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Surgical Correction of Chronic Hypertropic Pyloric Gastropathy in a Dog

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Abstract: A ten-year-old, female Yorkshire terrier dog with intermittent vomiting, weight loss, polydipsia and depression was referred to the Veterinary Medical Teaching Hospital of Konkuk University. On the radiological survey, gas and fluid-filled gastric distention was detected. No contrast medium entranced to the small bowel, in 60 minutes after contrast medium administration. And marked fluid accumulation in gastric body and thickening of the pyloric mucosa were found with ultrasonograph. So gastric obstruction by reason of pyloric stenosis was strongly suspected. At the surgery, hypertrophied mucosa was resected adequately, and Heineke-Mikulicz pyloroplasty was applied. Microscopically the symptom was characterized by gastric mucosa hyperplasia and mild diffuse lymphoplasmocytic gastritis. So this condition was diagnosed as chronic hypertrophic pyloric gastropathy. Intermittent vomiting and mild depression were shown for 9 days after the operation. After that, the patient was recovered gradually. One month later, the patient had normal appetite and activity without complications.

Key words : chronic hypertrophic pyloric gastropathy, Heineke-Mikulicz pyloroplasty, dog.

Introduction

Gastric retention and outflow obstruction can be happened by various mechanisms. Acquired mucosal and muscular pyloric hypertrophy and hyperplasia are one of the reasons. And these are termed chronic hypertropic pyloric gastropathy (CHPG) or acquired antral pyloric hypertrophy (AAPH)[10]. Walter et al[15] used the term chronic hypertrophic pyloric gastropathy for series of 6 cases.

CHPG commonly occurs in middle-aged small-breed dogs and causes chronic intermittent vomiting and weight loss[6]. But these syndromes have not been clearly identified. Ultrasonographic and radiographic findings are support to a tentative diagnosis of CHPG. Gas and fluid-filled gastric distention, pyloric intraluminal filling defects, and a "break" or "tit" sign are detected on the VD and DV radiograph. And ultrasonographic findings showed thickened mucosa or muscular layers of the pylorus and no pyloric movement.

This report introduces the dog, which was suffered from affected by CHPG, the method of diagnosis, and the successful surgical treatment.

Case

History and physical findings

A ten years old, female Yorkshire terrier dog was referred to the Veterinary Medical Teaching Hospital of College of Veterinary Medicine of Konkuk University. The chief complaints were chronic intermittent vomiting and chronic weight loss. Although the medical treatment was performed for two months at local animal hospital, the symptoms had not been disappeared. Vomiting was even worsened and the patient couldn't eat anything.

In the physical examination, the dog had pain in the cranial abdominal palpation. Body temperature was 38.3°C and respiratory rate was 30 breaths/min. Heart rate was 159 bpm at the resting state. The patient showed lethargy, depression, and low appetite.

Hematologic and serological findings

In CBC, there were no specific abnormalities except mild increasing PCV (52%). Electrolyte analysis was abnormal (K; 3.3 mmol/L, Na; 133 mmol/L, Cl; 81 mmol/L). In serum biochemical abnormalities included increasing albumin concentration (4.1 g/dl), it may demonstrated dehydration.

Radiological findings

On radiography, gas and fluid-filled gastric distention was detected (Fig 1). With contrast radiography, pyloric intraluminal filling defects and a "break" sign was shown (Fig 2). Delayed gastric emptying was suspected as no contrast medium entranced to the small bowl, in 60 minutes after contrast medium administration of. Although peristaltic waves were seen pushing the contrast agent, barium up to the pyloric canal, contrast media couldn't passes into the duodenum with fluoroscopy.

Ultrasonographic findings

Two-dimentional ultrasonograph showed marked fluid-filled gastric distention, and uniform thickening of the mucosa layers of the pylorus. There was pyloric movement but no ingesta could pass through pyloric canal (Fig 3).

Surgical treatment and findings

Because the patient couldn't eat anything and gastric emptying was not detected any more, no more examination was

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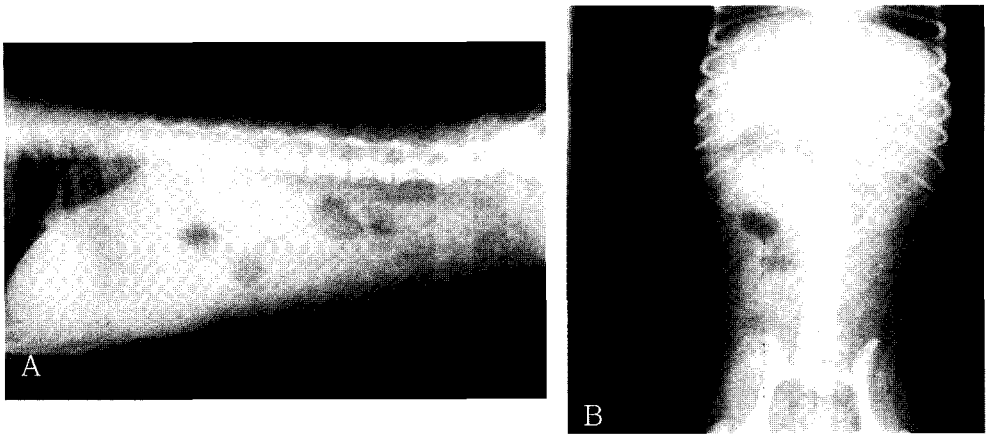

Fig 1. Survey radiography. Lateral (A) and ventrodorsal (B) view of a dog with chronic hypertropic pyloric gastropathy. Gas and fluid-filled gastric distention was showed.

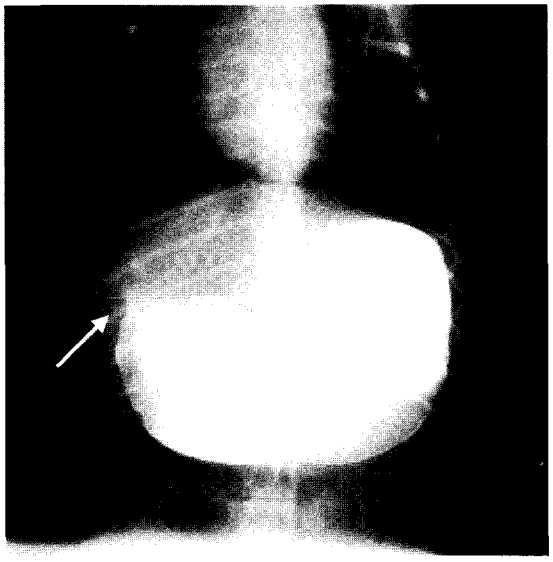

Fig 2. Contrast radiography. 60 minutes after administration contrast medium. Pyloric intraluminal filling defects, and a "break" sign (arrow) was shown. Delayed gastric emptying was suspected as no contrast medium entranced to the small bowel.

performed. And emergency operation was needed.

Dehydration, electrolyte and acid base abnormalities were corrected before surgery. H2-receptor blocker (Ranitidine HCl, Sam Chun Dang Pharm. Co. Ltd., 2 mg/kg, i.v.) was treated.

Prophylactic antibiotics (Cephradine®, Korea Schnell Pharma., 20 mg/kg, i.v.) and analgesic (Butorphan®, Myungmoon pharm., 0.4 mg/kg, i.v.) were administrated before surgery. After anticholinergic (Atropine Sulfate, Kwang Myung pharm. Co. Ltd., 0.02 mg/kg, s.c.) was injected, anesthesia was inducted with thiopental sodium (Pentotal Sodium®, Choong wae Pharm. Co., 15 mg/kg, i.v.). Anesthesia was maintained with isoflurane (Rodia Isoflurane Solution®, Rhodia Organique Fine Ltd., 0.5~2.5%) and mechanical ventilation was performed.

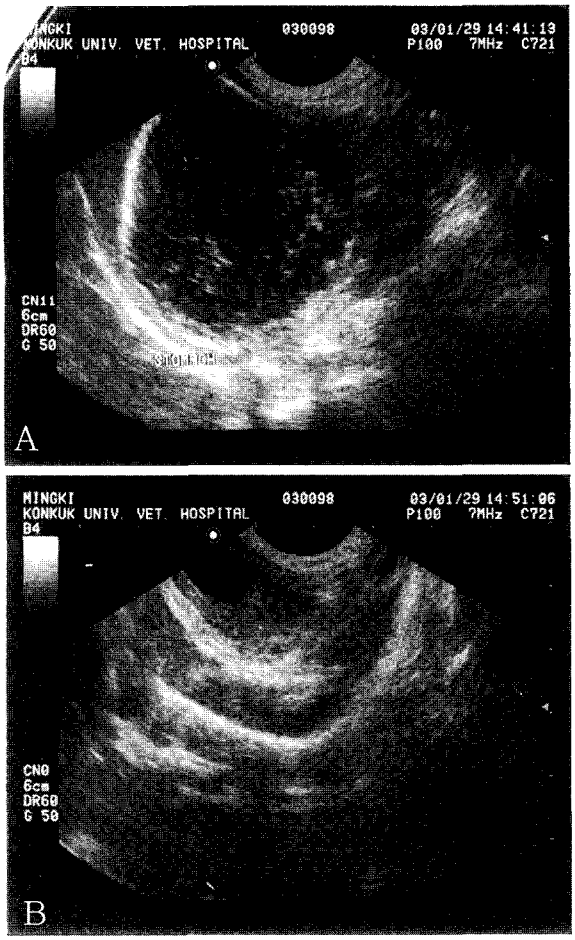

Fig 3. Ultrasonograms of stomach before operation. (A) Gastric distention with fluid was showed. (B) Pyloric mucosal hypertrophy was showed.

The dog was positioned in dorsal recumbency, for a ventral midline coeliotomy from xiphoid process to the umbilicus. For adequate exposure of the gastrointestinal tract, abdominal

wall was retracted. After investigation of the stomach, the pyloric region of the stomach was identified and palpated.

Heineke-Mikulicz pyloroplasty was adjusted for improving gastric outflow. For biopsy of thickened mucosa with hyperplastic and cystic changes in submucous glands, pyloromyotomy was performed. After sutures of gastric mucosa, the stomach was carefully closed. Abdominal cavity was closed routinely.

Postoperative care and evaluation, histopathological findings

After the operation, butorphanol (Butorphan®, Myungmoon pharm., 0.2 mg/kg, i.v.) was given for analgesia. Hartman dextrose was administered intravenously for 3 days. The patient was given small amount of water in two days after the surgery but didn't vomit. Then the patient was offered of liquid diet. Gastric emptying was evaluated with fluoroscopy. After confirming normal gastric emptying, metoclopromide was given intravenously. The amount of diet was increased gradually. Amoxicillin, cimetidine, and metoclopromide were administered orally for 7 days after eating. Intermittent vomiting and mild depression was shown for 9 days after the operation.

For one month, the symptoms had been decreased gradually, and appetite was improved. One month later, the patient was successfully treated by surgical correction of the gastric outlet obstruction.

Histopathologically, biopsy specimen was confirmed as gastric mucosa hyperplasia, and mild diffuse lympoplasmocytic gastritis. So this was diagnosed as type II chronic hypertropic pyloric gastropathy consequently.

Discussion

Benign hypertrophy of the stomach has been reported. But little is known about the specific causes and pathophysiologic changes of the disease processes. Experimental and clinical evidences suggest that the cause of CHPG is immune-mediated inflammation, hormonal influence, and the maintenance of benign gastric hypertrophy[2,3,9-11,13,14]. CHPG is classified to three types with histological descriptions. Type I is pyloric muscle hypertrophy, type II is pyloric mucosa hyperplasia with glandular cystic dilation and type III is pyloric muscle hypertrophy and mucosa hyperplasia[5]. On histological examination, this case was diagnosed as Type II CHPG. The typical dog suffering from chronic hypertrophic pyloric gastropathy is a small, middle-aged dog that has been vomiting intermittently for months to years[15]. It was considered that neuroendocrine events associated with excitable or vicious behaviors contribute to the pathogenesis of the gastric lesions[15]. In this study, the patient was the small, excitable, old-aged dog that had been vomiting intermittently for a month.

CHPG with gastric outlet obstruction is treated and diagnosed by pyloroplasty and billroth I technique[5]. The choice of corrective methods is made during surgery, base on the visual appearance and physical characteristics of the obstructive lesion. Heineke-Mikulicz pyloroplasty have the advantage of easily peforming operation. But mucosal exposure is limited and it does not allow adequate resection of hypertrophied mucosa. So, If not muscular but mucosal hypertrophy, a Y-U pyloroplasty is usually preferred. Billroth I procedure has risk of dehiscence and leakage, so this method is not usually selected in benign pyloric outlet obstruction[5]. In this case, with inspect of stomach, we thought that pyloric outlet was not severally obstructed. So Heineke-Mikulicz pyloroplasty was selected. During pyloroplasty, full-thickness resection of hypertrophied mucosa was performed. Although intermittent vomiting was shown for one month, this patient was recovered gradually. And no complication was shown.

Conclusion

A ten years old, female Yorkshire terrier dog which had gastric obstruction was diagnosed as type II CHPG. For removing the obstruction and reestablishment of normal gastric emptying, Heineke-Mikulicz pyloroplasty was selected with full-thickness resection of hypertrophied mucosa. After surgical treatment the patient was recovered gradually for one month.

Reference

1. Bellenger CR, Maddison JE, Macpherson GC, Ilkiw JE. Chronic hypertrophic pyloric gastropathy in 14 dogs. Aust Vet J 1990; 67: 317-320.
2. Chusid EL, Hirscj RL, Colcher H. Spectrum of hypertrophic gastropathy. Arch Int Med 1964; 114: 621-628.
3. Dodge JA, Karim AA. Induction of pyloric hypertrophy by pentagastrin. Gut 1976; 17: 280-284.
4. Douglas SW. Lesions involving the pyloric region of the canine stomach. J Am Vet Radiol Soc 1968; 9: 89-93.
5. Fossum TW. Surgery of the stomach, Benign gastric outflow obstruction, In: Small Animal Surgery, 2nd ed. St. Louis: Mosby. 2002: 337-352, 360-363.
6. Leib MS, Saunder GK, Moon ML, Mann MA, Martin RA, Matz ME, Nix B, Smith MM, Waldron DR. Endoscopic diagnosis of chronic hypertrophic pyloric gastropathy in dogs. J Vet Intern Med 1993; 7: 335-341.
7. Rhodes WH, Brodey RS. The differential diagnosis of pyloric obstructions in the dog. J Am Vet Radiol Soc 1965; 6: 65-73.
8. Root CR, Morgan JP. Contrast radiography of the upper gastrointestinal tract in the dog. J Small Anim Pract 1969; 10: 279-285.
9. Schindler R. On hypertrophic glandular gastritis, hypertrophic gastropathy, and parietal cell mass. Gastroenterology 1963; 45: 77-85.
10. Scotti TM. Simian gastropathy with submucosa glands and cysts. Arch Pathol 1975; 96: 403-408.
11. Siang Go T, Morse WH. Hypertrophic pyloric stenosis in adults. Am J Gastroenterol 1973; 60: 400-465.
12. Rasmussen L. Stomach, In: Textbook of Small Animal Surgery, 3rd ed. Philadephia: Saunders. 2002: 592-640.

13. Guilford WG, Strombeck DR. Chronic gastric diseases, In: Strombecks small animal gastroenterology, 3rd ed. Philadephia: Saunders. 1996: 275-302.
14. Sussaman HM, Weingarten B, Mossberg SM. Localized gastric mucosal hypertrophy simulating tumor. Am J Dig Dis 1965; 10: 710-717.
15. Walter MC, Goldschmidt MH, Stone EA, Dougherty JF, Matthiesen DT. Chronic hypertrophic pyloric gastropathy as a cause of pyloric obstruction in the dog. J Am Vet Med Assoc 1985; 186: 157-161.
16. Walter MC, Matthiesen DT, Stone EA. Pylorectomy and gastroduodenostomy in the dog: Technique and clinical results in 28 cases. J Am Vet Med Assoc 1985; 187: 909-914.

개에서의 만성 비후성 유문 위장애에 대한 수술적 치료

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요 약: 10년령의 암컷 요크셔테리어 개가 간헐적인 구토와 만성 체중감소, 다갈증 그리고 쇠약증으로 건국대학교 수의과대학 부속 동물병원에 내원하였다. 방사선 검사에서 가스와 액체가 찬 위 확장이 확인 되었고, 조영제 투여 후 60분이 경과하여도 조영제가 위에서 소장으로 내려가지 않았다. 초음파 상에서는 위체 부위에 상당량의 액체가 저류 되어 있었고, 유문부 점막이 비후되어 있는 것이 확인되었다. 그래서 유문부 협착으로 의한 위 폐쇄가 강력하게 의심되었다. 수술에서 비대된 위점막은 충분히 제거하였고, Heineke-Mikulicz 유문성형술을 적용하였다. 조직학적 소견상 위점막의 비후와 미약한 미만성의 림포구성 형질세포성 위염을 나타내었다. 모든 소견을 종합하여 볼 때, 만성 비후성 유문 위장애로 진단하였다. 간헐적인 구토와 활력저하는 수술 후 9일간 보였다. 이후 환축은 점차적으로 회복되었다. 한달 뒤 환축은 정상적인 식욕과 활력을 나타내었다.

주요어 : 만성 비후성 유문 위장애, Heineke-Mikulicz 유문성형술, 요크셔테리어