CASE REPORT

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Double lumen appearance of the cecum presumably caused by acetyl salicylic acid- induced ileocecal valve ulcers and deformation

Asetil salisilik asit kullanımına bağlı oluşan ileoçekal valv ülserleri ve deformasyonu sonucunda gelişen çekumda çift lümen görünümü

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The use of non-steroidal anti-inflammatory drugs, especially aspirin, has continued to increase with aging and related cardiovascular diseases. Since their well-known side effect is gastroduodenal mucosal injury, aspirin-induced intestinal damage has also become a growing problem. Herein, we present a case with an advanced anatomical impairment of the ileocecal valve deformation, presumably caused by enteric-coated acetyl salicylic acid induced recurrent ulcers.

Keywords: *Colonoscopy, intestinal ulcers, enteric-coated acetyl salicylic acid*

Günümüzde; non steroidal anti-inflamatuvar ilaçların, özellikle aspirinin kullanımı yaşlanma ve yaşlanmayla ilişkili kardiyovasküler hastalıkların sıklığındaki artışla birlikte giderek artmaktadır. Aspirinin; gastroduodenal mukozal hasar yapıcı etkisi gibi en iyi bilinen yan etkisinin yanında yine aspirin nedeniyle oluşan intestinal hasar da daha sık karşımıza çıkmaktadır. Burada muhtemelen enterik-kaplı asetil salisilik asit kullanımı nedeniyle oluşan rekürran ülserlere bağlı gelişen ileoçekal valv deformasyonlu olguyu sunuyoruz.

Anahtar kelimeler: *Kolonoskopi, intestinal ülserler, enterik-kaplı asetil salisilik asit*

INTRODUCTION

The prevalence of non-steroidal anti-inflammatory drugs (NSAIDs) (including aspirin)-induced intestinal injury is higher than had been expected (1). The appearance of NSAID-induced intestinal injury varies, appearing variously as diaphragm-like strictures, ulcers, erosions, and mucosal redness (2-5). Herein, we present a case with an advanced anatomical impairment of the ileocecal valve deformation, presumably caused by enteric-coated acetyl salicylic acid (EC-ASA)-induced recurrent ulcers.

CASE REPORT

A 68-year-old male was admitted to the hospital with the complaints of weakness and dyspnea. He had a history of myocardial infarction 12 years ago, and he had been followed until the admission for congestive heart failure. He was still using medications intended for this disease, including 300 mg/day EC-ASA. He did not have any gastrointestinal complaints. On the laboratory examination, deep anemia was determined (hemoglobin: 6.8 g/dl, hematocrit level: 20%, and ferritin level: 2.4 ng/ml). Additionally, occult blood test in the stool examination was found to be positive. Subsequently, upper gastrointestinal endoscopy (revealing pangastritis) and colonoscopy were performed. During colonoscopy, double lumen appearance of the proximal side of the colon was determined (Figure 1). During the procedure, it was realized that one of the lumens was the base of the cecum (larger arrow #1), and the other was the ileum (larger arrow #2). Both on the mucosa of the terminal ileum and the anatomically disrupted ileocecal valve, multiple ulcers were present (small arrows in Figure 1 and Figure 2). Biopsy specimens taken from the edges of the ulcers were reported as non-specific inflammatory findings. The valve deformation on the cecum resulted in pseudodiverticulum formation (larger arrow #3). Furthermore, two polyps on the cecum determined during colonoscopy were excised (black, broken arrows). The one on the left side was adenomatous and hyperplastic (mixed polyp), while the other one had only adenomatous characteristics. The remaining part of the colon appeared normal. The anemia of the patient was secondary to the bleeding of these ulcers. The sedimentation rate, high sensitive C-reactive protein, anti-nuclear antibody (ANA), anti-neutrophil cytoplasmic antibody (ANCA) levels, PPD and pathergy test, abdominal computerized tomography, and small bowel passage radiography evaluation were also reported as normal. After exhaustive investigation, these interesting findings in ileocolonoscopy were thought to be associated with the long-term administration of EC-ASA. Consequently, EC-ASA treatment was stopped, and 60 mg/day lansoprazole with 1000 mg/day met-

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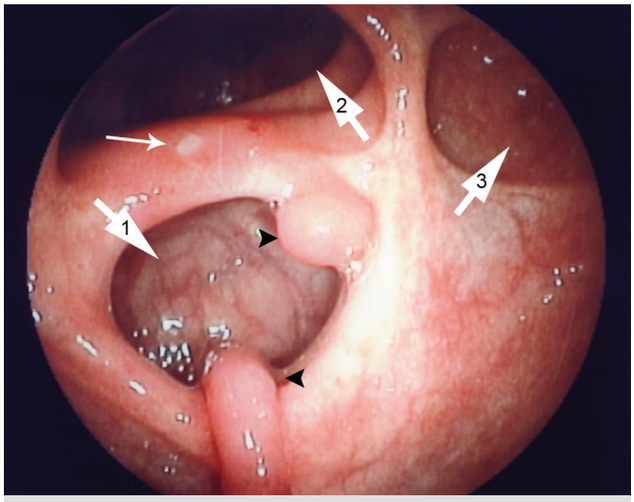

Figure 1. Double lumen appearance of the cecum. Entrance of the base of the cecum (larger arrow #1), ileal orifice (larger arrow #2), ileocecal valve deformation, and ulcer are shown. Addionally, two polyps (black broken arrows) at the level of the ileocecal valve and pseudodiverticulum formation at the right side of the figure (larger arrow #3) are shown.

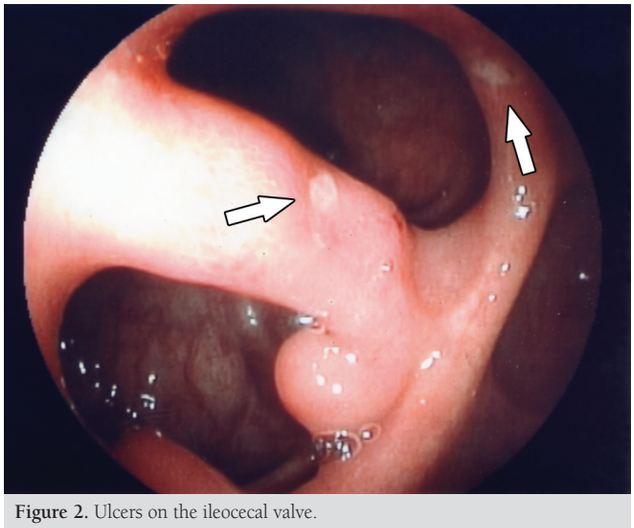

Figure 2. Ulcers on the ileocecal valve.

ronidazole was administered to the patient. After two weeks, treatment with metronidazole was stopped and at the end of one month, EC-ASA was added to lansoprazole. During this period, no decrease in hemoglobin levels was seen, but follow-up ileocolonoscopy could not be performed due to the patient's refusal.

DISCUSSION

Prostaglandins (PGs) are involved in the regulation of gastrointestinal blood flow and various mucosal functions such as increasing mucus secretion. The decrease in PG production is considered to be the main cause of small bowel injuries due to NSAIDs (6-10). First, NSAIDs solubilize lipids of phospholipids on the mucosal surface, so the epithelial mitochondria are directly damaged. Second, the mitochondrial damage depletes intercellular energy and leads to calcium efflux and induction of free radicals, a disruption of intercellular junctions occurs, and mucosal permeability increases in the small intestinal mucosa. Finally, the mucosal barrier becomes weakened, so bile acid, proteolytic enzymes, intestinal bacteria, or toxins can easily penetrate into the epithelial cells, resulting in mucosal injury (9). EC-ASA has been developed to prevent gastric damage and dissolves in the proximal small intestine, which might allow aspirin to have contact with the intestinal mucosa at a high concentration. Enteric-coated aspirin might injure the small bowel through a topical irritant effect as well as via the inhibitory effect on cyclooxygenase (COX) activity (1).

In our patient, existing congestive heart failure could have aggravated the intestinal toxic effects of EC-ASA due to circulatory disorder. On the other hand, we could not determine any other pathologies causing intestinal ulcers (e.g., Crohn disease, tuberculosis) in spite of detailed immunological, histological and serological evaluation.

The basic treatment for NSAID-induced injury is discontinuation of the NSAIDs (1). However, even if temporary cessation of the NSAIDs is possible, long-term cessation of NSAIDs is frequently impossible, and long-term administration of prophylactic drugs is needed for chronic users of NSAIDs or aspirin, especially patients who experience small intestinal bleeding. Some trials have shown the efficacy of metronidazole, sulfasalazine and misoprostol for treatment of NSAID-induced injury (11-13). Clinically, proton pump inhibitors (PPIs) and PG analogs are the first-choice drugs for the prevention of NSAID-induced peptic ulcers and bleeding (14). It is useful to use such drugs to prevent the adverse effects of NSAIDs not only in the stomach but also the small intestine.

In conclusion, to the best of our knowledge, this is the first case of an advanced anatomical impairment of the ileocecal valve deformation presumably occurring due to EC-ASA-induced recurrent ulcers. Thus, routine endoscopic evaluation of the gastrointestinal tract may be beneficial in determining EC-ASA-induced intestinal injury as well as in preventing the associated serious complications, especially in geriatric patients with a history of long-term EC-ASA use.

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