Collagenous colitis and collagenous gastritis in a 9 year old girl : a case report and review of the literature

C. Camarero Salces[1], P. Enes Romero[1], C. Redondo[3], J.M. Rizo Pascual[1], G. Roy Ariño[2]

(1) Department of Pediatrics, (2) Department of Immunology, (3) Department of Pathology, Hospital Universitario Ramón y Cajal. Carretera de Colmenar Km 9,100, Madrid 28034, Spain.

Abstract

Collagenous gastritis is a rare disease in the general population and collagenous colitis has seldom been reported in children. We report a girl with both diseases and review the literature on this association afetr a systematic search of Pubmed, Medline and Embase databases.. The girl, diagnosed of collagenous colitis at the age of 2 years, started with abdominal pain and anaemia at the age of 9 years and was diagnosed of collagenous gastritis in the gastric biopsies. After review of the literature, we found 66 reported cases (33 children, 33 adults, 68% females), 56 patients with collagenous gastritis and 16 children with collagenous colitis. Both disorders coexisted in 20 patients. The main presenting symptoms are abdominal pain and anaemia in patients with collagenous gastritis and diarrhoea and weight loss in patients with both disorders. Hypoalbuminemia was found in 9 patients with both diseases and protein losing enteropathy was demonstrated in 3 cases. Deposits of collagen in the duodenum were observed in 13 of 19 patients with both diseases. Seventeen of 66 patients had associated autoimmune disorders, particularly in patients with both diseases (35%). These conditions have a chronic course but gastric or colonic malignancies have not been communicated to date. In conclusion, collagenous gastritis and collagenous colitis mainly affects women and can occur at any age. Their association is exceptional. These disorders, although rare, should be considered in patients with anaemia and epigastric pain, watery diarrhoea or protein losing enteropathy. (Acta gastroenterol. belg., **2011**, 74, **468-474**).

Key words : collagenous gastritis, collagenous colitis, protein losing enteropathy, collagen, stomach, colon.

Introduction

Collagenous gastritis (CG) and collagenous colitis (CC) are characterized by the deposition of a subepithelial collagen band in the mucosa (SCB). CG is an extremely rare disease. Collagenous colitis is primarily a disorder of middle-aged women, with more than 500 reported cases (1), it is very rare in children, and only 15 cases have been reported in the literature. Available data on the coexistence of both conditions have seldom been reported and consist mainly of case reports.

Here we report a case describing the clinical-pathological aspects of a patient diagnosed of collagenous colitis at the age of 2 years and of collagenous gastritis 7 years later. We also review the literature on these two entities gathering information about clinical characteristics and outcomes.

Case report

A 9 year old girl, previously reported (2), was diagnosed of collagenous colitis at the age of 2 years. An upper gastrointestinal endoscopy performed at the initial evaluation was normal and gastric biopsies were not taken. *Aeromonas hydrophila* was isolated in faeces and the diarrhoea resolved gradually with antibiotic treatment and a stool culture performed 1 month later was negative for bacterial pathogens. The following year, the patient presented two episodes of watery diarrhoea, with the isolation of *A. hydrophlila* in the stool, which again resolved with antibiotic treatment. A third episode of diarrhoea was treated with bismuth for 7 weeks with partial and temporary improvement. However, watery diarrhoea subsequently reappeared and follow-up was characterized by persistent watery diarrhoea and intermittent colicky abdominal pain. At the age of 6 years, she began oral budesonide (9 mg / day) for 6 months. A complete clinical improvement was observed, but the diarrhoea reappeared 1 month after discontinuing the medication. A new attempt was made with oral_mesalazine treatment (50 mg/Kg/day) but the diarrhoea still continued and, as she reported increased abdominal cramps, this therapy was discontinued one month later. Symptoms persisted during the following years. Routine blood parameters were normal and the erythrocyte sedimentation rate was mildly elevated. She underwent colonoscopy on 2 occasions, and colonic findings both at endoscopy and histology were unchanged. Multiple stool cultures performed during the follow-up were sterile *and A. hydrophila* was not isolated again.

At the age of 9 years she started with epigastric pain. On physical examination, her weight and height were in the 10th and 3rd percentiles for age, respectively, she was pale and other system examinations were normal. Routine blood parameters showed a haemoglobin level

Correspondence to : Cristina Camarero Salces, Hospital Universitario Ramón y Cajal. S. de Pediatría, Carretera de Colmenar Km 9,100, Madrid 28034, Spain. E-mail : ccamarero.hrc@salud.madrid.org

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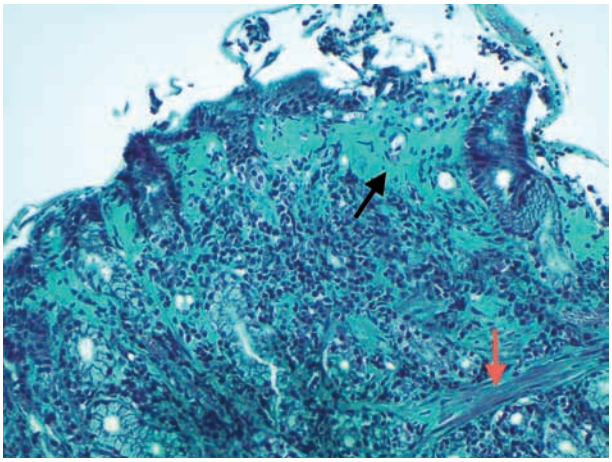

Fig. 1. — Masson trichrome staining of the gastric biopsy with thickened subepithelial collagenous band (*black arrow*), moderate chronic inflammatory infiltrate and striking subepithelial and interlacing bundles of thick collagen fibers between the atrophic glands (*red arrow*).

of 9.5 g/dl, ferritin 2.71 ng/ml, erythrocyte sedimentation rate 34 mm/h. Serum electrolytes, glucose, creatinine, urea, transaminases, albumin and immunoglobulins were within normal limits. Serum IgA antiendomysial, IgA antitransglutaminase, antinuclear and antimitochondrial antibodies, rheumatoid factor, pANCA and cANCA were negative. The stool culture was negative. Due to abdominal complaints, she underwent an upper gastrointestinal endoscopy, which revealed the nodularity of gastric corpus and antrum. The duodenum and oesophagus were normal. Multiple biopsy specimens from the stomach showed moderate-intense glandular atrophy, a moderate chronic inflammatory infiltrate without signs of activity and striking subepithelial and interlacing bundles of thick collagen fibers between the atrophic glands (Fig. 1). *Helicobacter pylori* was not detected. Duodenal biopsies showed no pathologic findings. Flexible sigmoidoscopy was normal and colonic mucosal biopsies demonstrated typical features of collagenous colitis.

Literature review

Design and data sources

We performed a search in Pubmed, Ovid Medline and Elsevier Embase with the terms "collagenous gastritis" and "collagenous colitis and children". Pubmed identified 697 potential citations, Ovid Medline 753 and Elsevier Embase 942. We also added cases reported at international Gastroenterology meetings. Two authors independently selected potentially relevant articles by screening retrieved citations and abstracts. We limited the articles to those that included pediatric patients diagnosed of collagenous colitis and both pediatric and adult patients diagnosed of collagenous gastritis with or without associated collagenous colitis. Exclusion criteria were based on the lack of information about the clinical data. A final search was undertaken 10/15/2010 before reporting our findings.

Data extraction

One researcher abstracted key elements from all included publications into standardized tables. A second researcher verified these data. Four researchers, including the two named before, critically appraised all the findings. Disagreements were resolved by discussion with the clinical leaders.

Limitations

In some cases further details were unavailable, as the reports were only in abstract form and part of a collection of several cases.

Results and discussion

Collagenous gastritis is an extremely rare condition whose aetiology, pathogenesis and long-term prognosis are unknown. Collagenous colitis is more frequent in the adult age1 but is extremely rare in the paediatric population. Information on collagenous gastritis in children and adults and collagenous colitis in children rely on case reports and when both diseases coexist, the data are even scarcer. Our exhaustive review of the literature on these diseases enabled us to collect all the patients and draw some conclusions about these rare pathologies. In this review we found 66 cases (33 children, 33 adults) with these collagenous inflammatory diseases : 56 patients with collagenous gastritis (3-40,this case) and 16 children with collagenous colitis (2,13,29-31,this case,41-49). In 20 patients both diseases coexisted (2,6,13,14,25, 26,29-40,this case).

A substantial proportion of patients (68%) were women with a similar proportion in children as in adults. It has been pointed out that in the collagenous mucosal inflammatory diseases of the gastrointestinal tract there is a predilection for women (50) and these results confirm this observation.

In some patients, as the girl presented here, the involvement is not limited to only one area and the SCB may appear in other locations. In this review we found that colonic biopsies were obtained during colonoscopy in 38 of the 56 patients with CG. In 18 of them the mucosa was normal or showed minimal changes (6-12,15-20,22,24) and 20 demonstrated CC. In 10 adults (6,10,13,14,25-28) and 8 children (3,5,10,13, 14) the colon was not investigated. It is unlikely that these patients had CC since diarrhoea,a characteristic symptom of CC,was present in only 2 patients (25,26) who had a collagen sprue. In 36 patients with CG and normal or not investigated colon, epigastric pain and anaemia, sometimes severe, were the most common symptoms.

The association of CG with CC has been reported in 6 children (2,13,29-31,this case), including the girl described here, and in 14 adults (6,14,25,26,32-40) (Tables 1 and 2). The main symptoms in all patients with this association were diarrhoea, present in all but 2 patients (26,31) (90%) and an important weight loss, reported in 50% of the patients (13,25,31,32,35-39). Anaemia and epigastria pain, the main symptoms in patients with CG without CC, were observed less frequently in patients with both diseases : 7 patients (35%), 3 children (13,31,this case) and 4 adults (33,37,39,40), referred to abdominal pain and 3 (14,26,37) adult patients and the girl reported here (20%) presented anaemia. An outstanding clinical finding is that hypoalbuminemia was observed in 9 patients (3 children (29,30,31), and 6 adults (25,32,36,38-40)) with CG and CC and protein losing enteropathy was demonstrated in 3 (32,36,40). In all of them, subepithelial collagen deposits were present in the duodenum. Collagenous sprue or coeliac disease was observed in 3 patients (25, 29,38), but in the other patients no other possible cause of hypoalbuminemia was discovered. Protein losing enteropathy associated to collagenous colitis has been previously reported in only 4 patients (51,52,53,54) in addition to those described here, but there is no report of this finding in any patient with collagenous gastritis without collagenous colitis. Probably a greater extent of mucosa involvement favours enteral protein loss. Although the mechanisms of enteric protein loss in patients with collagenous colitis have not been clarified, abnormalities of the surface epithelium, superficial capillaries and pericryptal fibroblasts have been hypothesized (51) and this enteral protein leakage could be an additional argument for the inflammatory aetiology of collagenous colitis (52). On the other hand, gastrointestinal protein loss has been suggested to be a marker of the severity of activity in inflammatory bowel disease (55). The enteral protein loss in these patients with extensive mucosal collagenous changes could indicate the presence of inflammatory changes responsible for the collagenous deposits.

The relationship of CG and CC with diseases thought to have an autoimmune pathogenesis is evident in these series. Seventeen (2,11-13,17,18,25-29,39,44,47) of sixty-six patients (26%) had associated diseases and these were more frequent in patients with CC and GC (35%) than in those who had a less extensive disease, 22% of the CG and 20% of the children with CC. Coeliac disease (5 patients (12,17,18,29,44)) and collagenous sprue (6 patients (25-27)) were the most frequent. Other diseases observed were autoimmune hypothyroidism, type I diabetes mellitus, autoimmune haemolytic anaemia, Plummer-Vinson syndrome, Biermer's gastritis, Juvenile Scleroderma, Crohn's disease and spinocerebellar degeneration with cerebellar ataxia.

Gastric nodularity, a frequent finding in collagenous gastritis (11 children, 7 adults) and observed in the girl described here, was only reported in 2 adult patients with CC and CG. Some patients showed mucosal involvement beyond the colon and stomach. A sub-epithelial collagen band in duodenum was observed in two of 30 patients with CG (10,25) where the information was available and in 13 of 19 patients with CG and CC (6,13,25,26,29,30,32,34,35,38,40) where the duodenum was investigated. These phenomena suggest a far more extensive disease process and that both collagenous gastritis and collagenous colitis diseases may share common pathogenic mechanisms. It is postulated that a generalized inflammatory response to a multitude of causes exists in these patients (25) and the concomitant autoimmune disorders that these patients present support the participation of genetic and immunologic factors in the pathogenesis of these disorders. Both collagenous gastritis and collagenous colitis are more frequent in women and they are associated with each other and with immunological disorders. This could indicate that they are really the same disease. It has also been said that collagenous sprue could be a related entity and be included in the same spectrum, in which collagenous deposition is a diffuse alteration that involves the entire gastrointestinal tract (2,3,37). The three of them have in common : histological definition, prevalence in women, association among themselves and with immunological disorders and, as the main difference, the area of the gastrointestinal tract affected. As a possible explanation to the last point, we suggest that the location of the affected area could be determined by the nature of the trigger. In a deregulated inflammatory context, the gluten could precipitate the disease in the duodenum as a final stage in predisposed patients, NSAIDs (56-58) and certain infections such as *Aeromonas hydrophila* in colon (2), and other toxic or infectious agents could produce the damage in the stomach. The extension of the injury may be related with the severity of the disease.

Protocols regarding therapeutical management for collagenous gastritis are not established : steroids, proton pump inhibitors, sucralphate and others have all been used with limited results. Steroids (e.g ; budesonide) remain the best-documented treatment for collagenous colitis (49,59). The natural course of these patients remains unclear although most of them seem to have a persistent chronic disease. An interesting point is that clinical improvement may be achieved despite the persistent abnormalities in the biopsies, as has occurred in some patients (7,9,12,14,18,20,22,32,33,36). In 15 patients with CG, gastric biopsies were obtained at follow-up (3 months to 12 years) and SCB persisted in all of them. In one of these patients, Winslow *et al*. (4) examined 109 biopsy specimens of gastric mucosa from 19 different endoscopic procedures during a 12-year period. Specimens showed a significantly lower number of antral gastrin cells, along with a significant corpus endocrine cell hyperplasia, suggesting an increased risk of endocrine neoplasia. Gastric corpus biopsy specimens revealed an active, chronic gastritis, subepithelial collagen deposition, smooth muscle hyperplasia, and mild to

Table 1. — **Cases of collagenous gastritis and collagenous colitis in children**

Age (yrs)/ sex	Duration	Presentation	Associated diseases	Endoscopy UGI and colonoscopy	Other histopathologic findings	Treatment	Outcome	Ref.
9/M	9 mo	Watery diarrhoea vomiting since birth. Hypoalbuminemia hypogamma-globulinemia lymphocytosis	Celiac disease	Gastric erosions. Colon: NA 3yrs, 10 yrs: Stomach: Pseudopolyps diffuse atrophic area Colon: erythema nodules	SCD villous atrophy in D Diffuse atrophic gastric mucosa increase in the SCB in the gastrointestinal tract.	Prednisone, budesonide, night enteral nutrition, gluten free diet (13 yrs) 14 yr: Total parenteral nutrition	Clinicopathological disease progression Complete clinical improvement	29
2/M	2 mo	Secretory diarrhoea Emesis intermittent fevers Hypoalbuminemia	Spinocerebellar degeneration with cerebellar ataxia	Stomach: erythema petechiae Colon: Increased vascularity diffuse oedema erythema	SCB in D	Steroids, proton pump inhibitor mesalamine. Bismuth subsalicylate	Clinical improvement. Relapse after finishing steroids Resolution of symptoms	30
15/M	3 mo	Diarrhoea abdominal pain, intermittent fever weight loss fatigue	Autoimmune haemolytic anaemia	Stomach: patchy erythema. Colon: normal.	SCB in D	Prednisone, Lansoprazole mesalamine.	Clinical improvement (steroids dependent) Resolution of symptoms One year later, asymptomatic on therapy	13
15/F	14 yrs	Watery diarrhoea Aeromonas hydrophila in faeces	Congenital autoimmune hypothyroidism Type I Diabetes mellitus	Normal	Normal D	Antibiotics, loperamide	Unchanged	2
1,5/ M	3 mo	Weight loss, abdominal pain GI protein loss?		NA	NA	Elemental diet	Improvement	31
2,25/ F	3 mo	Watery diarrhoea. Aeromonas hydrophila in feces 9 years: abdominal pain Ferropenia	Congenital cardiac defect	Normal stomach Normal duodenum Normal colon Gastric Nodularity Normal colon	Collagenous colitis Lymphocytic colitis Gastric biopsies not taken. Normal D SCB in stomach and colon. Normal D	Antibiotics, colloidal bismuth subcitrate Budesonide for 6 months <u>Mesalazine</u>	Temporary clinical improvement Complete clinical improvement One month after discontinuation diarrhoea reappeared <u>Unchanged</u>	This case

UGI :Upper gastrointestinal . NA :not available. SCB : subepithelial collagen deposition.. D : Duodenum.

moderate glandular atrophy. Additional findings of intestinal metaplasia and reactive epithelial changes indeterminate for dysplasia raise concern about the potential for adenocarcinoma. In 8 adult cases (6,32,33,36,37,39,40) and in 1 child (29) with CG and CC, biopsies were obtained during the follow-up (1 month to 3 years). Two patients (6,40) showed normal gastric and colonic biopsies two years later. Both of them received treatment with steroids and parenteral nutrition. In another case (39) SCB had disappeared in the stomach and was decreased in the colon after 4 months of treatment with budesonide. In the remaining patients, SCB remained unchanged. Although concern for the eventual development of adenocarcinoma was raised in CG, gastric epithelial or lymphoid malignancies have not been reported to date and the overall colorectal cancer risk is similar to the general population in patients with CC50.

In summary, we describe the case of a 9-year old girl with collagenous gastritis and collagenous colitis. As available data on these rare entities, especially about their association, rely mainly on case reports, we have performed an exhaustive search in the literature in order to obtain reliable and useful clinical information about them. So far, 56 patients (33 children) have been report-

Table 2. — **Cases of collagenous gastritis and collagenous colitis in adults**

Age / sex	Duration	Presentation	Associated diseases	Endoscopy Stomach/colon/ others	Other histopathological findings	Treatment	Outcome	Ref.
54/F	3 mo	Watery Diarrhoea weight loss Protein losing enteropathy		S,D,C and ileum : normal	SCB in D and Ileum	Budesonide	Resolution of symptoms Three months later : SCB persisted in C,S disappeared in D and ileum	**32**
72/M	NA	Weight loss diarrhoea Hypoalbuminemia	Collagenous sprue	NA	SCB and Subtotal villous atrophy in D	Steroids	Clinical improvement	**25**
9/F	NA	Intractable chronic diarrhoea Weight loss	Collagenous sprue	NA	SCB and partial villous atrophy in D	NA	NA	**25**
80/M	NA	Chronic diarrhoea Anaemia		Gastric nodularitiy Colon : normal	D : normal	Budesonide	NA	**14**
74/F	3 mo	Diarrhoea abdominal pain		Stomach : loss of vascular pattern Colon : normal	D : normal	Omeprazole	Symptoms resolved SCB in D and C persisted after treatment (1 month).	**33**
67/F	2 yrs	Diarrhoea.		Normal	SCB in D	Loperamide	No response One year later : SCB persisted	**34**
75/F	6 mo	Diarrhoea Loss of weight		Normal	SCB in duodenal bulb	NA	NA	**35**
57/F	6 mo	Watery diarrhoea Weight loss Anorexia Protein losing enteropathy	Nonspecific articular pains	Erythematous and atrophic gastric mucosa Bulbar erythema Colon : normal	SCB in duodenal bulb	Prednisone	Resolution of symptoms (steroids dependent) Six months later : SCB persisted in C	**36**
40/F	NA	Diarrhoea		Normal	SCB and subtotal villous atrophy and increase of IELs in D	Prednisolone Salazopyrine complete parenteral alimentation	Two years later : normal gastric and colonic biopsies	**6**
20/M	2 yrs	Epigastric pain, vomiting fatigability Severe anaemia 6 yr : diarrhoea weight loss		Diffuse gastric nodularity Colon : normal	SCB and foci of atrophy in stomach Colonic biopsies not taken 6 yr : SCB in C. D : normal	H2 receptors blockers antihelminthics oral iron NA	No clinical improvement Three years later SCB persisted NA	**37**
25/F	2 yrs	Anaemia	Collagenous sprue Pernicious anaemia	NA	D : SCB and marked inflammation in lamina propia	Gluten-free diet	After 3 and 9 years continues to be asymptomatic.	**26**
51/F	1,5 mo	Watery diarrhoea weight loss emesis Hypoalbuminemia	Collagenous sprue Bacterial overgrowth syndrome	Normal	SCB in D	Steroids Azathioprine	Clinical improvement with azathioprine	**38**
73/F	4 mo	Diarrhoea abdominal pain weight loss Hypoalbuminemia hypokalemia	Hashimoto's thyroiditis Biermer's gastritis	Normal S and C	Atrophic gastritis D : normal. SCB in ileum.	Budesonide	After 4 months : Clinical improvement disappearance of SCB in S. CC persisted	**39**
41/F	1,5 mo	Watery diarrhoea abdominal pain vomiting Protein losing enteropathy		S,D,C and ileum : normal	SCB in D	Parenteral nutrition Gluten free diet Prednisolone Salazopyrine	After 2 years : normal biopsies of S, D, C Five years follow up : asymptomatic	**40**

NA : not available. S : stomach. D : duodenum. C : colon. SCB : subepithelial collagen band.

ed with collagenous gastritis and 16 children with collagenous colitis. In 21 cases, both diseases coexisted. Most patients were female. Clinical presentation is similar in children and adults. Diarrhoea and important weight loss are the main symptoms in patients with collagenous colitis and collagenous gastritis. Abdominal pain and anaemia are presenting symptoms in a large proportion of patients with collagenous gastritis. The association of both conditions must be borne in mind in cases of protein losing enteropathy. A subepithelial collagen band is not always limited to a single location, so when detected in an area, its presence should be investigated in other possible locations. Concomitant autoimmune disorders are often seen especially in patients with both diseases. Specific therapy has not been established but it seems that budesonide may be useful in patients with collagenous colitis. The natural history of both conditions is still unknown. The normalization of the mucosa of the affected areas is rare and both entities seem to follow a chronic course. The development of malignant diseases has not been described in these patients.

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