epithelial dysplasia.

Bacteriological staining of the lesion demonstrated involvement of numerous spirochetes and Vibrio-like organims. The bacteriological findings on these cases was coincided with that of Hughes'[11] experimental dysentery of swine which was induced by oral inoculation of *Treponema hyodysenteriae*. Spirochetes appeared most numerously wherever coagulative necrosis was extensive. Invasion of spirochetes into the damaged mucosa was appeared at the level of the upper half of the colonic mucosa. Involving spirochetes was more revealed and more closely associated with crypt epithelium than involvement of Vibrio-like organisms. There were also masses of numerous spirochetes in the tubercle embeded in the colonic wall mixed in necrotic debris. Therefore the spirochetes would play role as primary etiologic agent to produce severe acute coagulative necrosis of the colon or the tubercle lesion. The primary etiologic and opportunistic organisms such as *Vibrio coli* probably induce the inflammatory respons that was seen as a zone of superficial necrosis of the mucosa and underlying tissue respons.

Conclusion

Authors investigated an outbreak of a highly infective disease with persistant diarrhea in a piggery at Gimhae district. Severe mucohemorrhagic diarrhea was characteristic signs and the lesion was chiefly a catarrhal or necrotic inflammation of the large intestine. Necrosis of the mucosa appeared superficially. The characteristic minute tucercles were observed extensively in the colonic mucosa. Formation of the tubercle would be atributed to dilation of colonic crypts with epithelial dysplasia due to accumlation of exudate or cellular debris in the crypts.

In the colonic mucosa numerous large spirochetes and Vibrio-like organisms were seen in closer association of the former than that of the latter with the lesion. On these findings of the disease it was diagnosed as swine dysentery presenting the first case report in Korea.

Legends for Figures

Fig. 1. An acute catarrhal inflammation of the colonic mucosa showing hyperplasia of goblet cells to make distinct and deeper cryps. Desquamation of surface epithelium is shown (arrow). Hematoxylin and eosin (HE) stain, ×100.

Fig. 2. An acute necrotic inflammation of the colonic mucosa. Notice fibrinonecrotic membrane of superficial mucosa (arrows). HE stain, ×100.

Fig. 3. A cyst like tubercle embeded in colonic mucosa. The tubercle is formed by distension of a colonic crypt in which massive mucus, fibrin and cellular debris (D) are accumulated while neck of the crypt is constricted (arrow). HE stain, ×23.

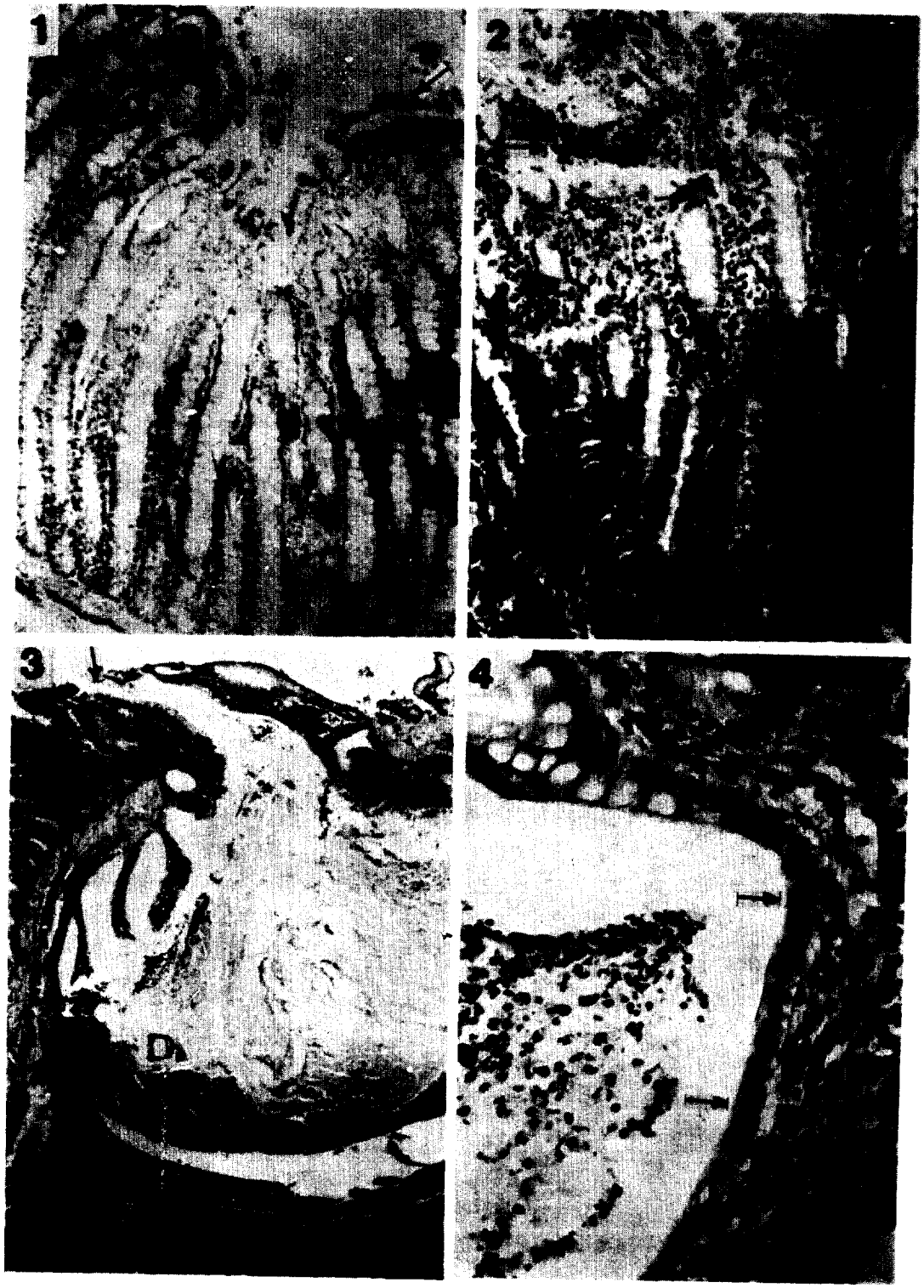
Fig. 4. High-power magnification of Fig. 3. shows dysplastic changes of covering epithelium manifested by converting from columnar goblet cells to cuboidal cells (arrow) with pyknotic nuclei. Necrotic cellular debris are seen in distended lumen of the crypt. HE stain, ×400.

Fig. 5. Degenerative neck of colonic crypts showing close association of spirochetes (arrows) with the apical surface of crypts. Warthin-Starry stain, ×100.

Fig. 6. Numerous serpentine spirochetes (arrows) in the degenerative superficial colonic mucosa. Warthin-Starry stain, ×1,000.

Fig. 7. Masses of numerous spirochetes (S) and cellular debris (D) which are contained in the tubercle of Fig. 3. showing embeshed structure. Warthin-Starry stain, ×400.

Fig. 8. A pathchy focus of numerous Vibrio-like organisms (arrows) is shown in surface exudates of the colonic mucosa. Gram stain, ×1,000.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돼지 赤痢의 發生報告 : 臨床 및 病生學的 所見

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국문초록

慶尙南道 金海郡 S養豚場에서 1975년 4월 하순부터 5월 하순 사이에 飼育豚 1,200두중 약 350두에서 出血性下痢를 主症으로 하는 傳染性이 강한 消化器疾患이 발생하였다. 患豚은 7週齡에서부터 14週齡까지에서 가장 많이 발생하고 急性 또는 亞急性으로 경과하였으며 다량의 黃白色, 水樣下痢를

발하다가 黃褐色, 泡沫性下痢 또는 暗赤褐色 粘液性下痢를 나타내었다. 肉眼的 病變은 大腸에 한하였으며 카타아르性, 出血性 내지 壞死性腸炎을 示顯하였으며 壞死는 廣汎하나 表在性이고 어떤 예에서는 結腸粘膜 속에 大小의 微細結節이 密發한 것이 특징이었다.

組織學的檢査에서는 大腸粘膜에 현저한 杯細胞增殖, 鬱血 및 圓形細胞浸潤과 더불어 表在性壞死가 관찰되었고 上記 結節은 腸腺 속에 粘液, 纖維素 및 崩壞細胞가 축적되어 腺窩가 확장되고 被覆上皮의 異形成(dysplasia)을 일으키고 있음이 판명되었다. 病變部의 細菌染色에 의하여 大型 Spirocheta와 Vibrio 孤菌이 無數히 발견되었고 Spirocheta가 病變形成에 더 밀접하게 관련되어 있음이 밝혀졌다.