EJCRIM
European Journal of Case Reports in Internal Medicine

PAEDIATRIC COLLAGENOUS GASTRITIS WITH TRANSIENT RESPONSE TO STEROID THERAPY

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Received: 08/04/2026
Accepted: 18/04/2026
Published: 04/08/2026

Conflicts of Interests: The Authors declare that there are no competing interests.
Patient Consent: Full information on publication was provided to the patient and next of kin (parent) with written consent obtained from the patient's parent.
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How to cite this article: O'Connor R, Standish R, Karunadasa H, Chan R, Hodge A. Paediatric collagenous gastritis with transient response to steroid therapy. *EJCRIM* 2026;**13**:doi:10.12890/2026_006709

ABSTRACT

Introduction: Collagenous gastritis is a rare condition characterised by subepithelial collagen deposition and an inflammatory infiltrate. Despite being first described in 1989, the pathogenesis remains poorly understood. Clinical manifestation and histological features have been extensively described in the literature; however, treatment strategies are largely based on expert opinion and show variable efficacy.

Case description: We describe a case of paediatric collagenous gastritis presenting with dyspepsia, nausea and iron deficiency anaemia initially attributed to menorrhagia. Endoscopy demonstrated nodular gastritis and histology confirmed the diagnosis. The patient had initial symptomatic improvement with budesonide; however, this was not sustained.

Conclusion: This case highlights the importance of considering collagenous gastritis as a cause of iron deficiency anaemia, particularly in young female patients. It also demonstrates the management challenges in achieving sustained clinical response, reflecting the limited evidence base guiding management.

KEYWORDS

Collagenous gastritis, paediatric, iron deficiency anaemia

LEARNING POINTS

- Collagenous gastritis is a rare but important cause of iron deficiency anaemia, particularly in young patients with persistent gastrointestinal symptoms.
- Alternative causes of iron deficiency beyond menstruation should be considered in young female patients, with early consideration for endoscopy.
- Symptomatic improvement with corticosteroids may be transient and sustained clinical or histological remission remains difficult to achieve.

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INTRODUCTION

Collagenous gastritis is a rare disease characterised by subepithelial collagen deposition greater than 10 µm in thickness. It has been reported in approximately 13 per 100,000 upper gastrointestinal endoscopies[1]. Distinct clinical phenotypes have been described according to the age at presentation[2]. The pathogenesis remains unclear, but it is considered to represent part of a spectrum of collagenous gastrointestinal disorders, including collagenous sprue and collagenous colitis. Management is often challenging and largely based on case reports and small retrospective cohort studies. We describe a case of paediatric onset collagenous gastritis presenting with symptoms of upper gastrointestinal disease.

CASE DESCRIPTION

A 15-year-old female was referred to an outpatient gastroenterology service with a seven-month history of dyspepsia and nausea, as well as symptomatic iron deficiency anaemia. The patient had presented to her general practitioner with fatigue and dyspepsia and was found to have iron deficiency anaemia, which was presumed to be secondary to menorrhagia. She was started on an oral contraceptive pill and received one iron infusion. She continued to experience daily dyspepsia and nausea, which was exacerbated by spicy, oily or sugary food, resulting in a restricted diet of plain vegetables and carbohydrates such as rice. She was prescribed 40 mg of daily pantoprazole by her GP, with a modest improvement of dyspepsia after three months. The patient reported significant disruption to her daily life and ability to attend school. Notably, there was no significant weight loss.

The patient's only other medical condition was a recent diagnosis of supraventricular tachycardia which was well controlled with 75 mg of daily atenolol and managed by her cardiologist. Her only other medication was melatonin 2–4 mg every night. She had no allergies and had a family history of an aunt with coeliac disease.

The clinical examination was normal, and the patient's abdomen was soft and non-tender. Laboratory investigations revealed normal full blood count, renal function, liver function, thyroid function, lactate dehydrogenase and C-reactive protein (<0.7 mg/l). Her iron stores were replete with an iron of 23.6 µmol/l (10.0–30.0 µmol/l), transferrin of 2.72 g/l (2.10–3.80 g/l, transferrin saturation of 35% (15–45%) and a ferritin of 82 µg/l (20–140 µg/l). Folate levels were normal but total B12 levels were low at 171 pmol/l (200–700 pmol/l) and holo-transcobalamin was 53 pmol/l (>37 pmol/l). Coeliac serology did not demonstrate elevated titres, although the patient had an HLA-DQA1*05 genotype.

Initial upper gastrointestinal endoscopy showed mild nodular gastritis involving the gastric body, with a normal oesophagus and duodenum *(Fig. 1)*.

The biopsies of specialised gastric mucosa from the first gastroscopy showed patchy irregular thickening of the subepithelial collagen band – confirmed on trichrome staining – along with a mild to moderate inactive chronic inflammatory cell infiltrate, and some features suggesting atrophy *(Fig. 2 and 3)*. The antral mucosa was relatively spared, with reflux/chemical gastropathy-like changes. No intestinal metaplasia or Helicobacter organism was identified. Biopsies taken at subsequent endoscopies showed similar changes – most prominent in the gastric specialised mucosa, with persistent subepithelial collagen, inflammation and atrophy 18 months after diagnosis. No abnormalities were seen in the small bowel (duodenal or ileal), or large bowel mucosa; a diagnosis of collagenous gastritis was made.

A prolonged course of 9 mg daily of topically targeted budesonide was prescribed for 1 year and 7 months. It was administered with the open capsule method and was mixed with yogurt or fruit to increase contact with gastric mucosa on an empty stomach. The patient was commenced on regular subcutaneous B12 injections. Pantoprazole 40 mg daily was re-prescribed for dyspepsia symptoms 15 months after diagnosis, and a second iron infusion was administered 16 months after diagnosis.

In the first six months of budesonide treatment, the patient had symptomatic improvement in dyspepsia and nausea, allowing her to return to school and eat a wider variety of foods. There were no adverse effects of budesonide. In the subsequent 18 months she experienced a reduction in general health due to a number of symptoms that varied in intensity. Significant fatigue was attributed to a new diagnosis of chronic fatigue syndrome. Pantoprazole had a modest improvement in dyspepsia. Prochlorperazine followed by ondansetron were trialled for significant nausea but did not have lasting efficacy. Iron infusions were effective in maintaining replete iron stores and a normal haemoglobin; B12 levels responded appropriately to subcutaneous replacement.

There was no significant endoscopic or histological improvement in the subsequent three upper gastrointestinal endoscopies, and colonoscopy demonstrated no colonic involvement. Budesonide was

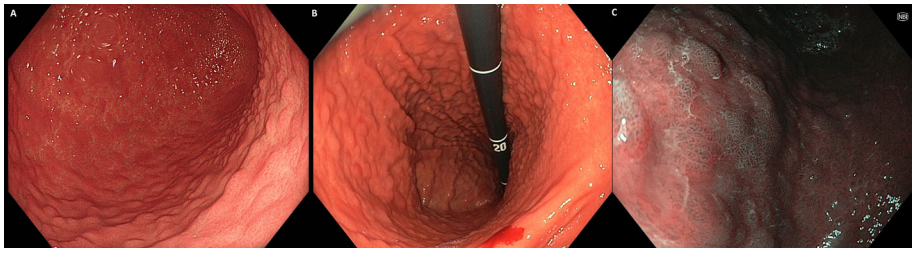


Figure 1. *Upper gastrointestinal endoscopy findings. Diffuse nodularity of gastric body (A, B). Narrow-band imaging revealing absent or amorphous pit patterns in depressed mucosa (C).*

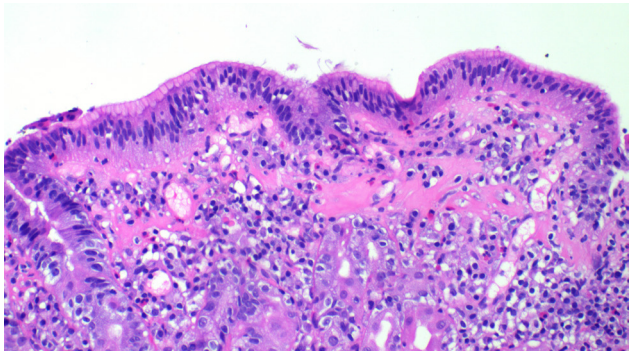


Figure 2. Irregular thickening of the subepithelial collagen band, with embedded capillaries and inflammatory cells, and inactive chronic atrophic gastritis (haematoxylin and eosin, ×20 objective).

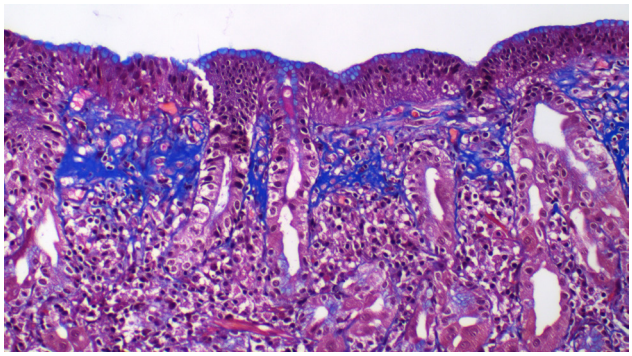


Figure 3. Irregular thickening of the subepithelial collagen band, with inactive chronic atrophic gastritis (Masson trichrome, ×20 objective).

ceased after 19 months due to the lack of persistent improvement.

DISCUSSION

Collagenous gastritis is characterised by subepithelial deposition of collagen and an inflammatory infiltrate in the gastric mucosa. Paediatric patients typically present with symptomatic iron deficiency anaemia and abdominal pain, and the disease is usually limited to the stomach. In contrast, adult patients commonly have more extensive disease with additional involvement of the small intestine or colon, presenting with diarrhoea and weight loss[2].

Endoscopic findings are variable, with the majority of patients demonstrating gastric nodularity[3]. Less common findings include mucosal erythema, erosion and, rarely, haemorrhage[4]. Diagnosis is confirmed histologically by the presence of variably thickened subepithelial collagen[3]. Within the stomach, distribution of disease typically favours the body or fundus in paediatric patients, and the antrum in adults[5].

The pathogenesis of collagenous gastritis remains poorly understood. Multiple theories have been proposed, including autoimmune, medication and infectious triggers[6,7]. The most popular theory suggests that the collagen formation is a response to mucosal injury[5].

Management remains challenging due to limited understanding of disease pathophysiology and the low quality of evidence base regarding treatment options. Symptomatic treatment with proton pump inhibitors or H2 receptor antagonists along with replacement of nutritional deficiencies remain the cornerstone of management[3]. Steroids, including prednisolone and budesonide, have been used with varying efficacy. More recently, interest in vedolizumab has shown benefit in one case study and has prompted investigation with a clinical trial in an early phase[8,9]. Our patient achieved replete iron and B12 levels but remained symptomatically burdened, with significant impact on daily function.

CONCLUSION

Although an initial symptomatic response to budesonide was observed, this was not sustained and lacked histological improvement. This case demonstrates the typical clinical and histological manifestations of paediatric collagenous gastritis and highlights the management challenges often encountered. Further investigation with prospective studies and a review of therapeutic options are needed to establish effective evidence-based treatment strategies.

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