Korean Journal
of Veterinary Service

Kor J Vet Serv(2001) 24(4) : 375~377

Rectal stricture in a finishing swine : Case

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(Received 17 September 2001, accepted in revised from 7 October 2001)

Abstract

Rectal stricture occurred in 2 finishing pigs submitted for necropsy from Moguchon, the meat processing plant, Chonbuk. Grossly, the wall of the rectum was harden and thickened by fibrous tissue. Anterior to the stricture, the descending colon was dilated up to 30㎝ in diameter, filled with gas and pasty green fluidal feces. Histologically, the epithelia of rectal mucosa were necrotized. The mucosa and submucosa of rectum were infiltrated by macrophages, eosinophils and lymphocytes. This infiltration was the most extensive in the deeper layer of submucosa and intensive fibrosis was observed in deeper submucosa layer. This case is report for rectal stricture of finishing pig.

Key words : Rectal stricture, Finishing pig, Fibrosis

Introduction

Rectal stricture is characterized by a narrowing of the rectum near the anus, resulting in an obstruction to normal defecation[1-6]. Rectal stricture is a acquired annular fibrous constriction of the rectum 2.0 ~5.0㎝ anterior to the anus. It results in the clinical and pathologic features of chronic constipation, and it is a frequent disease reported to occur in young swine[2,3,7,8].

Rectal strictures were first reported in Illinois in 1967[1], and have been reported from most of the swine raising areas of the United States[1,2,7,9], the Netherlands[11], and the England[3]. But any report on rectal stricture in swine was not found in Chonbuk area.

Clinical features are progressive emaciation, depression, abdominal distension and marked abdominal tympany, which may be intermittently relieved by the passage of watery, dark, foul smelling feces[2]. Also, the appetite and weight are decreased[3]. Rectal stricture is seems to be unrelated to sex, breed, management conditions or concurrent disease[2,7].

This case is the gross and microscopic observation of the rectal stricture of swine.

Case

Abdominal distension was observed in

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both finishing pig from a slaughterhouse, Moguchon, the meat processing plant in Chonbuk when incision was made into the abdominal skin. Each gastrointestinal track was collected and submitted to Diagnostic Laboratory, Chonbuk National University.

Grossly, lumen was narrowed in one part of rectum. The wall of the rectum was harden and thickened by fibrous tissue. Anterior to the stricture, the descending colon was dilated up to 30㎝ in diameter, filled with gas and pasty green fluidal feces. The lumen of the spiral colon is increased up to 3 or 4 times normal diameter. The mucosal surface anterior to the stricture was ulcerated and covered by a green membrane (Fig 1).

Samples of colon and rectum were collected into neutralized 10% buffered formalin and processed routinely and stained with hematoxylin-eosin(H-E). Microscopically, there was a necrosis in surface of mucosa in rectum(Fig 2). Accidentally, *Balantidiuim* spp was observed in the necrotized mucosal layer. The mucosa and submucosa of rectum were infiltrated by macrophages, eosinophils and lymphocytes. This infiltration was the most extensive in the submucosa and intensive fibrosis was extended to deeper submucosa layer(Fig 3). The vessels in the submucosa were intensely congested.

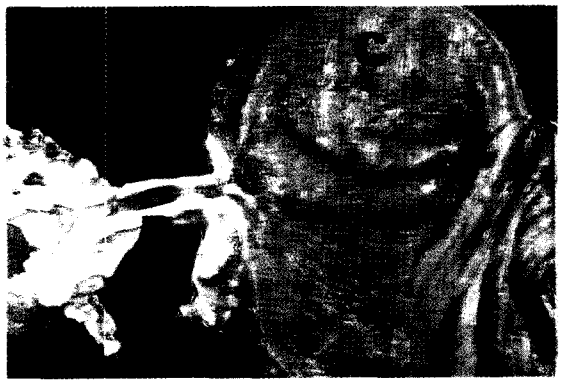

Fig 1. Gross view of a specimen from a pig with rectal stricture (R) and spherically dilated descending colon (C).

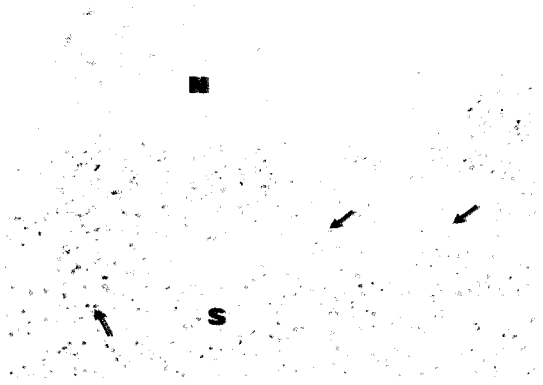

Fig 2. A necrosis area(N) and submucosa(S) in rectum, which were infiltrated with macrophages, eosinophils(arrows) and lymphocytes, H-E, ×200.

Fig 3. Intensive fibrosis was extended to deeper submucosa layer. H-E, ×200.

Discussion

Rectal stricture is a well recognized syndrome but the main causes has not been clear[1]. Also, the sources of exposure to the disease have not been determined. It has been reported that rectal stricture is induced by various causes. Spellman[10] reported that rectal stricture may be induced by chronic

enteritis, peritonitis around the pelvic inlet, polyserositis, occlusion of the distal branch of the caudal mesenteric artery and *Salmonella* as a cause of arterial thrombosis. Also Wilcock and Olander[4,6] reported that *S. typhimurium* as a causative agent was isolated from swine with rectal stricture, and rectal stricture was experimentally induced oral *Salmonella. typhimurium* infection in pigs. They also pointed out that not only other serotypes of *Salmonella* but also other members of the *Enterobacteriacae* could induce rectal stricture But microbiological test for pathogen was not examined in our case.

Saunders et al. reported that rectal stricture was considered to be a sequel to a genetic predisposition toward rectal prolapse[3]. It has been cited that mycotic enteritis[1], chlamydial proctitis[2] and insult from toxic compounds in the feed[1,7] might be capable of induce rectal stricture. One researcher[3] demonstrated that rectal stricture was sequel to resolve rectal prolapse by surgery to cut off prolapsed rectum. One researcher[2] emphasized that rectal stricture in pig should be differentiated from muscular hypertrophy of the ilium, regional and proliferative ileitis, diverticulitis, persistent Meckel's diverticulum, prolapsed rectum, atresia of the rectum or anus, and intestinal emphysema.

Generally, in severe cases or as a sequel to rectal stricture, there are adhesions or anastomosis of large intestine or small intestine to a descending colon[2,11]. In this case, however, these signs was not observed.

The occurrence of rectal stricture in swine is probably endemic or individual pattern and cause the economical problem if the fre quency of occurrence increases. Even the surgical treatment was described, the detection of this stricture is still difficult and the surgery is unclear. Therefore, the further investigation should be done on the slaughterhouse of pig. This is the case of rectal stricture in pig.

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