Case Report

Collagenous gastroduodenocolitis in a Korean adolescent: first pediatric case report in Asia

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Abstract: Collagenous gastritis (CG) is a rare disease diagnosed histologically by the subepithelial deposition of collagen bands thicker than 10 μm and the infiltration of inflammatory mononuclear cells in the lamina propria. The definite pathophysiology is yet to be elucidated. However, recent studies have suggested that the collagen deposition may be the result of a reparative process in response to an earlier inflammatory, autoimmune, infectious, or toxic insult. CG is divided into the pediatric- and adult-type. While the pediatric-type is limited to the stomach, the adult-type involves not only the stomach but also the intestine and/or colon. We report a rare case of adult-type CG in a 15-year-old boy who initially presented with abdominal pain and iron-deficiency anemia. Esophagogastroduodenoscopy (EGD) revealed findings suspicious for *Helicobacter pylori* (*H. pylori*) gastritis. Although histology did not reveal the organism, campylobacter-like organism (CLO) test was positive. Based on the diagnosis of suspicious *H. pylori* gastritis, eradication was conducted using the triple drug regimen. However, symptoms of intermittent abdominal pain persisted and diarrhea newly developed one year later. Histologic results from biopsies from the stomach, duodenum, and colon revealed findings compatible with CG, collagenous duodenitis (CD), and collagenous colitis (CC). This is the first pediatric case of collagenous gastroduodenocolitis (CGDC) reported in Asia. It is no longer assumed that adult-type and pediatric-type CG should be classified as an independent disease, but should be considered as similar diseases on a continuous spectrum. Therefore, children and adolescents diagnosed with CG should also consider undergoing a colonoscopy for the evaluation of possible coexisting CC when concurrent lower gastrointestinal symptoms are present. Moreover, considering the possibility of negative findings on the first endoscopy, repeat endoscopy should be considered when symptoms persist.

Keywords: Case report; collagenous colitis (CC); collagenous duodenitis (CD); collagenous gastritis (CG); colonoscopy

Submitted Jul 25, 2021. Accepted for publication Sep 29, 2021.
doi: 10.21037/tp-21-342
View this article at: https://dx.doi.org/10.21037/tp-21-342

Introduction

Collagenous gastritis (CG) is a rare disease diagnosed histologically by the subepithelial deposition of collagen bands thicker than 10 μm in the lamina propria (1). The definite pathophysiology is not proven. However, there is a suggested hypothesis that collagen deposition may be the result of a reparative process in response to an earlier inflammatory, autoimmune, infectious or toxic insult (2-4).

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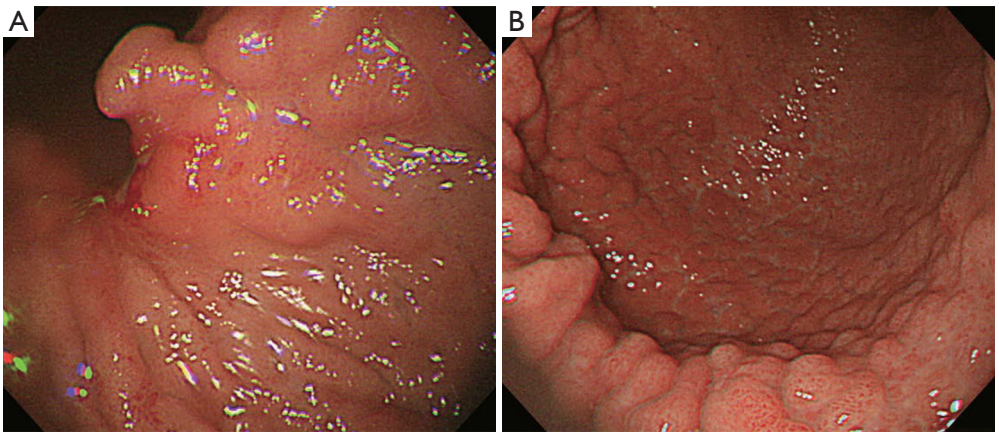

Figure 1 Medium to large sized gastric ulcers in the cardia with diffuse nodular hyperplasia were observed on EGD. EGD, esophagogastroduodenoscopy.

CG is divided into two phenotypes: pediatric- and adult-type. Involvement of the pediatric-type CG is confined to the stomach, while the adult-type CG has concurrent involvement in the small and/or large bowel. It is now acknowledged that adult-type and pediatric-type CG should not be classified as an independent disease, but should be considered as similar diseases on a continuous spectrum (5).

There are only few reports in literature of children and adolescents diagnosed as CG with concurrent collagenous duodenitis (CD) and/or collagenous colitis (CC) (1,2,5-16). We present a rare pediatric case of collagenous gastroduodenocolitis (CGDC) in whom subepithelial deposition of thickened collagen bands were observed in the stomach, duodenum and colon.

We present the following case in accordance with the CARE reporting checklist (available at https://dx.doi.org/10.21037/tp-21-342).

Case presentation

A 15-year-old boy was admitted due to chronic abdominal pain for one year. His abdominal pain was mainly epigastric, and it rapidly aggravated during fasting. Subsequently, unformed stool was observed for one month. Additionally, his weight had decreased from 49.6 to 47.6 kg (4 percentile) during a period of two weeks. His height was 168.8 cm (35 percentile) and his body mass index was 16.71 kg/m^2 (2 percentile). He had been diagnosed with IDA at 11-year-old and had been treated with oral iron supplements continuously. At that time, his hemoglobin level was 7.1 mg/dL; hematocrit 25.0%; white blood cell (WBC) count 5,010/μL; platelet count 465,000/μL; iron 12 μg/dL; ferritin 2 ng/mL; total iron binding capacity (TIBC) 448 μg/dL; transferrin saturation (TS) 2.7%; total protein 6.7 g/dL; and albumin 4.1 g/dL.

On admission, there was no tenderness or rebound tenderness in the abdomen, and his bowel sound was normoactive. Other physical exams were unremarkable. Laboratory exams at admission showed hemoglobin 9.8 mg/dL; hematocrit 32.9%; WBC count 8,130/μL; platelet count 357,000/μL; iron 10 μg/dL; ferritin 5 ng/mL; TIBC 352 μg/dL; TS 2.8%; total protein 6.4 g/dL; albumin 3.9 g/dL; C-reactive protein (CRP) 0.25 mg/dL; and erythrocyte sedimentation rate (ESR) 32 mm/h. His fecal immunochemistry test (FIT) was positive, and fecal calprotectin (FC) level was 573 mg/kg. Other laboratory tests, including liver enzyme, thyroid function test, stool bacterial and virus antigen test were normal. Esophagogastroduodenoscopy (EGD) revealed diffuse mucosal nodularity and multiple large ulcers at the anterior portion of the cardia in the stomach (*Figure 1*). The diffuse nodular hypertrophic mucosa extended through the pylorus to the bulb and second portion of the duodenum. Colonoscopy was grossly normal. Histologic findings of biopsies from the stomach antrum, body, duodenum, and all segments of the colon revealed findings of chronic inflammation, and special stains were not conducted. Campylobacter-like organism (CLO) test was positive, and lansoprazole, amoxicillin, and clarithromycin were prescribed for two weeks to eradicate the suspected *Helicobacter pylori* (*H. pylori*) infection. Urea breath test, which was conducted six weeks later, was negative. There were no adverse events during eradication treatment of

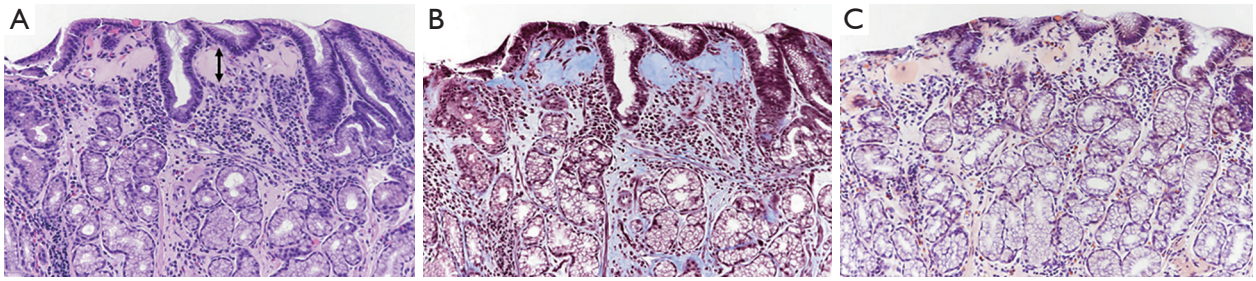

Figure 2 Microscopic images of the mucosal biopsy specimen from the stomach antrum. (A) A dense irregular eosinophilic band is noted below the gastric surface epithelium measuring 60 µm (double headed arrow), and there is chronic inflammation in the lamina propria (H&E, ×200). (B) The thickened subepithelial layer stain blue with Masson trichrome stain (×200). (C) Congo red stains are negative (×200).

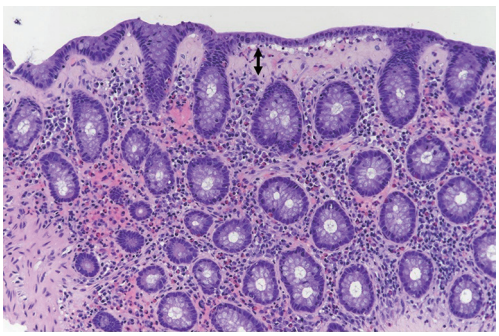

Figure 3 Histopathological examination of colon biopsy shows acellular collagenous dense bands (double headed arrow) underneath the epithelium, measuring 30 µm in thickness (H&E, ×200).

the suspected *H. pylori* infection. After treatment, his weight recovered within two weeks.

However, symptoms of intermittent abdominal pain persisted and diarrhea newly developed one year late. His IDA also did not improve, despite successful *H. pylori* eradication and continuous oral iron supplementation. Laboratory exams showed hemoglobin 9.3 mg/dL; hematocrit 31.3%; WBC count 6,410/µL; platelet count 551,000/µL; iron 11 µg/dL; ferritin 4 ng/mL; TIBC 354 µg/dL; TS 3.1%; total protein 6.2 g/dL; albumin 3.9 g/dL; CRP 0.09 mg/dL; and ESR 5 mm/h. His FIT was negative, and FC level was 331 mg/kg. EGD showed diffuse mucosal nodularity, active ulcers and scars. Histology of the gastric mucosal biopsy showed irregularly thickened subepithelial collagenous bands measuring 60 µm in the stomach antrum, body, and pylorus (*Figure 2A*) and 50 µm in the duodenum, which strongly stained with Masson-trichrome (*Figure 2B*) but negative for amyloid with Congo red stain (*Figure 2C*). CLO test was negative. The gross appearance of the colonic mucosa on colonoscopy again showed no abnormalities. However, on random colonic biopsy from the cecum, ascending colon, transverse colon, descending colon, and sigmoid colon, subepithelial collagenous bands of 30 µm were observed in all biopsy specimens (*Figure 3*).

He was diagnosed with CG with concurrent CD and collagenous colitis. He started treatment with lansoprazole and oral iron supplementation was continued. Further serum immunological tests, including antinuclear antibody, anti-smooth muscle antibody, and antibodies to autoimmune diseases commonly observed in adult-type CG, were all negative.

Treatment with lansoprazole and oral iron supplementation was continued for a year. However, his IDA had not improved (*Table 1*). Follow-up EGD conducted one year after the diagnosis of CGDC, showed improvement of previously observed active ulcers and scars, while diffuse nodularity, and mucosal erythema and erosions were continuously observed. Follow-up colonoscopy repetitively showed no endoscopic abnormality. Histology again revealed collagen deposits with chronic inflammation in the stomach, duodenum and colon.

There were no significant changes in findings at the second- and third-year follow-up. To treat his IDA that did not improve by continuous treatment with lansoprazole and continuous iron supplements, systemic corticosteroids were attempted between the second- and third-year follow-up. However, two months treatment with oral prednisolone was not effective.

He has been followed for three and a half years. He currently does not have symptoms of abdominal pain and diarrhea. Despite continuous treatment with lansoprazole and oral iron supplementation, his IDA has not shown a

Table 1 Laboratory results of the patient over time

Laboratory tests	Initial diagnosis of IDA, 3 years before visit (11-year-old)	Initial visit (15-year-old)	At diagnosis of CGDC (16-year-old)	One year after diagnosis of CGDC (17-year-old)	Two years after diagnosis of CGDC (18-year-old)	Three years after diagnosis of CGDC (19-year-old)
WBC count, /μL	5,010	8,130	6,410	5,870	10,040	3,810
Hemoglobin, g/dL	7.2	9.8	9.3	8.7	9.2	10.3
Hematocrit, %	25.0	32.9	31.3	30.8	30.0	35.7
MCV, fL	60.4	63.8	61.0	62.3	58.6	61.7
MCH, pg	17.4	19.0	18.1	17.6	18.0	17.8
Platelet count, /μL	465,000	357,000	551,000	461,000	434,000	461,000
Iron, μg/dL	12	10	11	11	11	16
Ferritin, ng/mL	2	5	4	3	3	4
TIBC, μg/dL	448	352	354	372	374	370
TS, %	2.7	2.8	3.1	3.0	2.9	4
Total protein, g/dL	6.7	6.4	6.2	6.2	6.7	6.3
Albumin, g/dL	4.1	3.9	3.9	4.1	4.0	3.9
Globulin, g/dL	2.6	2.5	2.3	2.1	2.7	2.4
ESR, mm/h	NA	32	5	3	9	7
CRP, mg/dL	NA	0.25	0.09	0.07	NA	0.08
FC, mg/kg	NA	573	331	NA	NA	674
FIT, ng/mL	NA	357	53	NA	NA	NA

IDA, iron defiency anemia; CGDC, collagenous gastroduodenocolitis; WBC, white blood cell; MCV, mean corpuscular volume; MCH, mean corpuscular hemoglobin; TIBC, total iron binding capacity; TS, transferrin saturation; ESR, erythrocyte sedimentation rate; CRP, C-reactive protein; FC, fecal calprotectin; FIT, fecal immunochemistry test; NA, not available.

remarkable improvement (*Table 1*).

This case report was approved by the Institutional Review Board (IRB) of Soonchunhyang University Bucheon Hospital (IRB number SCHBC-2021-01-021-001). All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and national research committees and with the Helsinki Declaration (as revised in 2013). Written informed consent was obtained from the patient's parent or legal guardian for publication of this case report and accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

Discussion

CG is a rare disease, characterized by marked subepithelial collagen deposition. The incidence rate of childhood-onset CG has been estimated as 0.25 per 100,000 person-years, and its prevalence is 2.1 per 100,000 (14). In children, it usually occurs before adolescence and is approximately 1.6–4 times more frequent in females than in males (14,17). The definite pathogenesis is yet to be elucidated. However, it is assumed that CG may occur due to an abnormal response to toxic or infectious stimuli, which results in chronic inflammation associated with abnormal collagen deposition and secretion from fibroblasts (2-4). The pathological features of CG must include the deposition of collagen bands thicker than 10 μm in the subepithelial layer, and it is often accompanied by a chronic inflammatory infiltrate.

Traditionally, there are two phenotypes of CG, which are the pediatric-type and the adult-type. These phenotypes are classified according to the involved location in the gastrointestinal tract. Involvement of the pediatric-type CG is confined to the stomach, while the adult-type CG

has concurrent involvement in the small or large bowel. Nodularity of the stomach body is the characteristic finding in pediatric-type CG. The size and number of nodularity depend on the severity of the inflammation (18). This typical nodularity is not the result of mucosal thickening, but due to the depressed mucosa surrounding the nodules (18). In the pediatric-type, symptoms include intractable anemia and predominant epigastric pain, dyspepsia and vomiting. Symptoms such as weight loss and diarrhea are not typical.

The anemia of pediatric-type CG is probably a result of these dilated capillaries leading to microscopic blood loss and/or decreased iron absorption (2,14,18). A recent large pediatric cohort has provided evidence of positive FIH tests and presence of GI bleeding supporting the hypothesis that IDA results from GI blood loss rather than poor absorption (2). Meanwhile, another study suggested that IDA results from decreased iron absorption due to gastric hypochlorhydria or other mechanisms (14).

In contrast, adult-type CG occurs commonly with concurrent CD and/or CC, presenting with watery diarrhea. It is also frequently accompanied with other autoimmune conditions (5,10). Colonoscopic findings of CC shows a relatively even distribution of inflammation and atrophic changes or edematous colonic mucosa with pseudopolyps in the colon (18,19). Because of these differences, it has been suggested that the pathogenesis differs between the two phenotypes (20). However, recent studies have suggested that these two phenotypes should be conceived as a continuous disease spectrum rather than separate diseases, using the terms of CG with or without CG instead of pediatric-type and adult-type CG (2,5,14).

CG with concurrent CD and/or CC is very rare, and to date there are 24 pediatric cases reported in case reports or in cohort studies (1,2,5-16) including this case (*Table 2*). Recently, Beinvogl *et al.* have reported the largest cohort of CG in children of 40 patients (2). In this study, concurrent CD was noted in 7/40 (17.5%), CC in 3/40 (7.5%) and ileitis in 1/40 (2.5%). One patient (2.5%) had involvement in the entire GI tract, who had been previously reported in a case report (13). In the second largest pediatric cohort of 15 CG patients reported by Käppi *et al.*, CD was noted in 1/15 (6.7%), and CC in 1/15 (6.7%). Considering that approximately 120 cases of pediatric CG have been reported in literature, cases with pediatric CG with concurrent CD and/or CC comprises approximately 20% of the total cases with pediatric CG (1,2,5-16). Cases of pediatric CGDC as in this case are even more scarce, and to our knowledge there are only five cases including ours, comprising approximately 4% of the total pediatric cases of CG (5,8,9,13).

While, IDA, epigastric pain, and upper endoscopic and histologic findings were typical in our case, a suspicious preceding infection with *H. pylori* was a unique finding. There are only a few cases of patients diagnosed with CG after infection and successful treatment with *H. pylori* (15,21,22). All patients were treated with triple therapy with amoxicillin, clarithromycin and proton pump inhibitor (PPI) for the eradication of *H. pylori*. Despite the absence of a definite diagnosis of *H. pylori* infection, our patient was treated with triple therapy. However, his symptoms did not improve. Hence, endoscopy was redone, and CG was diagnosed.

The association between CG and *H. pylori* infection is yet to be elucidated. However, it is known that the clinical outcome of *H. pylori* infection is highly variable owing to the diversity of microorganism virulence factors and the inflammatory response of the host (22,23). *H. pylori* are also capable of binding to connective tissue proteins and induce the release of pro-inflammatory chemokines leading to an inappropriately upregulated inflammatory reaction, resulting in fibrosis and collagen deposition (22,24). Despite these hypotheses, there are few reports related to *H. pylori*. Meanwhile, current evidence suggests that the vast majority of pediatric CG cases are not associated with *H. pylori*. (2,14) Considering the relatively high prevalence of *H. pylori* in South Korea compared to Western countries, the concurrent finding of *H. pylori* and CG may be merely just a coincidence. Future large-scale studies are required to elucidate whether *H. pylori* possesses a risk for the development of CG.

An important lesson from this case would be the necessity of repeat endoscopies if symptoms are to persist. In this case CGDC was not initially diagnosed but was diagnosed on the second EGD and colonoscopy. Although endoscopic findings of the first EGD was suspicious for either *H. pylori* gastritis or CG, histology revealed findings of only chronic inflammation. No thickened subepithelial collagenous bands were detected. Masson-trichrome stain was retrospectively conducted later after diagnosis. However, subepithelial collagenous bands were not detected. Follow-up upper and lower endoscopies were conducted 1-year later, after the new development of diarrhea and persistent intermittent abdominal pain for a year. This is when subepithelial collagenous bands were detected in the stomach, duodenum, and colon, and the patient was finally diagnosed with CGDC. This shows that

Table 2 Reported pediatric cases of CG with concurrent CD and/or CC in English literature

Author (publication year)	Age (y)	Gender	Clinical presentation	CD	CC	Treatment
Colletti *et al.* (1998) (6)	11	M	Anemia, abdominal pain, diarrhea	No	Yes	Oral iron, PPI, sucralfate, 5-ASA
Camarero *et al.* (2003) (7)	15	F	Diarrhea	No	Yes	NA
Kori *et al.* (2007) (15)	12	F	Nausea, vomiting	Yes	No	PPI
Leiby *et al.* (2008) (8)	2	M	Diarrhea, weight loss, vomiting, low-grade fever	Yes	Yes	Systemic corticosteroids, PPI, 5-ASA, bismuth subsalicylate
Billiémaz *et al.* (2009) (9)	9 mon	M	Diarrhea, acute severe dehydration	Yes	Yes	Prednisolone, budesonide, night enteral nutrition, gluten-free diet
Suskind *et al.* (2009) (10)	15	M	Diarrhea, oral ulcers, abdominal pain	No	Yes	PPI, steroid, 5-ASA
Camarero Salces *et al.* (2011) (11)	9	F	Anemia, abdominal pain	No	Yes	5-ASA
Ma *et al.* (2015) (5)	11	F	Diarrhea, nausea, vomiting	Yes	Yes	Iron
Koide *et al.* (2015) (16)	12	F	Hematemesis	Yes	No	Famotidine
Matta *et al.* (2018) (1)	5	F	Anemia, diarrhea	No	Yes	Oral iron, gluten free diet
Matta *et al.* (2018) (1)	13	M	Anemia, abdominal pain, diarrhea	No	Yes	Oral iron, gluten and dairy free diet
Beinvogl *et al.* (2020) (13)	2	F	Diarrhea, vomiting, low-grade fever	Yes	Yes	Prednisolone, gluten and dairy free diet, elemental formula, PPI, methotrexate, budesonide
Käppi *et al.* (2020) (14)	4	F	Diarrhea	No	Yes	NA
Käppi *et al.* (2020) (14)	8	M	Anemia	Yes	No	NA
Beinvogl *et al.* (2021) (2)—7 patients	NA	NA	NA	Yes	No	NA
Beinvogl *et al.* (2021) (2)—3 patients	NA	NA	NA	No	Yes	NA
This case	15	M	Anemia, abdominal pain, diarrhea	Yes	Yes	PPI, oral iron, prednisolone

CG, collagenous gastritis; CC, collagenous colitis; CD, collagenous duodenitis; M, male; F, female; PPI, proton pump inhibitor; 5-ASA, 5-aminosalicylic acid; NA, not available.

initial histologic findings may be negative, and that repeat endoscopy is warranted if symptoms persist.

As the pathophysiology of CG is yet unclear, there is no obvious standard for the treatment of CG, and there is currently lack of efficient treatment (25). Several therapies have been attempted in CG patients, including corticosteroids, ranitidine, misoprostol, sucralfate, 5-aminosalicylate acid, and hypoallergenic diets (7). It has been reported that among patients with CG with CC, few have experienced clinical, endoscopic, histologic improvement even after treatment with corticosteroids and azathioprine (1). Our patient started treatment with a PPI and oral iron supplementation for IDA. During follow-up for three years, his abdominal pain and diarrhea improved. However, his IDA and histologic findings have not shown improvement even after treatment with systemic corticosteroids.

Meanwhile, Winslow *et al.* (26) described the 12-year clinicopathologic evolution of CG in a single patient. Gastric corpus biopsy specimens revealed active chronic gastritis, subepithelial collagen deposition, smooth muscle hyperplasia, and mild to moderate glandular atrophy. Particularly, the patient's biopsy specimens showed a significantly lower number of antral gastrin cells, along with a significant corpus endocrine cell hyperplasia. These findings are suggestive of an increased risk of endocrine neoplasia, especially adenocarcinoma. Therefore, careful

observation and follow-up is required in patients with CG.

Our case is the first to report CG with concurrent CD and CC in an Asian child. Children diagnosed with CG who present with lower GI symptoms such as diarrhea should also consider undergoing a colonoscopy for the evaluation of coexisting CC. Moreover, considering the possibility of negative findings on the first endoscopy, repeat endoscopy should be considered when symptoms persist. Furthermore, large scale longitudinal cohort studies are required in the future to better understand and reveal the pathogenesis of these collagenous diseases.

Acknowledgments

Funding: This work was supported by the Soonchunhyang University Research Fund (No. 20210021) granted to YML.

Footnote

Reporting Checklist: The authors have completed the CARE reporting checklist. Available at https://dx.doi.org/10.21037/tp-21-342

Peer Review File: Available at https://dx.doi.org/10.21037/tp-21-342

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://dx.doi.org/10.21037/tp-21-342). YML reports that this work was supported by the Soonchunhyang University Research Fund (No. 20210021) granted to her. The other authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and national research committees and with the Helsinki Declaration (as revised in 2013). Written informed consent was obtained from the patient's parent or legal guardian for publication of this case report and accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

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Cite this article as: Kang B, Um SH, Yun J, Kim HK, Choe BH, Lee YM. Collagenous gastroduodenocolitis in a Korean adolescent: first pediatric case report in Asia. Transl Pediatr 2021;10(11):3096-3103. doi: 10.21037/tp-21-342