A 34-year-old man was admitted to hospital complaining of epigastric pain and chronic diarrhea without blood or mucus. He had no weight loss. His hematologic, biochemical, and serologic tests and stool examination showed normal results, but his levels of immunoglobulin A (IgA) were under the lower limit. Endoscopic examination revealed multiple millimetric polypoid lesions throughout the duodenum and terminal ileum (Fig. 1). Histologic examination showed findings of chronic *Helicobacter pylori* gastritis and lymphoid follicular hyperplasia in the small bowel. A small-bowel series revealed rapid small-bowel transit and numerous small nodular filling defects throughout the small bowel (Fig. 2). The patient was treated for *H. pylori* gastritis with a regimen containing levofloxacin, a proton pump inhibitor, and amoxicillin for 14 days. At 2 months after the end of the eradication therapy, C14 urea breath test gave a negative result. At follow-up endoscopy, the duodenal nodules had regressed (Fig. 3). The patient was referred to an immunologist for further investigation.

Nodular lymphoid hyperplasia (NLH) of the gastrointestinal tract is a rare pathology in adults. It is characterized by the presence of numerous visible mucosal nodules measuring up to 5 mm in diameter. Histologically, hyperplastic lymphoid follicles with large germinal centers are seen in the lamina propria and superficial submucosa. The etiology is unknown. Viral agents, giardiasis, and common variable immunodeficiency have been suggested to be associated with NLH [1]. The association of NLH with *H. pylori* and IgA deficiency has been rarely described in the literature [2, 3]. Treatment of the *H. pylori* infection can provide regression of nodules. However, treatment of all infections is very difficult, and infections tend to be more persistent in patients with immune deficiencies. Endoscopists should consider the possibility of IgA deficiency in patients who present with diffuse nodular lesions in the small bowel, and *H. pylori* infection should be tested for and, if found, treated with an appropriate drug regimen.

**Competing interests:** None
References

Bibliography
DOI http://dx.doi.org/10.1055/s-0034-1377938
Endoscopy 2014; 46: E568–E569
© Georg Thieme Verlag KG Stuttgart · New York
ISSN 0013-726X

Corresponding author
Sebahat Basyigit, MD
Department of Gastroenterology
Kecioren Research and Training Hospital
Pınarbaşı Mah. Sanatoryum Cad.
Ardahan Sok. No: 25
06380 Keciören
Ankara
Turkey
Fax: +90-312-3569003
sbuyuktemiz@yahoo.com