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# Comparison of Duodenal Strictures Between Crohn's and Non-Crohn's Diseases in Children

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## ABSTRACT

**Purpose:** This study aimed to compare the clinical features and laboratory findings of duodenal strictures caused by Crohn's disease (CD) with those of other etiologies in children.
**Methods:** We recruited children diagnosed with duodenal strictures through a full investigation. Clinical, laboratory, endoscopic, and radiological data at diagnosis were collected retrospectively.
**Results:** Of the 11 included patients, four were diagnosed with CD, four with eosinophilic gastrointestinal disorder (EGID), and the other three were grouped together (*Helicobacter*-associated in one and idiopathic disease in two). Serum anti-*Saccharomyces cerevisiae* antibody (ASCA) was positive in four of four (100%) CD cases, four of four (100%) EGID cases, and none (0%) of the other cases ($p$=0.011). The median fecal calprotectin concentration was 994 mg/kg in the CD group (range: 626–2,118 mg/kg), 548 mg/kg in the EGID group (range: 458–1,056 mg/kg), and 124 mg/kg in one patient in the other group ($p$=0.313). Surgery for duodenal obstruction was performed in four patients (one with CD and three with idiopathic and *Helicobacter*-associated conditions) ($p$=0.021), and balloon dilation was performed in one patient with CD and one other patient.
**Conclusion:** Although acquired duodenal strictures are rare in children, they can develop in pediatric patients with CD or EGID. The measurement of serum ASCA and fecal calprotectin levels before endoscopic and histopathological investigations may identify the presence of organic causes of duodenal strictures in children.

**Keywords:** Duodenal obstruction; Duodenal ulcer; Crohn disease; Child

## INTRODUCTION

Duodenal stricturing is a rare but significant condition in children, which often leads to gastrointestinal obstruction and other complications. Duodenal stricturing refers to the narrowing of a duodenal segment due to fibrotic scar tissue formation following ulceration [1]. The incidence of duodenal ulcers is currently estimated to be 2–3 per 1,000 individuals annually [1], and the prevalence of duodenal strictures is rare, and they have only been reported in case reports [2].

Primary duodenal ulcers are uncommon in children aged <10 years but are more prevalent in adolescents [1]. Duodenal ulcers develop because of the corrosive effect of gastric acid secretion on the duodenal epithelium. This damage leads to persistent inflammation and eventual ulceration [3,4].

The main recognized causes of duodenal ulcers include *Helicobacter pylori* infection and prolonged use of nonsteroidal anti-inflammatory drugs (NSAIDs), both of which damage the gastric mucosa and induce ulceration [2]. However, less common inflammatory conditions can also play a significant role in the development of duodenal strictures. Among these, Crohn's disease (CD) and eosinophilic gastrointestinal disorder (EGID) have emerged as important etiologies, underscoring the need for precise differential diagnosis and timely intervention in children.

CD is a chronic inflammatory bowel condition that affects the gastrointestinal tract and develops in up to 25% of children or adolescents [1]. A study in South Korea involving 52 patients found upper gastrointestinal tract involvement in 50% of patients with CD, with gastric ulcers being the most common (19.2%), followed by duodenal ulcers (15.4%) [3]. Additionally, CD can be associated with complications such as strictures, fistulas, abscesses, and perforations [5,6].

EGIDs, excluding eosinophilic esophagitis (non-EoE EGIDs), are rare, chronic inflammatory conditions of the gastrointestinal tract [7]. These disorders share symptoms with CD and other gastrointestinal conditions, including abdominal pain, nausea, vomiting, hematemesis, dyspepsia, diarrhea, hematochezia, and abdominal distension [7]. The diagnosis of EGID relies on mucosal biopsies from the esophagus, stomach, duodenum, and colon to measure eosinophil counts per high-power field (HPF) according to the diagnostic criteria suggested by Papadopoulou et al. [7] in 2023. Endoscopic findings of EGID include deep ulcers that can bleed and even perforate, erosions, mucosal friability, nodularity, edema, and erythema; normal findings can also be observed [7]. Duodenal strictures, obstructions, and perforations in children with EGID have also been reported [8-10].

Despite the risks associated with diseases that cause duodenal strictures, few studies have comprehensively compared duodenal strictures with distinct etiologies in children and adolescents. This gap may increase the likelihood of delayed diagnosis and treatment of duodenal stricture, emphasizing the importance of greater clinical awareness. Furthermore, although less invasive tests such as serum anti-*Saccharomyces cerevisiae* antibody (ASCA) and stool calprotectin levels can be beneficial in distinguishing CD or EGID from other conditions before confirming the diagnosis through endoscopic biopsy, to our knowledge, no studies have been conducted on these tests in cases of duodenal strictures.

Therefore, this study aimed to investigate the causes of duodenal strictures in children and evaluate noninvasive diagnostic markers by comparing the clinical characteristics of CD, EGID, and other potential causes. By analyzing laboratory findings, clinical features, and treatment outcomes, we sought to provide valuable insights into pediatric duodenal strictures and improve the differentiation of these overlapping conditions.

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## MATERIALS AND METHODS

### Study design and participants

This retrospective cohort study was conducted in the Department of Pediatrics, Seoul University Bundang Hospital. Data were collected from patients aged <19 years who were diagnosed with a duodenal ulcer and stricture (or duodenal obstruction) between June 2003 and March 2023.

The diagnosis of CD in children was based on the 2020 guidelines of the European Crohn's and Colitis Organization and the European Society for Pediatric Gastroenterology, Hepatology, and Nutrition (ESPGHAN) on the medical management of pediatric CD [7] as well as the revised 2014 Porto criteria for the diagnosis of inflammatory bowel disease (IBD) in children and adolescents by the ESPGHAN [5]. Histologically, CD is characterized by chronic focal inflammation with or without granulomas, which is essential for diagnosis [5,6].

EGID was diagnosed according to the Joint ESPGHAN and North American Society for Pediatric Gastroenterology, Hepatology, and Nutrition guidelines for children, with a diagnostic criterion of a duodenal eosinophil count of >50 per HPF [7].

### Diagnosis of duodenal stricture

The diagnosis of a duodenal stricture was confirmed using esophagogastroduodenoscopy (EGD), upper gastrointestinal series (UGIS), ultrasonography (USG), computed tomography (CT), magnetic resonance enterography (MRE), and capsule endoscopy. EGD revealed mucosal ulceration, friability, and edema that hindered advancement of the scope into the second portion of the duodenum (**Fig. 1A**). Targeted biopsies of the duodenal bulb, second portion (if passable), and area surrounding the ulcer were performed during EGD, and eosinophil counts, chronic inflammation, and granulomas were histopathologically assessed. Capsule endoscopy detected duodenal ulcers and mild strictures in one patient (**Fig. 1B**).

Radiological investigations, including UGIS, USG, CT, and MRE, revealed severe narrowing immediately after the pylorus, along with focal duodenal dilation, lumen deformation, narrowing, tortuosity, and other abnormalities (**Fig. 1C, D**).

### Laboratory investigations

Blood and stool test results of each patient were retrospectively collected and analyzed. Blood tests included a complete blood cell count, which measured the white blood cell (WBC) count, hemoglobin level, platelet (PLT) count, and absolute eosinophil counts, as well as albumin and ASCA levels. ASCA has been identified in 50–70% of patients with CD, 10–15% of patients with ulcerative colitis, and <5% of healthy controls [5]. This antibody is associated with a severe disease course in CD, and ASCA positivity is defined as a level of >20 U/mL.

Inflammatory markers such as the erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) levels were analyzed. Fecal calprotectin levels, an inflammatory marker in the stool, were collected and analyzed using a fluorescent enzyme immunoassay, with the normal range set at ≤100 mg/kg. Additionally, fecal human hemoglobin was collected, with a level of >0 ng/mL defined as positive.

The allergy-related markers included total immunoglobulin E (IgE) and eosinophil cationic protein (ECP) levels. Total IgE was measured using the electrochemiluminescence


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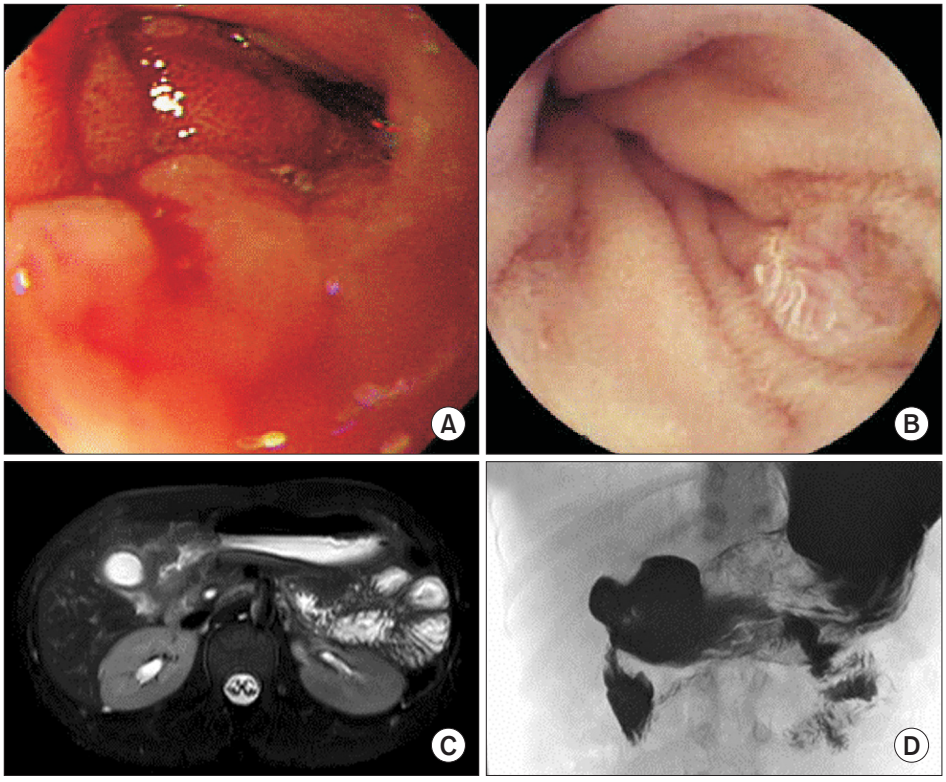


**Fig. 1.** Image distribution of duodenal strictures. (A) Esophagogastroduodenoscopy image showing a duodenal stricture, edematous and erythematous mucosa, and mucosal ulcers. (B) Capsule endoscopy showing duodenal ulcers and strictures. (C) Magnetic resonance enterography image showing severe duodenal wall thickening. (D) Upper gastrointestinal series showing severe duodenal narrowing.

immunoassay method, with a normal range of ≤100 U/mL. ECP levels were measured using the fluoroenzyme immunoassay, with a normal range of ≤13.3 mcg/L.

## Statistical analyses

Statistical analyses were performed using IBM SPSS Statistics for Windows, Version 27.0 (IBM Co.). The chi-squared test was used to assess the relationship between categorical variables, the Kruskal–Wallis test was used to compare median differences between three or more independent groups, and the Mann–Whitney U-test was used to evaluate median differences between two independent groups, all of which provided significant evidence to support our conclusions. Statistical significance was set at $p<0.05$.

## Ethical considerations

This study was approved by the Institutional Review Board of Seoul National University Bundang Hospital (IRB approval number: B-2307-841-102). This study was exempt from the Medical Research Involving Human Subjects Act, as the analyzed data were collected from routine medical practices. Patient confidentiality was ensured by documenting the data securely and anonymously.


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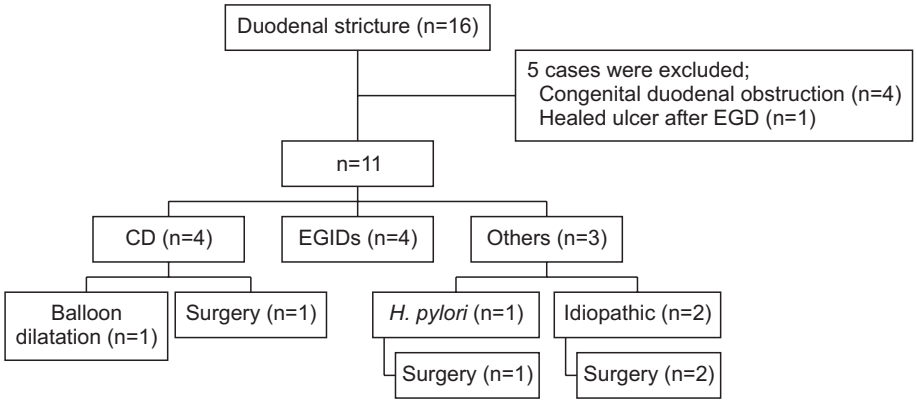


**Fig. 2.** Flow chart of the study patients and treatment.
CD: Crohn's disease, EGIDs: eosinophilic gastrointestinal disorder, EGD: esophagogastroduodenoscopy; *H. pylori: Helicobacter pylori*.

# RESULTS

## Patient characteristics

Of the initially recruited 16 patients, four were excluded owing to congenital duodenal obstruction, and one was excluded because her duodenal ulcer had healed. Thus, the final cohort comprised 11 children: four with CD (including two initially diagnosed with EGID), four with EGID, one with *H. pylori* infection, and two with idiopathic conditions (**Fig. 2**).

The demographic and clinical characteristics of the children with duodenal strictures and ulcers are summarized in **Table 1**. Of the 11 children with duodenal strictures, four (36.4%) were diagnosed with CD, four (36.4%) with EGID, and the remaining three (27.2%) were grouped together (*Helicobacter*-associated in one, idiopathic in two). There were no age or sex differences between the three groups (all $p>0.05$) (**Table 2**).

**Table 1.** Demographic and clinical characteristics of children with duodenal strictures

| Variable | Total (n=11) | *p*-value |
|---|---|---|
| Underlying disease | | |
| Crohn's disease | 4/11 (36.4) | |
| Eosinophilic gastrointestinal disorder | 4/11 (36.4) | |
| Others (*Helicobacter pylori*-associated n=1, idiopathic n=2) | 3/11 (27.2) | |
| Male | 8/11 (72.7) | 0.963 |
| Crohn's disease | 3/4 (75) | |
| Eosinophilic gastrointestinal disorder | 3/4 (75) | |
| Others | 2/3 (66.7) | |
| Median duration of follow-up (y) | 6.8 (0.1–16.7) | 0.302 |
| Crohn's disease | 7.8 (6.8–8.7) | |
| Eosinophilic gastrointestinal disorder | 1.6 (0.5–7.8) | |
| Others | 1.1 (0.1–16.7) | |
| Age at diagnosis (y) | 14.6 (5.4–19.5) | 0.313 |
| Crohn's disease | 12.0 (5.4–15.6) | |
| Eosinophilic gastrointestinal disorder | 13.3 (6.3–17.4) | |
| Others | 16.5 (12.9–19.5) | |
| Number of patients who underwent surgery | 4/11 (36.4) | 0.021 |
| Crohn's disease | 1/4 (25.0) | |
| Eosinophilic gastrointestinal disorder | 0 (0.0) | |
| Others | 3/3 (100.0) | |

Values are presented as number (%) or mean (range).
Statistical significance is considered at $p<0.05$.


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**Table 2.** Comparison of laboratory findings among the three groups according to underlying cause of duodenal strictures in children

| Variable | Total (n=11) | CD (n=4) | EGID (n=4) | Others (n=3) | *p*-value§§ | *p*-value‖‖ |
|---|---|---|---|---|---|---|
| Serum ASCA* (U/mL) | 36.8 (13.3–103.9) | 37.65 (23.9–38.5) | 57.25 (22.1–103.9) | 13.3 | 0.301 | 0.011 |
| Stool calprotectin† (mg/kg) | 731.7 (124–2,118) | 994.5 (837–2,118) | 548 (458–1,056) | 124 | 0.143 | 0.313 |
| Stool hemoglobin‡ (ng/mL) | 557 (0→1,000) | >1,000 (0→1,000) | 114 (0–144) | 0 | 0.823 | 0.801 |
| Serum albumin§ (g/dL) | 4.2 (3.2–4.9) | 4.1 (3.2–4.4) | 4.3 (4.1–4.9) | 3.7 (3.7–4.6) | 0.412 | 0.284 |
| Serum ESR‖ (mm/h) | 3 (2–29) | 3 (2–3) | 11.5 (3–35) | - | 0.719 | 0.572 |
| Serum CRP¶ (mg/dL) | 0.02 (0–10.4) | 0.015 (0.01–1.01) | 0.15 (0–0.26) | 5.205 (0–10.4) | 0.435 | 0.340 |
| Serum total IgE** (U/mL) | 775.5 (4.3–2,500) | 4.3 (4.3–2,500) | 946.5 (279.6–946.5) | 775.5 | 0.344 | 0.265 |
| Serum ECP†† (mcg/L) | 38.4 (4.06→200) | 38.4 (4.06–47.7) | 67.73 (7.45→200) | - | 0.275 | 0.306 |
| Serum eosinophil count‡‡ (/mm$^3$) | 432 (78–1,202) | 441 (90–1,202) | 744 (78–1,089) | 233 (202–532) | 0.824 | 0.341 |

Values are presented as mean (range).
CD: Crohn's disease, EGID: eosinophilic gastrointestinal disorder, ASCA: anti-*Saccharomyces cerevisiae* antibody, ESR: erythrocyte sedimentation rate, CRP: C-reactive protein, ECP: eosinophil cationic protein, IgE: immunoglobulin E.
*CA (ASCA) positivity is over 20 U/mL; †Stool calprotectin positivity is over 100 mg/kg; ‡Stool hemoglobin positivity is over 0 ng/mL; §Serum albumin positivity is below 3.3 g/dL; ‖Serum ESR positivity is over 9 mm/h; ¶Serum CRP positivity is over 0.5 mg/dL; **Serum total IgE is over 100 U/mL; ††Serum ECP positivity is over 13.3 mcg/L; ‡‡Serum eosinophil count positivity is over 500/mm$^3$; §§*p*-value is by *Kruskal-Wallis* test, representing the statistical values among the three groups (CD, EGID, and Others); ‖‖*p*-value is based on the chi-Squared test, representing the statistical value by categorizing each laboratory findings as positive or negative. Statistical significance was set at $p<0.05$.

## Laboratory findings

Among the laboratory findings, ASCA was positive in all patients with CD (4/4, 100%) and EGID (4/4, 100%), but in none of the patients in the other groups (0%) ($p$=0.011), with a cut-off value of 20 mg/kg (**Table 2**).

The median fecal calprotectin level was 994 mg/kg in the CD group (range: 626–2,118 mg/kg), 548 mg/kg in the EGID group (range: 458–1,056 mg/kg), and 124 mg/kg in one of the other patients ($p$=0.313).

Stool hemoglobin was positive in one of four (25%) patients with CD, one of four (25%) patients with EGID, and none (0%) in the other-causes group, revealing no significant differences ($p$=0.801).

Peripheral absolute eosinophil counts increased by more than 500/mm$^3$ in one of four (25%) patients with CD, two of four (50%) patients with EGID, and one of three (33%) patients with other diagnoses. Hemoglobin levels decreased in one of four (25%) patients with CD, one of four (25%) patients with EGID, and one of three (33%) patients with other diagnoses. Serum hemoglobin and eosinophil counts, WBC counts, PLT counts, ESR levels, serum albumin levels, and CRP levels did not significantly differ between the three groups (all $p>0.05$).

## Treatment outcomes

Among the four patients in the CD group, one was treated with laparoscopic gastrojejunostomy after endoscopic diagnosis of a duodenal ulcer with obstruction. The patient was diagnosed with CD 4 years after presenting with melena and was receiving azathioprine monotherapy. Another patient was initially treated with steroids, methotrexate (MTX), and infliximab, but was receiving vedolizumab when the data were recorded. Two individuals who were initially diagnosed with EGID and later diagnosed with CD were initially treated for EGID with dietary restrictions, montelukast, and steroid nasal sprays. After re-diagnosis with CD, treatment was changed to steroids, MTX, partial enteral nutrition, and a Crohn's disease exclusion diet. However, one of the two patients continued to have stenosis despite medical treatment, leading to endoscopic balloon dilatation five times.

All four patients in the EGID group were initially treated with dietary restrictions (based on individual food allergies), montelukast, and proton pump inhibitors; two of them also received steroid treatment.

Among the three patients with other causes of duodenal strictures, one was *H. pylori* positive and underwent gastrojejunostomy after being diagnosed with a duodenal stricture and ulcer via upper endoscopy. Another patient underwent an uncut Roux-en-Y gastrojejunostomy and small bowel resection for duodenal perforation while undergoing endoscopic balloon dilation for a duodenal stricture. The third patient underwent duodenoduodenostomy and gastrojejunostomy because of duodenal obstruction between the second and third portions.

Overall, duodenal stricture surgery was performed in four patients (one of four patients with CD and three in the other-diagnoses group) ($p$=0.021), and balloon dilation was performed in one patient with CD and one in the other-diagnoses group.

## DISCUSSION

To our knowledge, this study is the first to clarify the underlying causes of duodenal strictures, a rare condition in children, and compare the differences in clinical and laboratory features by dividing them into three groups: CD, EGID, and other diagnoses (including *H. pylori* infection and idiopathic cases).

Acquired duodenal strictures develop as a complication of duodenal ulcers. Numerous studies investigating the prevalence of duodenal ulcers have estimated an incidence of approximately 5–15% in Western populations [2]. The primary etiologies of duodenal ulcers are recurrent or prolonged NSAID use and *H. pylori* infection [2]. Additionally, previously rare causes such as Zollinger–Ellison syndrome, malignancies, vascular insufficiency, and a history of chemotherapy are gaining recognition as increasingly prevalent factors [2]. However, recently, the prevalence of *H. pylori* infections has decreased owing to effective prevention through environmental improvements and antibiotic treatment, and the appropriate use of NSAIDs has increased [11].

Furthermore, with the introduction of tissue eosinophil count measurements during endoscopic biopsy, the diagnostic rate of EGID has significantly increased, with duodenal ulcers being identified most frequently in patients diagnosed with EGID [11]. In a multicenter retrospective cohort study of 317 children and 56 adults with non-EoE EGIDs, gastroduodenal ulceration was noted in 6% of patients with EGID, whereas the predominant finding was a normal mucosal appearance in the stomach (66%), duodenum (83%), jejunum (67%), and ileum (81%) [7]. In another study of 16 patients, gastroduodenal ulcers were detected in three patients (12.5%) [7]. Nevertheless, when a high eosinophil count is detected on endoscopic biopsy, the differential diagnosis should include parasitic infections, *H. pylori* infection, IBD, connective tissue diseases with vasculitis, collagenous colitis, idiopathic hypereosinophilic syndrome, autoimmune diseases, and adenocarcinoma [7].

Duodenal stricturing, a rare complication of duodenal ulcers, has an incidence rate of approximately 3% [12,13]. Regarding duodenal strictures in EGID, 13 case reports have been published in adults [14-26], while six cases have been reported in children, with ages ranging from 4 months to 14 years [8-10,27-29]. In a case report by Maria et al. [8], a 14-year-old boy

with a 6-year history of recurrent aphthous stomatitis and allergic rhinitis, along with a 1-year history of abdominal pain, nonbilious vomiting, dysphagia, and weight loss, was diagnosed with duodenal stenosis using EGD and biopsy, confirming a diagnosis of EGID. In a report by Tan et al. [9], a 2-year-old boy with a 6-week history of weight loss and bilious vomiting underwent a contrast study and EGD, which revealed a duodenal stricture and eosinophilic infiltrates on biopsy. In a case study by Riggle et al. [10], a 16-year-old boy with no prior history of allergies, NSAID use, or cocaine use presented with acute epigastric pain, and CT tomography revealed perforation, which was diagnosed as EGID by biopsy. Our study included two patients with allergies (rhinitis, atopic dermatitis, asthma, and food allergies) who had recurrent abdominal pain, weight loss, and vomiting for years and were diagnosed with EGID through endoscopy, MRE, and UGIS. In contrast, the other two patients without a history of allergies, who had abdominal pain, diarrhea, bloody stools, and vomiting for months, were also diagnosed with EGID through endoscopic biopsies.

Duodenal stricturing is also a rare complication of CD, particularly in pediatric patients, with an incidence of approximately 1% at diagnosis. Moreover, clinically significant duodenal involvement is reported in 0.5–4.0% of all patients with CD [30,31]. CD-related upper gastrointestinal tract (UGT) strictures predominantly occur in the duodenum because CD is often characterized by sustained digestive inflammation that leads to strictures, abscesses, and fistulas [32]. The first report of duodenal CD was presented by Gottlieb and Alpert in 1937, after which Ross reported gastric CD in 1949 [30,33]. Shah et al. [34] detailed the case of a 31-year-old man who presented with a 3-year history of upper abdominal pain, nausea, and vomiting and underwent a UGT series with a small bowel series and an abdominal CT scan, which revealed partial duodenal obstruction. After surgical intervention for the duodenal strictures, the patient was diagnosed with CD based on histopathological examination. In the CD group in the present study, two patients were diagnosed at 10 years of age and two at 17 years of age; two patients exhibited both L1 and L4 involvement, one exhibited only L4 involvement, and one exhibited only L3 involvement (based on the Paris Classification of IBD). Two patients with a history of atopic dermatitis, asthma, and food allergies were first diagnosed with EGID and subsequently diagnosed with CD after presenting with abdominal pain and melena, whereas the other two patients were diagnosed with CD through full investigations after presenting with melena, abdominal pain, and recurrent duodenal ulcers.

Regarding treatment outcomes, in our study, four of the 11 children with duodenal strictures underwent surgical treatment, including one with CD, while the remaining three were from the other diagnoses group. Surgical treatment options for duodenal strictures include duodenal stricturoplasty, duodenal stricture bypass surgery, pancreaticoduodenectomy with gastroduodenoplasty, stricturoplasty, and Roux-en-Y bypass, as well as other approaches such as duodenal balloon dilatation, endoscopic stricture incision, and self-expanding metal stent [35].

Medical (antitumor necrosis factor therapy and immunosuppressants) and endoscopic treatments help prevent surgery in 50% of patients with CD with complications [32]. A retrospective cohort study conducted in 25 French and Belgian centers of (in French) the Groupe d'Etude sur les Affections Inflammatoires Digestives (GETAID) found that approximately two-thirds of non-passable strictures in the UGT owing to CD occurred in the duodenum [32]. However, medical and endoscopic treatments prevented the need for surgery in half of the patients with severe UGT strictures [32]. Schwartzberg et al. [30] reported that approximately one-third of patients ultimately required surgical intervention, which may include options such as resection, bypass, or stricturoplasty.

In this study, one patient with CD underwent repeated balloon dilatation. Perforation occurred during balloon dilatation in one patient with idiopathic disease, necessitating surgery. A study conducted in 86 patients with benign duodenal stenosis between 2002 and 2018 showed a technical success rate of 97.4% and a clinical success rate of 77.8% following balloon dilatation [36]. However, endoscopic dilatation, as an alternative to surgery or medical treatment, is limited to straight, non-angulated strictures up to 5 cm in length and within reach of the endoscopic instruments [37]. Therefore, further research is needed to explore the combination of medical treatment for the underlying disease, balloon dilatation, and surgery to establish more accurate diagnoses and appropriate treatment methods for patients with duodenal strictures.

The long-term follow-up of patients with duodenal strictures is an important issue that should not be overlooked. A study conducted at the Lahey Clinic with 89 patients found that 92% of patients with primary gastroduodenal CD developed distal disease during a follow-up period of 11.7 years [30]. During the 10-year follow-up period in our study, two patients were initially diagnosed with EGID and later re-diagnosed with CD during long-term follow-up. Therefore, continuous follow-up may be important to clarify potential progression to CD in patients initially diagnosed with EGID. Furthermore, continuous follow-up is crucial for thorough monitoring of the disease course in patients with CD with UGT involvement because other distal diseases or duodenal strictures can develop as long-term complications.

A significant finding of this study was that both the patients with CD and EGID with duodenal strictures exhibited positive serum ASCA and elevated fecal calprotectin levels compared with that by patients with other causes, although the number of patients was too small to show statistical significance for fecal calprotectin positivity. In this study, CD and EGID accounted for 36.4% of the causes of duodenal strictures in children, indicating that, in addition to the commonly known causes such as *H. pylori* infection or NSAID use, CD and EGID are important etiologies that require careful differentiation. Additionally, among the four patients in the CD group, two were initially diagnosed with EGID before treatment and were later rediagnosed with CD. Therefore, serum ASCA and fecal calprotectin levels may serve as valuable biomarkers for the differential diagnosis, enabling the timely initiation of targeted therapy for the underlying condition, thereby mitigating the risk of complications.

This study had several limitations. The sample size was relatively small (11 patients) because duodenal stricturing is a rare condition. Additionally, as this was a retrospective study, not all tests were performed on all patients (e.g., ASCA, stool calprotectin, and stool hemoglobin levels). Moreover, it was not possible to confirm a family history of CD or allergies. Furthermore, there has been no consistent long-term follow-up, leading to varying follow-up periods, particularly for patients diagnosed with EGID. The median follow-up period in our study was 1.6 years for patients with EGID and 7.8 years for those with CD; additional long-term monitoring for the possibility of developing distal CD should not be overlooked, considering that patients with EGID also showed serum ASCA positivity at diagnosis. Future studies should be prospective, with a larger number of patients and consistent long-term follow-up.

In conclusion, CD and EGID are common causes of duodenal strictures, emphasizing the importance of thorough differential diagnosis. Our findings also demonstrate that serum ASCA levels and endoscopic biopsy can serve as important markers for these diagnoses. Therefore, this study provides valuable foundational data for the diagnosis of underlying

causes and treatment of duodenal strictures in children, potentially contributing to the improvement of diagnostic and treatment strategies for related diseases.

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