# Oral manifestations of gastrointestinal diseases in children. Part 3: Ulcerative colitis and gastro-oesophageal reflux disease

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**ABSTRACT**

*Alterations of the oral cavity are common in children: 22% of children aged less than 4 years and 44% of those aged more than 12 develop dental erosion, 9-95% of children in Europe and in North America develop gingivitis, with adolescents showing a prevalence of more than 60% [Italian Ministry of Health, Guidelines 2013]. Alterations within the oral cavity can be the first sign of systemic diseases and may thus allow for an early diagnosis and treatment. In particular, being the oral cavity a part of the gastrointestinal system, oral alterations can be an expression of a gastrointestinal disease. Pyostomatitis vegetans can be a sign of ulcerative colitis. Dental erosion with enamel loss in facial, occlusal, and lingual surfaces, and an increased risk of dental caries have been reported in children and adolescents with gastro-oesophageal reflux with varying prevalence. A prompt recognition of systemic diseases through a careful examination of the oral cavity could allow proper investigations and management in a timely fashion.*

***Keywords*** *Alterations of the oral cavity; Children; Gastrointestinal diseases; Gastro-oesophageal reflux disease; Ulcerative colitis.*

## Ulcerative colitis

Ulcerative colitis (UC) is the most common type of inflammatory disease of the bowel, whose peak incidence is between the ages of 15 and 25 years, with a second, smaller peak between 55 and 65 years. In the United Kingdom incidence of ulcerative colitis in children aged less than 16 years has been reported to be 1.4 per 100,000 with Asian children mostly involved and a median age for diagnosis of 11.7 years [Sawczenko, 2001]. The cause of the disease is still unknown [Loftus, 2004]. Diagnosis of UC relies on the presence of bloody diarrhoea with negative stool cultures and evidence of diffuse continuous mucosal inflammation involving the rectum and extending to a point more proximal in the colon at the endoscopic evaluation [Kugathasan, 2003; Bentsen, 2002] (Fig. 1).

UC share some clinical manifestations with Crohn's disease (CrD), and all the non-specific oral manifestations seen in the latter, may also occur in the former but less commonly except for pyostomatitis vegetans, that is more frequent in UC [Ficarra, 1993; Litsas, 2011]. Oral involvement in paediatric patients with UC may be present in up to one-third of the patients and it is usually non-specific [Katsanos, 2015].

Pyostomatitis vegetans is characterised by multiple miliary white or yellow pustules that can coalesce into "snail-track" ulcers, with an erythematous and edematous mucosal base and involve mostly the labial gingiva and the labial and buccal mucosa. A combination of clinical features of inflammatory bowel disease, peripheral eosinophilia, histological findings,

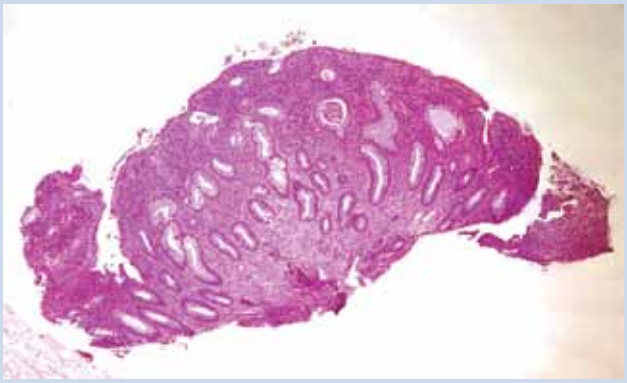

FIG. 1 Histological image of ulcerative colitis: architectural, epithelial and inflammatory alteration of the colorectal mucosa with cryptitis and chronic inflammation of the lamina propria 100X.

and negative culture of the exudate allow diagnosis of pyostomatitis vegetans [Mijandrusić-Sincić, 2010]. Average age at diagnosis occurs usually around 34 years, no reports of pyostomatit vegetans and ulcerative colitis in children can be found in the literature.

Other non-specific oral manifestations in UC are oral aphthae, seen in at least 5% to 10% of the patients, glossitis, cheilitis, stomatitis, lichen planus, mucosal ulcers, diffuse pustules, and non-specific gingivitis [Folashade, 2008; Leković, 2011; Krebs, 2011] (Fig. 2, 3)

Treatment of the underlying disease allows the management of oral alterations in UC; oral steroids are considered the treatment of choice [Thrash, 2013].

## Gastro-oesophageal reflux disease

Gastro-oesophageal reflux is a physiologic event which allows movement of gastric content into the esophagus and oropharyns through relaxation of the lower oesophageal sphincter; gastroesophageal reflux becomes pathologic if symptoms or complications are present, in which case the term GERD is used [Colletti, 2003]. Repeated regurgitation, nausea, heartburn, coughing, laryngitis, asthma, or pneumonia can be signs of GERD. Infants and young children may demonstrate irritability or arching of the back while feeding, symptoms that might lead to refusal of feeds and therefore poor growth [Alfaro, 2008]. Oesophagitis, hemorrhage, stricture, Barrett's oesophagus, and adenocarcinoma can be complications due to GERD [Vakil, 2006] .

Oral manifestations have also been described in GERD, in particular the 2009 NASPGHAN and ESPGHAN guidelines on reflux in children report evidence of correlation between dental erosions, defined as the loss of tooth substance by a chemical process that does not involve bacteria, and GERD [Vandenplas, 2009; Pindborg, 1970]. Prevalence of dental erosions in children with GERD is quite variable: while Dahashan et al. [2010] demonstrated a 83,3% prevalence of dental erosions in their enrolled children suffering from GERD, O'Sullivan et al. [1998] reported just a 17% prevalence of perimylolysis in their cohort of children between 2 and 16 years of age with moderate to severe GERD [O'Sullivan, 1998]. Perimylolysis may be due to extrinsic and intrinsic factors such as exposure to acidic foods, beverages, or contaminants, salivary flow, eating disorders, and acid regurgitation [Gudmundsson, 1995; Dahshan, 2010]. The pattern of dental erosion in GERD involves initially the posterior teeth in particular the facial, occlusal, and lingual surfaces and mainly the mixed dentition [Dahshan, 2010].

GERD may also lead to changes of the soft oral tissues and salivary flow [Silva, 2001]. Di Fede et al. [2008] demonstrated that oral acid/burning sensation, xerostomia, subjective halitosis and soft/hard palate and uvula mucosal erythema are significantly associated with GERD in the adult population, conversely they found that only 9% of patients with GERD presented dental erosions, compared to 13% of healthy subjects. Moshkowitz et al. [2007] found a strong association between the occurrence and severity of halitosis and GERD in adults. No data on these last oral cavity alterations are available in children.

Inspection of the oral cavity in search for oral alterations is therefore advisable in patients with known GERD. Following the 2009 NASPGHAN and ESPGHAN guidelines on reflux, those children aged more than 8 years with heartburn can be "trialed" with a proton pump inhibitor for 2-4 weeks time; infants and children aged less than 8 years, with atypical symptoms or with possible complications need further investigations such as pHimpedence or endoscopy [NASPGHAN ESPGHAN Guidelines, 2009]. Lifestyle changes such as a regular diet, weight loss, smoking avoidance, correct sleeping

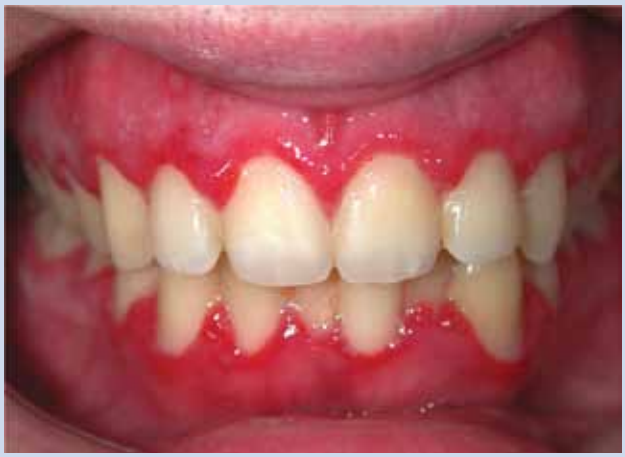

FIG. 2 Gingivitis.

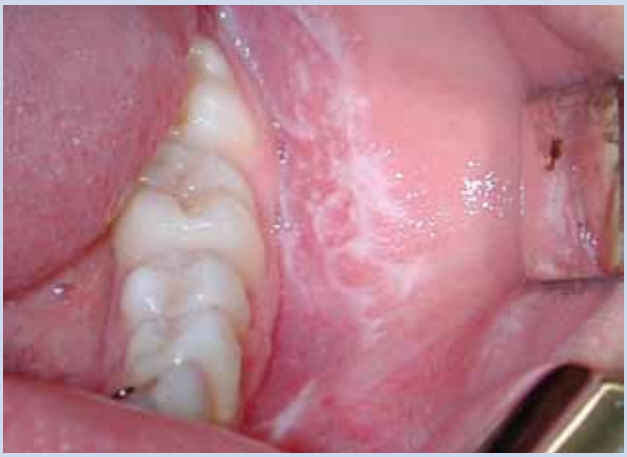

FIG. 3 Reticularis oral lichen planus of the left cheek.

position, no late night eating, and acid suppression are the main management options for GERD.

The use of proton pump inhibitors has been demonstrated to be effective in suppressing tooth erosion in GERD [Wilder, 2009].

## References


› Alfaro EV, Aps JK, Martens LC. Oral implications in children with gastroesophageal reflux disease. Curr Opin Pediatr 2008;20:576-583.
› Bentsen BS, Moum B, Ekbom A. Incidence of inflammatory bowel disease in children in southeastern Norway: a prospective popula- tion-based study 1990 – 94. Scand J Gastroenterol 2002; 37:540-5.
› Colletti RB, Di Lorenzo C. Overview of pediatric gastroesophageal reflux disease and proton pump inhibitor therapy. J Pediatr Gastroenterol Nutr 2003;37 Suppl 1:S7-S11.
› Dahshan A, Patel H, Delaney J, Wuerth A, Thomas R, Tolia V. Gastroesophageal reflux disease and dental erosion in children. J Pediatr. 2002 Apr;140(4):474-8.
› Di Fede O, Di Liberto C, Occhipinti G, Vigneri S, Lo Russo F, Fedele S, Lo Muzio S, Campisi G. Oral manifestations in patients with gastro-oesophageal reflux disease: a single-center case–control study. J Oral Pathol Med 2008;37: 336-340.
› Ficarra G, Cicchi P, Amorosi A, et al. Oral Crohn's disease and pyostomatitis vegetans. An unusual association. Oral Surg Oral Med Oral Pathol 1993; 75:220-4.
› Folashade AJ, Melvin B. Heymant extraintestinal manifestations of inflammatory bowel disease. J Pediatr Gastroenterol Nutr 2008 Feb; 46(2): 124-133.
› Gudmundsson K, Kristleifsson G, Theodors A, Holbrook WP. Tooth erosion, gastroesophageal reflux, and salivary buffer capacity. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1995;79:185-9.
› Italian Ministry of Health, Guidelines. Linee guida nazionali per la promozione della salute orale e la prevenzione delle patologie orali in età evolutiva, 2014 Ministero del lavoro, della Salute e delle Politiche Sociali. 2013.
› Katsanos KH, Torres J, Roda G, Brygo† A, Delaporte‡ E, Colombel J-F. Review article: non-malignant oral manifestations in inflammatory bowel diseases. Aliment Pharmacol Ther 2015; 42: 40–60.
› Krebs KT. Case study: aphthous ulcers in a 14-year-old girl. Pediatr Nurs 2011 May-Jun;37(3):115-8.
› Kugathasan S, Judd RH, Hoffmann RG et al. Epidemiologic and clinical characteristics of children with newly diagnosed inflammatory bowel disease in Wisconsin: a statewide population-based study. J Pediatr 2003;143:525-31.
› Leković Z, Radlović N, Brdar R, Vuletić B, Janić N, Ristić D, Stojsić Z, Radlović V, Simić D, Nikolić D. Clinical characteristics of idiopathic ulcerative colitis in children. Srp Arh Celok Lek 2011 Mar-Apr;139(3-4):170-3.
› Litsas G, AriDemirkaya A. Crohn's disease of the mouth: report of a case. Eur J Paediatric Dent 2011; 12 (1): 198-200.
› Loftus EV Jr. Clinical epidemiology of inflammatory bowel disease: Incidence, prevalence, and environmental influences. Gastroenterology 2004; 126(6):1504-1517.
› Mijandrusić-Sincić B, Licul V, Gorup L, Brncić N, Glazar I, Lucin K. Pyostomatitis vegetans associated with inflammatory bowel disease-report of two cases. Coll Antropol 2010; 34 Suppl 2:279-82
› Moshkowitz M, Horowitz N, Leshno M, Halpern Z. Halitosis and gastroesophageal reflux disease: a possible association. Oral Dis 2007; 13:581–585.
› O'Sullivan EA, Curzon MEJ, Roberts GJ, Milla PJ, Stringer MD. Gastroesophageal reflux in children and its relationship to erosion of primary and permanent teeth. Eur J Oral Sci 1998;106:765-9.
› Pindborg JJ. Chemical and physical injuries. In: Pindborg JJ ed. Pathology of the dental hard tissues. Philadelphia: Saunders; 1970. p 312-325.
› Sawczenko A, Sandhu BK, Logan RF, Jenkins H, Taylor CJ, Mian S et al. Prospective survey of childhood inflammatory bowel disease in the British Isles. Lancet 2001; 357(9262):1093-1094.
› Silva MA, Damante JH, Stipp AC, Tolentino MM, Carlotto PR, Fleury RN. Gastroesophageal reflux disease: new oral findings. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001; 91: 301–10.
› Thrash B, Patel M, Shah KR, Boland CR, Menter A. Cutaneous manifestations of gastrointestinal disease: part II. J Am Acad Dermatol 2013; 68: 211.e1-33; quiz 244-246.
› Vakil N, van Zanten SV, Kahrilas P, Dent J, Jones R. Global Consensus Group. The Montreal definition and classification of gastroesophageal reflux disease: a global evidence-based consensus. Am J Gastroenterol 2006;101:1900-1920.
› Vandenplas Y, Colin DR. Pediatric Gastroesophageal Reflux Clinical Practice Guidelines: Joint Recommendations of the North American Society for Pediatric Gastroenterology, Hepatology, and Nutrition (NASPGHAN) and the European Society for Pediatric Gastroenterology, Hepatology, and Nutrition (ESPGHAN). Committee Members: Di Lorenzo C, Hassall E, Liptak G, Mazur L, Sondheimer J, Staiano A, Thomson M, Veereman-Wauters G, Wenzl TG. J Pediatric Gastroenterology Nutrition 2009; 49:498-547.
› Wilder-Smith CH, Wilder-Smith P, Kawakami-Wong H, Voronets J, Osann K, Lussi A. Quantification of dental erosions in patients with GERD using optical coherence tomography before and after double-blind, randomized treatment with esomeprazole or placebo. Am J Gastroenterol 2009;104:2788-2795.