

Prevalence, clinical manifestations, and treatment of esophageal inlet patch

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ABSTRACT

Objectives: This study aimed to evaluate the prevalence, symptoms, and treatment of inlet patch (IP) in light of the literature.

Materials and methods: Between July 2020 and January 2022, among the 1,926 patients, a total of 48 patients (21 males, 27 females; mean age: 41.1±14.2 years; range, 18 to 82 years) who underwent esophagogastroduodenoscopy were included in the study. After defining the IP, the size of the IP was measured using an endoscope shaft. Following measurement, biopsy specimens were taken and the type of gastric mucosa and the presence of *Helicobacter pylori* in IP were evaluated.

Results: The mean IP diameter was 8.96±5.8 mm (range, 3-30 mm), with a maximum of two patches in the same case. Fourteen (29.2%) of the patients with IP were symptomatic. Of the symptomatic patients, eight (57.1%) were male and six (42.9%) were female. The most common presenting symptom was dysphagia (18.8%). The biopsies taken from the IP mucosa revealed that the most frequently detected mucosal type was oxyntic and 12 (25%) patients were positive for *Helicobacter pylori*.

Conclusion: Patients presenting with dysphagia, globus sensation, unexplained chronic cough, and painful swallowing should be carefully examined for cervical esophageal IP. Sedating the patients and evaluating the esophagus with optical chromoendoscopy, if possible, will improve the detection rate of IPs. Proton pump inhibitors may be a good option for symptomatic patients.

Keywords: Dysphagia, inlet patch, optic chromoendoscopy, proton pump inhibitors.

Heterotopic gastric mucosa of the proximal esophagus also referred to as inlet patch (IP), is usually an oval or round, well-demarcated, salmon-pink mucosal area of variable size, typically located in the proximal esophagus. The size of the patch is generally less than 1 cm and rarely up to 5 cm. It is most commonly detected as an incidental finding during meticulous endoscopic evaluations of the cervical esophagus. The patch mostly appears as a smooth surface but may sometimes be seen as a slightly raised or depressed surface. The inlet may very rarely appear as a polypoid lesion. It can be single or

multiple, or it can be visualized to surround the lumen in a circular fashion.^[1-4] Schmidt described it about 200 years ago, and it can be detected in different parts of the gastrointestinal tract, including the rectum, anus, duodenum, jejunum, gallbladder, and the ampulla of Vater.^[2,5,6] Although in the literature suggest that IP is a metaplastic transformation, the common view is that it is a congenital anomaly, resulting from the incomplete epithelialization of the esophagus during embryologic development.^[7]

The prevalence of IP in the literature ranges from 0.18 to 14%.^[8] Small peptic erosions, ulcers, stenosis, and fistulas may very seldom develop in the IP area. Inlet patches are usually asymptomatic and sometimes can present with symptoms such as globus, dysphagia, chest pain, sore throat, and tickly cough.^[9-13] Very rarely, adenocarcinoma may arise in the IP mucosa.^[14]

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This study aimed to retrospectively evaluate the prevalence, clinical manifestations, and treatment

of proximal esophageal IP in the light of the literature.

MATERIALS AND METHODS

This retrospective single-center study was conducted at the gastroenterology clinic of KTO Karatay University Medicaana Affiliated Hospital between July 2020 and January 2022. A total of 48 patients (21 males, 27 females; mean age: 41.1 ± 14.2 years; range, 18 to 82 years) who underwent esophagogastroduodenoscopy for any reason among the 1,926 patients were included in the study.

Biopsy specimens and measurements

The procedure was performed by a single gastroenterologist using a Fujifilm EG-760R video gastroscope (Fujifilm Medical Systems Inc., Tokyo, Japan) with high-resolution and optical chromoendoscopy feature under sedation with midazolam and propofol after at least eight hours of fasting.

Inlet patch was defined as the appearance of a salmon-colored, oval, or round patch clearly demarcated from the normal esophageal mucosa in the upper esophagus on endoscopy as shown in Figure 1 and Figure 2. An endoscopy shaft was used to measure IP size. Namely, the longitudinal length was measured by calculating the difference between the lower and upper ends, while the transverse dimension was measured using biopsy forceps. Following measurement, biopsy specimens were taken from the IP mucosa. After the biopsy materials were placed in a formalin-filled container, they were stained with Hematoxylin-eosin (H&E), and the type of gastric mucosa and the presence of *Helicobacter pylori* (*H. pylori*) in IP were evaluated.

The severity of esophagitis was graded according to the Los Angeles (LA) classification. Accordingly, mucosal breaks ≤ 5 mm across mucosal folds were defined as LA grade A, mucosal breaks >5 mm without continuity across mucosal folds as LA grade B, and mucosal breaks continuous between ≥ 2 mucosal folds but involving less than 75% of the esophageal circumference as LA grade C, and mucosal breaks involving $\geq 75\%$ of the esophageal circumference as LA grade D.

The diagnosis of Barrett's esophagus (BE) was based on the localization of salmon-colored

columnar epithelium of at least 3 cm in length instead of the squamous epithelium at the lower end of the esophagus, while hiatal hernia was defined as a distance >2 cm from the hiatus diaphragmaticus to the esophagogastric junction on endoscopy.

The examination records of the patients were reviewed via the hospital information system, and their age, sex, indication for endoscopy, the number and size of IPs, the presence of esophagitis, BE, and hiatal hernia were recorded.

Statistical analysis

Statistical analysis was performed using the IBM SPSS version 22.0 software (IBM Corp.,



Figure 1. Inlet patch white light image.

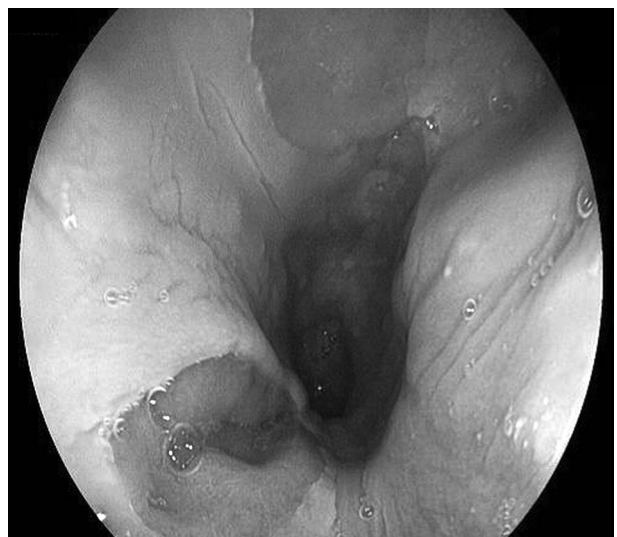


Figure 2. Inlet patch optical chromoendoscopic image.

Armonk, NY, USA). Quantitative data were expressed as mean \pm standard deviation (SD) and median range (maximum-minimum) values, while categorical data were expressed as numbers (n) and percentages (%).

RESULTS

Inlet patch was detected in 48 (2.5%) of patients who underwent esophagogastroduodenoscopy. The indications for endoscopy in patients diagnosed with IP were as follows: epigastric pain in 18 (37.5%) patients, epigastric burning sensation in 12 (25%) patients, dysphagia in nine (18.8%) patients, epigastric bloating in four (8.3%) patients, globus sensation in four (8.3%) patients, and tickly cough in one (2.1%) patient. Six (12.5%) patients had two IPs, while the remaining had a single IP. The mean number of IPs was 1.1 ± 0.3 . The mean IP diameter was 9.0 ± 5.8 mm, with the smallest IP measuring 3 mm and the largest IP measuring 30 mm. Of the patients with IP, five (10.4%) had esophagitis, six (12.5%) had hiatal hernia, and one (2.1%) had BE.

Fourteen (29.2%) of the patients with IP were symptomatic. The most common symptom was dysphagia (n=9, 64.3%), followed by

globus sensation (n=4, 28.6%) and tickly cough (n=1, 7.1%). Of the symptomatic patients, eight (57.1%) were male and six (42.9%) were female.

The biopsy specimens taken from the IP area revealed that heterotopic gastric mucosa was oxyntic in 26 (54%) patients and non-oxyntic (antral) in 22 (46%) patients, while none of the patients had metaplasia, dysplasia, or adenocarcinoma. Twelve (25%) patients were positive for *H. pylori* in the IP area. Of the *H. pylori*-positive patients, six (50%) had oxyntic mucosa and six (50%) had antral mucosa, and all of them had chronic active inflammation. The demographic, clinical, and endoscopic characteristics of the patients were shown in Table 1.

DISCUSSION

Cervical IP is a rare lesion that can be seen in all age groups, with a high prevalence of up to 14% reported in the literature.^[8] Moreover, it has a significant incidence of 70% in autopsy reports.^[15] The largest study investigating the prevalence of IP in our country was conducted by Senkaya et al.,^[16] who reported an IP prevalence of 1.24%. Furthermore, other studies conducted in our country have reported an IP prevalence ranging from 0.4 to 3.6%.^[3,17-20] The prevalence of IP in our study was 2.5%, which is consistent with the results reported in the literature. These differences in the prevalence of IP may be attributed to the following reasons: first, quickly passing the endoscope through the upper esophageal area by blind intubation while entering, and quick withdrawal of the endoscope from the lumen due to excessive gag reflex with the thought that the procedure is over and second, the endoscopist's insufficient awareness and knowledge of IP. It has been reported that the detection rates of IP increase as the withdrawal time and the use of optical chromoendoscopy techniques increase. Examination with routine narrow-band imaging has been shown to increase the detection rate of IP approximately three times compared to standard white light examination.^[21] In our study, all patients were sedated and examined using the optical chromoendoscopy mode on withdrawal of the endoscope from the esophagus.

In our study, 56.3% of the patients were female and 43.7% were male. While some studies in the literature have reported a higher prevalence

Table 1. Demographic, endoscopic, histopathological and clinical characteristics of the patients (n=48)

Parameters	n	%	Mean \pm SD
Age (year)			41.1 \pm 14.2
Sex			
Female	27	56.3	
Male	21	43.7	
Number of IPs			1.1 \pm 0.3
Inlet patch size (mm)			9.0 \pm 5.8
Hiatal hernia	6	12.5	
Esophagitis	5	10	
Barrett's esophagus	1	2.1	
Symptoms			
Dysphagia	9	18.8	
Globus sensation	4	8.3	
Cough	1	2	
Inlet patch mucosa type			
Oxyntic type	26	54	
Antral type	22	46	
<i>Helicobacter pylori</i> positivity	12	25	

SD: Standard deviation; IP: Inlet patch.

of IP in males,^[20,22] other studies have reported a higher prevalence in females.^[6,18,19]

In our study, the most common symptom associated with IP was dysphagia (18.8%), followed by globus and tickly cough. Most of our IP patients (70.8%) were asymptomatic. Most of the patients with a diagnosis of proximal esophageal IP are asymptomatic and the diagnosis is made incidentally during evaluation for other gastrointestinal complaints. However, some patients may develop symptoms secondary to acid secretion from the IP mucosa located in the cervical esophagus. The most common symptoms accompanying IP in the literature include globus sensation, a sensation of having something stuck in the upper cervical area, dysphagia, hoarseness, persistent cough, and odynophagia.^[23,24] Among these, the most common symptom has been reported as globus sensation, which has no correlation with IP size.^[25] However, some reports have shown that IP size may be associated with symptoms and the possible reason for this is believed to be more acid secretion and the consequent narrowing of the distal end of IP.^[26,27] In contrast, a recent study found no significant relationship between symptoms and the extent of IP.^[28]

Unlike IP, BE is a lesion that has a risk of acquired cancer, not congenital. However, since they show the same mucin core protein expression and cytokeratin pattern, it is believed that there is a pathogenetic relationship between both diseases.^[29,30] However, it has been reported that IP develops from embryonic gastric mucosa cells while Barrett's esophagus develops from immature multipotent stem cells.^[31] There are different results for the coexistence of IP and BE.^[5,32,33] Tang et al.^[5] found concurrent BE in 20% of patients with IP. A large case-control study found that BE was four times more common in patients with IP than in control patients without IP.^[34] Studies conducted in our country have reported a co-existence rate ranging from 3.5 to 13.2% for BE in patients with IP.^[6,18,19] In our study, the prevalence of BE in patients with IP was 2.1%, which was lower than the results reported in the literature. A study by Korkmaz et al.^[35] in our region found a prevalence of 2% for BE. This rate was lower than the results of the multicenter GORHEN study (4.2%) conducted in our country.^[36] We believe that the low

prevalence of IP and BE co-existence in our study may be related to the low prevalence of BE in our region.

Previous studies have reported the most common histological type detected in the IP mucosa as the oxyntic type. This is followed by the antral type.^[5,7,26,37] In our study, the most common histological type detected in the IP mucosa was the oxyntic type. None of our patients had intestinal metaplasia, dysplasia, or adenocarcinoma. Moreover, the positivity rate for *H. pylori* in the IP mucosa was 25% in our study. While Alagozlu et al.^[6] reported a positivity rate of 23.5% for *H. pylori* in the IP mucosa, Guiterrez et al.^[37] reported a very high prevalence of 73%. In addition, They reported that the density of *H. pylori* in the stomach was associated with the *H. pylori* positivity in the IP mucosa, while the type of IP mucosa was not associated with the *H. pylori* colonization; however, the non-oxyntic (antral or transitional) type of mucosa was associated with active inflammation.^[37] The results of our study showed no relationship between mucosal type and *H. pylori* colonization and active inflammation.

Previous studies have reported that proton pump inhibitors (PPIs) provide a significant reduction in symptoms.^[10,38,39] It has been reported that endoscopic treatments such as argon plasma coagulation or radiofrequency ablation are also safe and effective in patients who do not respond to PPI.^[27,40] In our study, we gave PPI to all of our symptomatic patients. In addition, eradication therapy was given to those who were positive for *H. pylori*. The proton pump inhibitor therapy was administered in the form of a gastroesophageal reflux treatment protocol. For the first 4-8 weeks, a single daily dose of PPI was given, then alternate-day or on-demand therapy was initiated. The proton pump inhibitor therapy provided a complete recovery in dysphagia and tickly cough complaints. However, despite a reduction in globus sensation, it did not disappear completely. Thereupon, anxiolytic therapy was initiated and the complaints were observed to have decreased significantly. None of our patients required endoscopic treatment.

Our study has some limitations. First, our study has a retrospective design. Second, the sample size is relatively small.

In conclusion, IP is a lesion that is underexplored or ignored, and its natural course and clinical significance are not yet well established. Although the condition is often asymptomatic, the cervical esophagus should be carefully examined for IP in patients with dysphagia, globus sensation, and unexplained chronic cough. Since this area is often blindly intubated, slow withdrawal of the endoscope and, if possible, the use of optical chromoendoscopy mode will increase visibility. Furthermore, sedating patients will reduce gag reflex, providing comfort to better assess the cervical esophagus. We are of the opinion that PPI therapy is effective in most symptomatic patients.

Ethics Committee Approval: The study protocol was approved by the Konya Medica Hospital Ethics Committee (date: 28.01.2022, no: 2022/01). The study was conducted in accordance with the principles of the Declaration of Helsinki.

Patient Consent for Publication: A written informed consent was obtained from each patient.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

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REFERENCES

- Raine CH. Ectopic gastric mucosa in the upper esophagus as a cause of dysphagia. *Ann Otol Rhinol Laryngol* 1983;92:65-6. doi: 10.1177/000348948309200115.
- Truong LD, Stroehlein JR, McKechnie JC. Gastric heterotopia of the proximal esophagus: A report of four cases detected by endoscopy and review of literature. *Am J Gastroenterol* 1986;81:1162-6.
- Akbayir N, Alkim C, Erdem L, Sökmen HM, Sungun A, Başak T, et al. Heterotopic gastric mucosa in the cervical esophagus (inlet patch): Endoscopic prevalence, histological and clinical characteristics. *J Gastroenterol Hepatol* 2004;19:891-6. doi: 10.1111/j.1440-1746.2004.03474.x.
- Borhan-Manesh F, Farnum JB. Incidence of heterotopic gastric mucosa in the upper oesophagus. *Gut* 1991;32:968-72. doi: 10.1136/gut.32.9.968.
- Tang P, McKinley MJ, Sporrer M, Kahn E. Inlet patch: Prevalence, histologic type, and association with esophagitis, Barrett esophagus, and antritis. *Arch Pathol Lab Med* 2004;128:444-7. doi: 10.5858/2004-128-444-IPHTA.
- Alagozlu H, Simsek Z, Unal S, Cindoruk M, Dumlu S, Dursun A. Is there an association between *Helicobacter pylori* in the inlet patch and globus sensation? *World J Gastroenterol* 2010;16:42-7. doi: 10.3748/wjg.v16.i1.42.
- Sahin G, Adas G, Koc B, Akcakaya A, Dogan Y, Goksel S, et al. Is cervical inlet patch important clinical problem? *Int J Biomed Sci* 2014;10:129-35.
- Peitz U, Vieth M, Evert M, Arand J, Roessner A, Malfertheiner P. The prevalence of gastric heterotopia of the proximal esophagus is underestimated, but preneoplasia is rare - correlation with Barrett's esophagus. *BMC Gastroenterol* 2017;17:87. doi: 10.1186/s12876-017-0644-3.
- Rodríguez-Martínez A, Salazar-Quero JC, Tutau-Gómez C, Espín-Jaime B, Rubio-Murillo M, Pizarro-Martín A. Heterotopic gastric mucosa of the proximal oesophagus (inlet patch): Endoscopic prevalence, histological and clinical characteristics in paediatric patients. *Eur J Gastroenterol Hepatol* 2014;26:1139-45. doi: 10.1097/MEG.000000000000177.
- Silvers WS, Levine JS, Poole JA, Naar E, Weber RW. Inlet patch of gastric mucosa in upper esophagus causing chronic cough and vocal cord dysfunction. *Ann Allergy Asthma Immunol* 2006;96:112-5. doi: 10.1016/S1081-1206(10)61049-6.
- Byrne M, Sheehan K, Kay E, Patchett S. Symptomatic ulceration of an acid-producing oesophageal inlet patch colonized by *helicobacter pylori*. *Endoscopy* 2002;34:514. doi: 10.1055/s-2002-32001.
- Bataller R, Bordas JM, Ordi J, Llach J, Elizalde JI, Mondelo F. Upper gastrointestinal bleeding: A complication of "inlet patch mucosa" in the upper esophagus. *Endoscopy* 1995;27:282. doi: 10.1055/s-2007-1005690.
- Jabbari M, Goresky CA, Lough J, Yaffe C, Daly D, Côté C. The inlet patch: Heterotopic gastric mucosa in the upper esophagus. *Gastroenterology* 1985;89:352-6. doi: 10.1016/0016-5085(85)90336-1.
- Orosey M, Amin M, Cappell MS. A 14-year study of 398 esophageal adenocarcinomas diagnosed among 156,256 EGDs performed at two large hospitals: An inlet patch is proposed as a significant risk factor for proximal esophageal adenocarcinoma. *Dig Dis Sci* 2018;63:452-65. doi: 10.1007/s10620-017-4878-2.
- Chong VH. Clinical significance of heterotopic gastric mucosal patch of the proximal esophagus. *World J Gastroenterol* 2013;19:331-8. doi: 10.3748/wjg.v19.i3.331.

16. Şenkaya A, Çelik F, Ünal N, Aslanov S, Sezak M, Doğanavşargil B, et al. İnlet patch olgularının retrospektif tek merkez değerlendirilmesi. *Endoskopi Gastrointestinal* 2020;28:82-7. doi: 10.17940/endoskopi.830763.
17. Kekilli M, Sayılır A, Yeşil Y, Beyazıt Y, Önal İK, Kurt M, et al. Servikal özofagustaki heterotopik gastrik mukozanın endoskopik sıklığı; bir referans merkez çalışması. *Akademik Gastroenteroloji Dergisi* 2009;8:119-22.
18. Yüksel I, Uskudar O, Köklü S, Başar O, Gültuna S, Unverdi S, et al. Inlet patch: Associations with endoscopic findings in the upper gastrointestinal system. *Scand J Gastroenterol* 2008;43:910-4. doi: 10.1080/00365520801986619.
19. Savaş N, Akbaş E. Heterotopik gastrik mukozanın sıklığı, klinik önemi ve eşlik eden diğer klinik bulgular. *Endoskopi Gastrointestinal* 2014;22:60-3. doi:10.17940/endoskopi.74772.
20. Poyrazoglu OK, Bahcecioğlu IH, Daglı AF, Ataseven H, Celebi S, Yalıniz M. Heterotopic gastric mucosa (inlet patch): Endoscopic prevalence, histopathological, demographical and clinical characteristics. *Int J Clin Pract* 2009;63:287-91. doi: 10.1111/j.1742-1241.2006.01215.x.
21. Al-Mammari S, Selvarajah U, East JE, Bailey AA, Braden B. Narrow band imaging facilitates detection of inlet patches in the cervical oesophagus. *Dig Liver Dis* 2014;46:716-9. doi: 10.1016/j.dld.2014.05.001.
22. Takeji H, Ueno J, Nishitani H. Ectopic gastric mucosa in the upper esophagus: Prevalence and radiologic findings. *AJR Am J Roentgenol* 1995;164:901-4. doi: 10.2214/ajr.164.4.7726045.
23. Ohara M. T1590: Incidence of heterotopic gastric mucosa in the upper esophagus in first time narrow banding image endoscopy of consecutive 900 patients. *Gastrointestinal Endoscopy* 2010;71:316-7. doi: 10.1016/j.gie.2010.03.804.
24. Maconi G, Pace F, Vago L, Carsana L, Bargiggia S, Bianchi Porro G. Prevalence and clinical features of heterotopic gastric mucosa in the upper oesophagus (inlet patch). *Eur J Gastroenterol Hepatol* 2000;12:745-9. doi: 10.1097/00042737-200012070-00005.
25. Ciocalteu A, Popa P, Ionescu M, Gheonea DI. Issues and controversies in esophageal inlet patch. *World J Gastroenterol* 2019;25:4061-73. doi: 10.3748/wjg.v25.i30.4061.
26. Behrens C, Yen PP. Esophageal inlet patch. *Radiol Res Pract* 2011;2011:460890. doi: 10.1155/2011/460890.
27. Shimamura Y, Winer S, Marcon N. A Giant circumferential inlet patch with acid secretion causing stricture. *Clin Gastroenterol Hepatol* 2017;15:A22-3. doi: 10.1016/j.cgh.2016.10.004.
28. López-Colombo A, Jiménez-Toxqui M, Gogeochea-Guillén PD, Meléndez-Mena D, Morales-Hernández ER, Montiel-Jarquín AJ, et al. Prevalence of esophageal inlet patch and clinical characteristics of the patients. *Rev Gastroenterol Mex (Engl Ed)* 2019;84:442-8. English, Spanish. doi: 10.1016/j.rgmx.2018.07.003.
29. Lauwers GY, Mino M, Ban S, Forcione D, Eatherton DE, Shimizu M, et al. Cytokeratins 7 and 20 and mucin core protein expression in esophageal cervical inlet patch. *Am J Surg Pathol* 2005;29:437-42. doi: 10.1097/01.pas.0000155155.46434.da.
30. Bogomoletz WV, Geboes K, Feydy P, Nasca S, Ectors N, Rigaud C. Mucin histochemistry of heterotopic gastric mucosa of the upper esophagus in adults: Possible pathogenic implications. *Hum Pathol* 1988;19:1301-6. doi: 10.1016/s0046-8177(88)80285-5.
31. Feurle GE, Helmstaedt V, Buehring A, Bettendorf U, Eckardt VF. Distinct immunohistochemical findings in columnar epithelium of esophageal inlet patch and of Barrett's esophagus. *Dig Dis Sci* 1990;35:86-92. doi: 10.1007/BF01537228.
32. Neumann WL, Luján GM, Genta RM. Gastric heterotopia in the proximal oesophagus ("inlet patch"): Association with adenocarcinomas arising in Barrett mucosa. *Dig Liver Dis* 2012;44:292-6. doi: 10.1016/j.dld.2011.11.008.
33. Chong VH, Jalihal A. Heterotopic gastric mucosal patch of the esophagus is associated with higher prevalence of laryngopharyngeal reflux symptoms. *Eur Arch Otorhinolaryngol* 2010;267:1793-9. doi: 10.1007/s00405-010-1259-2.
34. Avidan B, Sonnenberg A, Chejfec G, Schnell TG, Sontag SJ. Is there a link between cervical inlet patch and Barrett's esophagus? *Gastrointest Endosc* 2001;53:717-21. doi: 10.1067/mge.2001.114782.
35. Korkmaz H, Kerpiç O. Endoskopi yapılan hastalarda eroziv reflü hastalığının sıklığı, endoskopik, klinik ve histopatolojik özellikleri ve *Helicobacter pylori* ile ilişkisi. *Genel Tıp Derg* 2015;25:8-13.
36. Bor S, Vardar R, Vardar E, Takamz S, Mungan Z. T2014 Endoscopic findings of gastroesophageal reflux disease in Turkey: Multicenter prospective study (Gorhen). *Gastroenterology* 2008;134:A600. doi: 10.1016/S0016-5085(08)62804-8.
37. Gutierrez O, Akamatsu T, Cardona H, Graham DY, El-Zimaity HM. *Helicobacter pylori* and heterotopic gastric mucosa in the upper esophagus (the inlet patch). *Am J Gastroenterol* 2003;98:1266-70. doi: 10.1111/j.1572-0241.2003.07488.x.
38. Kim EA, Kang DH, Cho HS, Park DK, Kim YK, Park HC, et al. Acid secretion from a heterotopic gastric mucosa in the upper esophagus demonstrated by dual probe 24-hour ambulatory pH monitoring. *Korean J Intern Med* 2001;16:14-7. doi: 10.3904/kjim.2001.16.1.14.
39. Hamilton JW, Thune RG, Morrissey JF. Symptomatic ectopic gastric epithelium of the cervical esophagus. Demonstration of acid production with Congo red. *Dig Dis Sci* 1986;31:337-42. doi: 10.1007/BF01311666.
40. Dunn JM, Sui G, Anggiansah A, Wong T. Radiofrequency ablation of symptomatic cervical inlet patch using a through-the-scope device: A pilot study. *Gastrointest Endosc* 2016;84:1022-6.e2. doi: 10.1016/j.gie.2016.06.037.