Eosinophilic gastroenteritis is a rare disorder characterized by eosinophilic infiltration of mainly the stomach and small intestine. Primary EG is defined as a disorder of unknown etiology with eosinophilia and is strongly associated with concomitant atopic diseases, food allergies and family history of allergies in about 25-75% of cases (1). Secondary EG is reported to occur in the gastrointestinal tract in parasitic and bacterial infections, irritable bowel disease (IBD), hypereosinophilia syndrome, autoimmune diseases, celiac disease, connective tissue diseases, vasculitis, some neoplasms, after solid organ transplantations, or due to adverse effects of certain drugs (mesalazine, azathioprine, gemfibrozil, enalapril, carbamazepine, clofazimine, cotrimoxazole) (1). IBD very rarely results in EG.

Eosinophils are found normally in low levels in the stomach and they are located normally in the lamina propria (1, 2). Eosinophilic infiltration in Peyer's patches, intra epithelial area, superficial mucosa, or intestinal crypt regions results in eosinophilic gastrointestinal disorders. There are no specific symptoms or laboratory tests for diagnosing EG. It should be suspected in a patient that has a course of relapses and remissions of gastrointestinal symptoms. Peripheral eosinophilia, seen in two-thirds of cases in EG, may help but it is not necessary for the diagnosis of EG (1). On endoscopic examination, micronodules and/or polyposis containing lymphocytes and eosinophils can be seen in patients with EG (3). Demonstration of increased eosinophilic infiltration in the histopathologic examination of endoscopic specimens from the gastrointestinal tract is the gold standard for diagnosis (4). However, the patchy involvement of the gastrointestinal tract and sparing of the mucosa in muscular and serosal subtypes of EG complicate the taking of endoscopic biopsy. Thus, full thickness and multiple endoscopic biopsies are necessary for diagnosis. Elimination diet and anti-inflammatory and immunosuppressive drugs are therapy modalities that must be chosen according to the disease severity and relapsing course.

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An unusual etiology for adult intussusception: post-vagotomy jejunojejunal invagination

Erişkin intussepsiyon için nadir bir neden: Vagotomi sonrası jejuno-jejunal invajinasyon

To the Editor,

Invagination can be described as the telescoping of a proximal segment of the gastrointestinal tract and its associated mesentery (intussusceptum) into the lumen of the adjacent distal segment (intussusceptions) (1). The exact mechanism that precipitates intussusception is still unknown. In chil-

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dren, reasons for obstruction are almost invariably idiopathic. In contrast to its pediatric counterpart, in adults, it can lead to an identifiable lesion, and an etiologic reason is found in the majority of cases (2). In this present study, we report two cases of jejunojejunal intussusception, with no demonstrated etiologic reason except for truncal vagotomy.

The cases, male patients aged 26 and 28, presented with complaints of colic abdominal pain, nausea and vomiting. The first patient underwent an operation three months before due to peptic ulcer bleeding, and primary suture, truncal vagotomy, Heineke-Mikulicz pyloroplasty, and gastroduodenal artery ligation were performed. The second case underwent a truncal vagotomy and Jaboulay-type pyloroplasty operation 45 days before due to pyloric stenosis. Both cases were operated on an emergency basis via prediagnosis of intestinal obstruction-ileus following the physical examination and imaging scans. Jejunojejunal invagination was determined in both cases. One of the cases underwent an intestinal resection of 30 cm with double-barreled enterostomy (since the intestines were edematous in advanced level and dilated), and the other case underwent only reduction. No adhesion in the intestines or suture line or any intraluminal pathology that may cause invagination was determined during the operation. There was no postoperative complication.

Intussusception is an uncommon condition in adults in comparison with the pediatric population. Related reports indicate malignant and benign etiologies for adult invagination cases. Idiopathic cases without apparent etiologic reasons are classified as other causes (2, 3). Approximately 10–20% of cases are classified in the idiopathic invagination group due to no apparent etiologic reason (4). For another invagination type, postoperative invagination, the most important predisposition factor is known to be disruption in intestinal peristalsis. Most commonly, it develops within one month after the initial operation. Prolonged postoperative ileus, large dissection and prolonged surgery and postoperative chemoradiotherapy are mentioned as the probable risk factors (2, 5). None of these predisposing factors was present in the two cases presented herein. No adhesion in the invagination area was determined. Contrary to the postoperative invaginations, the first patient had applied to our hospital three months after the operation, while the second patient presented on the 45th day with invagination symptoms. Thus, when etiologic cause in the aforementioned cases was examined, it was showing as a pathology other than postoperative invagination.

Vagotomy is known to elevate and alter the transit time of the small bowel. As already known, vagal stimulation has two effects on the small bowel: first, the cholinergic (exciting) effect and second, the peptidergic (inhibitory) effect (6). The cases in our report and the two cases of jejunojejunal invagination after truncal vagotomy presented by Youssef et al. (7) had the same characteristics.

We believe that the disruption in peristalsis and the elevated transit time after truncal vagotomy may have contributed to the risk of invagination. Therefore, the risk of entero-enteral invagination formation in truncal vagotomy-applied patients should be remembered.

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