

**Aim of the study:** Heterotopic gastric mucosa of the upper esophagus (HGMUE) may be connected with disorders of the upper gastrointestinal tract, exacerbated by *Helicobacter pylori*. Furthermore, HGMUE may be the origin of malignant progression to cervical esophageal carcinoma.

**Material and methods:** In this work, 20 patients with diagnosed heterotopic gastric mucosa of the upper esophagus (HGMUE) were subjected to 5-year follow-up to determine the extent and structure of histopathological changes within HGMUEs, as well as HGMUE dysplasia and metaplasia, and risk of their malignant transformation. As a diagnostic tool to describe localization, form, size and surface feature of HGMUEs, endoscopy was used. At the same time, the biopsies were collected for histopathological and microbiological analysis.

**Results:** In examined patients, HGMUEs were associated with inflammation, chronic gastritis, hiatus hernia, duodenal bulb erosion and ulcer and infection of *H. pylori*. Intestinal metaplasia and low grade dysplasia were also indicated. During 5 years of observation, both the clinical and histopathological image of diagnosed HGMUEs was stable. The patients with detected presence of *H. pylori* were treated with triple or quadruple therapy.

These results show that HGMUEs may be associated with severe complications in the gastrointestinal tract, such as infection by *H. pylori*, hiatus hernia or duodenal ulcer. Although dysplasias and metaplasias found in diagnosed HGMUEs were not very numerous and relatively stable both clinically and histopathologically, at the present stage of the study we cannot exclude the possibility of HGMUE malignant transformation.

**Key words:** heterotopic gastric mucosa, esophagus, metaplasia, dysplasia, malignant transformations.

# Clinical evaluation of twenty cases of heterotopic gastric mucosa of upper esophagus during five-year observation, using gastroscopy in combination with histopathological and microbiological analysis of biopsies

Wojciech Latos<sup>1</sup>, Karolina Sieroń-Stołtny<sup>1</sup>, Aleksandra Kawczyk-Krupka<sup>1</sup>, Tomasz Operchalski<sup>2</sup>, Grzegorz Cieślar<sup>1</sup>, Sebastian Kwiatek<sup>1</sup>, Andrzej M. Bugaj<sup>1,3</sup>, Aleksander Sieroń<sup>1</sup>

<sup>1</sup>Department and Clinic of Internal Diseases, Angiology and Physical Medicine in Bytom, Medical University of Silesia, Katowice, Poland

<sup>2</sup>Surgical Ward of Hospital of Ministry of Interior and Administration, Katowice, Poland

<sup>3</sup>College of Health, Beauty Care and Education, Poznań, Poland

## Introduction

Heterotopic gastric mucosa of the upper esophagus (HGMUE), also termed “inlet patch”, is heterotopic gastric mucosa (HGM) typically found in the esophagus. It is often characterized as a congenital asymptomatic anomaly. However, there are case reports showing that this lesion may play a role in the development of web, stricture, ulcer, perforation, or esophagotracheal fistula related to its capability of hydrochloric acid secretion [1–5]. These complications may be exacerbated by *Helicobacter pylori*, which colonizes 73% of “inlet patches” [6, 7]. Furthermore, HGMUE may be the origin of malignant progression to cervical esophageal carcinoma [8, 9]. Malignant progression of HGMUE occurs in a stepwise pattern, following the metaplasia-dysplasia-adenocarcinoma sequence [1, 9]. Despite the low incidence of clinically relevant manifestations of HGMUE, further studies are required to better understand its clinical significance [10].

The aim of this study was to evaluate the clinical features of 20 patients with diagnosed HGMUE, to determine prevalence of HGMUE dysplasia and intestinal metaplasia, to characterize the extent and structure of histopathological changes within HGMUE and, especially, to determine directions of these changes and risk of their malignant transformation during 5-year observation. The association of diagnosed “inlet patch” with *H. pylori* infection is also discussed.

## Material and methods

In total, examinations were carried out in 1039 patients referred to the Endoscopy Ambulatory of Chair and Clinics of Internal Diseases, Angiology and Physical Medicine at the Silesian University of Medical Sciences in Bytom, from 1 September 2005 to 10 March 2006. In 20 of the examined patients 2–4 HGMUE were diagnosed. The mean age of patients with diagnosed HGMUE was 45.5 (from 18 to 67 years) and did not considerably differ from the mean age of all examined patients (46.7 years). The male : female ratio of patients with HGMUE was about 0.54 (7 : 13). All patients were carefully questioned about symptoms particularly involving upper esophageal and laryngopharyngeal regions.

Endoscopy was carried out using the video-gastrosopes GIF Q 145 and GIF Q 165, both made by Olympus Optical Co. Ltd (Tokyo, Japan), after standard premedication (topical 10% lidocaine spray) [11]. All detected HGMUEs were described in terms of localization, form, size and surface feature. Two to four biopsy specimens were obtained from each HGMUE as well as from the antrum and angular notch for urease test to determine the presence of *H. pylori*, as well as from associated mucosa lesions in the upper esophagus, as reference samples. All biopsy specimens were examined by a team of experienced histopathologists.

All patients with diagnosed HGMUEs were subjected to 5-year follow-up in the endoscopy ambulatory, finished in 2011. For diseases associated with HGMUE, these patients were treated according to the standard protocol. The patients with detected presence of *H. pylori* were subjected to 10-day eradication with triple therapy consisting of proton-pump inhibitors at two doses per day, metronidazole at a dose of 500 mg twice per day and amoxicillin at a dose of 500 mg three times per day [12]. From all 20 observed subjects, three control biopsies were collected: the first one during the first 9 to 13 months after HGMUE diagnosis, the second one within a period of 3 years (35–38 months) after this diagnosis, and the third one after 5 years, at completion of follow-up. Two patients with diagnosed intestinal metaplasia and two others with dysplasia were examined by endoscopy every 6 months for the first 3 years and every 12 months later. By the term “dysplasia”, we mean a pathological, in some cases reversible state of epithelium, associated with cellular polymorphism, with disturbances of cell maturation and differentiation as well as with loss of basal cell polarity and of nucleus stratification. On the other hand, dysplasia, usually divided into low and high grade, is the most stable histological marker of premalignant states.

Each time, the sections from the antrum were collected for histopathological examination as well as for microbiological study to detect possible presence of *H. pylori* infection. When we did not obtain successful eradication of *H. pylori* with the standard protocol, we used quadruple protocols, with the use of clarithromycin or bismuth salts [12, 13].

## Results

Heterotopic gastric mucosa of upper esophagus were found in 20 patients from 1039 examinations. In the major-

ity of patients, no special symptoms related to HGMUE were observed. Only one woman – a patient with diagnosed HGMUE – reported stomach itching and acidity sensation in the mouth with accompanying periodic sialorrhea, and one other patient was diagnosed due to the sensation of a foreign body in the esophagus which increased during swallowing.

The rate of endoscopic detection was determined as 1.92%. In general, HGMUE patches appeared as salmon-red lesions localized immediately below the upper esophageal sphincter. All HGMUEs appeared as oval patches with smooth and glossy surfaces that were discriminated from the surrounding esophageal mucosa by their well-defined margins. In the majority of patients (17 of 20) HGMUE lesions appeared as single patches and in 3 patients they were bifocal. The size of HGMUE patches ranged between 10 and 40 mm; in the majority of cases (18 of 23 patches; 81.8%) it was within the range 15–25 mm. Only in 3 patients was the size of “inlet patches” greater than 25 mm and in just one patient their diameter was less than 15 mm.

The results of histopathological examinations are shown in Table 1. Histopathological evaluation of 23 “inlet patches” revealed the presence of 17 patches of fundic type, 5 of antral type with noticeable parietal cells, and 1 patch of transitional (prepyloric) type. Inflammation was found in 16 of 23 detected HGMUEs; however, only one of these patches was infected with *H. pylori*. In 16 of 20 examined patients HGMUE was associated with chronic gastritis, in 8 with gastric infection of *H. pylori*, in 3 with duodenal bulb erosion, and in single cases with hiatus hernia and with duodenal ulcer. In 3 patients (15%) intestinal metaplasia in the stomach and in 2 patients (10%) atrophy of the mucosa was present. In 13 of the examined patients, a stomach infection of *H. pylori* was detected.

In 12 of 14 patients subjected to 10-day eradication using proton pump inhibitors, metronidazole and amoxicillin, the *H. pylori* gastric infection was eliminated. Efficiency of this antibacterial treatment protocol in these patients has been shown already during the first control endoscopy. The subsequent endoscopic analyses showed stable histopathological features of HGMUE and no reinfection of *H. pylori* in this group of patients. Apart from this, healing of all gastric and duodenal erosions as well as reduction of inflammato-

**Table 1.** Histopathological characteristics of 23 gastric mucosa ectopies found in upper esophagus of 20 patients, on the day of diagnosis

Coexistence of Hp infection with 23 HGMUE ectopies in 20 patients		Histological type of gastric ectopy in upper esophagus	Presence of metaplasia/dysplasia and presence of Hp in HGMUE	
present	absent		intestinal metaplasia	low-grade dysplasia
10 ectopies in 9 patients with Hp(+)	7 ectopies with Hp(-)	17 cases [1 case with Hp(+)] antral type	1 Hp(+) 1 Hp(-)	1 Hp(-)
5 ectopies in 3 patients with Hp(+)	no ectopies with Hp(-)	5 cases body type	no Hp	1 Hp(-)
1 ectopy with Hp(+)	no ectopies with Hp(-)	1 case prepyloric type	no Hp	no Hp

Hp – *Helicobacter pylori*

ry states in the examined patients was observed. The 2 patients in whom after a year of standard treatment the *H. pylori* infection was still detected were successfully subjected to quadruple therapy, as shown by the examination performed 6 months after starting the new protocol of pharmacotherapy. In patient with *H. pylori* infection detected with ectopies, we obtained successful eradication after one treatment cycle and we did not observe any reinfection during 5-year follow-up. In two patients with intestinal metaplasia within HGMUEs the changes were present during 5-year observation and their histopathological image was stable. The presence of low-grade dysplasia within 2 of 23 diagnosed HGMUEs was indicated. In both these HGMUEs, both image and grade of detected dysplasia were stable and did not change during 5-year observation. Observation of all described patients was not finished and all these persons are subjected to increased oncological supervision.

## Discussion

The “inlet patch” is found in 10% of the population with careful searching at endoscopy [14, 15] but its presence is often overlooked or underestimated by endoscopists so that studies frequently report a prevalence between 0.1 and 3% [1, 2, 16–18]. Thus, awareness and carefulness of the endoscopist considerably affect the detection rate of heterotopic gastric mucosa in the esophagus [2, 19]. In our study, the detection rate of HGMUE was 1.92%, which was similar to the result obtained by the endoscopist-aware group (2.3%) by contrast to that of the unaware group (0.2%), in the work of Korkut *et al.* [20].

The clinical importance of HGMUE is limited to symptomatic cases. Most “inlet patches” are clinically asymptomatic; only in 10% of cases do they produce clinical symptoms, such as chest and throat pain, dysphagia, globus sensation, shortness of breath, chronic cough and hoarseness [1, 16, 21, 22]. In our study only 2 of 20 patients reported clinical symptoms related to HGMUE.

It has been argued that acid secretion has a significant role in the symptomatic progression of HGMUE and, more importantly, can cause morphological changes such as stricture and ulceration in the mucosa [1, 23]. Acid secretion from HGM has been shown by various methods [24–27]. Acid secretion from the parietal cells has also been demonstrated [28–30]. Contrary to the results of Poyrazoglu *et al.* [23], we demonstrated the presence of parietal cells in HGMUE, but no correlation between clinical symptoms and presence of these cells was found. This suggests that not only the presence but also the quantity of parietal cells is associated with HGMUE [23]. Furthermore, it was confirmed that symptoms are more marked in patients who have larger HGM patches [1, 31]. However, in the present study, there was no correlation between clinical symptoms and HGMUE size or type.

A strong affinity of *H. pylori* to colonize gastric type mucosa has been well demonstrated [6, 32–35]. In our research, colonization of HGMUE with *H. pylori* was not observed in the absence of this bacterium in the stomach, and such coexistence was observed only in one case. In fact, according to Borhan-Manesh and Farnum [34] and Guttierrez *et al.* [6] there

is no correlation between positivity of *H. pylori* and inflammation in HGMUE patches. Whereas, we showed that the inflammatory process in HGMUE may be correlated with esophagitis, gastritis, or duodenitis, which may be severe complications of “inlet patches” [16, 35].

The question of malignant progression within HGMUE is controversial. The heterotopic, but otherwise not malignant epithelium may advance to invasive carcinoma following a metaplasia-dysplasia-carcinoma sequence [36]. Some authors [1] claim that the low frequency of malignant transformation of HGMUE as compared to Barrett’s esophagus suggests that it may not be regarded as a premalignant lesion. On the other hand, about 25 cases of adenocarcinoma which did not raise doubt to be developed from HGMUE were described [37–41]. Intestinal metaplasias have often been reported in association with adenocarcinoma developing in the inlet patch, but dysplasia has been reported only occasionally [42]. In our study, both metaplasia and dysplasia within HGMUE were found, contrary to the results of Tang *et al.* [16] and Akbayir *et al.* [17], who did not find metaplastic lesions in the HGMUE. Although these changes were not very numerous (about 12 and 9% for dysplasias and metaplasias respectively) and relatively stable, both clinically and histopathologically, at the present stage of the study, the possibility of malignant transformation within HGMUE cannot be excluded.

On the other hand, it is necessary to emphasize the advantages of endoscopy in combination with biopsy as a practical and safe method of inlet patch diagnosis [1, 11, 43–45]. This method is easy to carry out and repeat in ambulatory conditions and is well tolerated by patients. Due to its sensitivity to the mucosa, endoscopy is an effective tool for projecting deep mucosal visuals, allowing one to effectively evaluate the digestive system. It can also document diseases by generating three-dimensional images [19]. Thus, in our study we decided to use a method of endoscopic imaging allowing at the same time biopsies of gastrointestinal tissues for histopathological analysis.

In conclusion, the results of studies revealed that HGMUE should not be overlooked in the diagnostics. Although only severe symptomatic cases of this disorder require medical management, HGMUE may be associated with important complications in relation to gastric acid secretion, such as infection by *H. pylori*, hiatus hernia or duodenal ulcer, which should not be underestimated. Although the numbers of dysplasias and metaplasias in the diagnosed HGMUEs were limited (about 9 and 12%, respectively), and these lesions were relatively stable clinically, at the present stage of the study we cannot exclude the possibility of HGMUE malignant progression into adenocarcinoma. This may suggest the need for revision of the old paradigm of HGMUE as a benign and asymptomatic disorder. However, this question requires further studies.

On the other hand, the obtained results showed that the method of endoscopy combined with histopathological and microbiological analysis of biopsies is – due to its sensitivity, specificity and safety – a method “of choice” in clinical evaluation of metaplastic and non-metaplastic changes within HGMUE [43, 44].

*The authors declare no conflict of interest.*

## References

1. von Rahden BHA, Stein HJ, Becker K, Liebermann-Meffert D, Siewert JR. Heterotopic gastric mucosa of the esophagus: literature-review and proposal of a clinicopathologic classification. *Am J Gastroenterol* 2004; 99: 543-51
2. Azar C, Soweid A. Inlet patch: the "under-explored" island. *J Clin Gastroenterol* 2009; 43: 97-8.
3. Sánchez-Pernaute A, Hernando F, Díez-Valladares L, et al. Heterotopic gastric mucosa in the upper esophagus ("inlet patch"): a rare cause of esophageal perforation. *Am J Gastroenterol* 1999; 94: 3047-50.
4. Kohler B, Kohler G, Riemann JF. Spontaneous esophageal fistula resulting from ulcer in heterotopic gastric mucosa. *Gastroenterology* 1988; 95: 828-30.
5. Rosztóczy A, Izbéki F, Németh IB, et al. Detailed esophageal function and morphological analysis shows high prevalence of gastroesophageal reflux disease and Barrett's esophagus in patients with cervical inlet patch. *Dis Esophagus* 2012; 25: 498-504.
6. 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.
7. Katsanos KH, Kamina S, Christodoulou DK, Mitsi V, Tsianos EV. Ulcerated Helicobacter pylori negative gastric heterotopy in the upper esophagus causing foreign body sensation. *Ann Gastroenterol* 2009; 22: 123-5.
8. Noguchi T, Takeno S, Takahashi Y, Sato T, Uchida Y, Yokoyama S. Primary adenocarcinoma of the cervical esophagus arising from heterotopic gastric mucosa. *J Gastroenterol* 2001; 36: 704-9
9. Komori S, Osada S, Tanaka Y, Takahashi T, Nagao N, Yamaguchi K, Asano N, Yoshida K. A case of esophageal adenocarcinoma arising from the ectopic gastric mucosa in the thoracic esophagus. *Rare Tumors* 2010; 2: 12-5
10. Chong VH. Clinical significance of heterotopic gastric mucosal patch of the proximal esophagus. *World J Gastroenterol* 2013; 19: 331-8
11. Operchalski T, Latos W, Drozdowska B, Sieroń-Stotny K, Sieroń A. Two cases of dysplasia in heterotopic gastric mucosa in upper esophagus. *Chirurgia Polska* 2007; 9: 112-9.
12. O'Connor A, Gisbert JP, McNamara D O'Morain C. Treatment of Helicobacter Pylori infection 2011. *Helicobacter* 2011;16 Suppl 1: 53-8.
13. Treiber G, Ammon S, Schneider E, Klotz U. Amoxicillin/Metronidazole/Omeprazole/Clarithromycin: a new, short quadruple therapy for Helicobacter pylori eradication. *Helicobacter* 1998; 3: 54-8.
14. Behrens C, Yen PPW. Esophageal inlet patch (a case report). *Radiol Res Pract* 2011; 2011: 460890.
15. Borhan-Manesh F, Farnum JB. Incidence of heterotopic gastric mucosa in the upper esophagus. *Gut* 1991; 32: 968-972
16. 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.
17. Akbayir N, Alkim C, Erdem L, Sökmen HM, Sungun A, Ba ak T, Turgut S, Mungan Z. Heterotopic gastric mucosa in the cervical esophagus (inlet patch): endoscopic prevalence, histological and clinical characteristics. *J Gastroenterol Hepatol* 2004; 19: 891-6.
18. 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.
19. Moore LE. The advantages and disadvantages of endoscopy. *Clin Tech Small Animal Pract* 2003; 18: 250-3.
20. Korkut E, Bektas M, Savas B, Memmedzade F, Oztas E, Ustün Y, Idilman R, Ozden A. Awareness of the endoscopist affects detection rate of heterotopic gastric mucosa in esophagus. *Indian J Gastroenterol* 2009; 28: 75-6
21. 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.
22. Basseri B, Conklin JL, Mertens RB, Lo SK, Bellack GS, Shaye OA. Heterotopic gastric mucosa (inlet patch) in a patient with laryngopharyngeal reflux (LPR) and laryngeal carcinoma: a case report and review of literature. *Dis Esophagus* 2009; 22: E1-E5.
23. Poyrazoglu OK, Bahcecioglu IH, Dagli AF, Ataseven H, Celebi S, Yalniz M. Heterotopic gastric mucosa (inlet patch): endoscopic prevalence, histopathological, demographical and clinical characteristics. *Int J Clin Pract* 2009; 63: 287-91.
24. 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.
25. Nakajima H, Munakata A, Sasaki Y, Yoshida Y. pH profile of esophagus in patients with inlet patch of heterotopic gastric mucosa after tetragastrin stimulation. An endoscopic approach. *Dig Dis Sci* 1993; 38: 1915-9.
26. Galan AR, Katzka DA, Castell DO. Acid secretion from an esophageal inlet patch demonstrated by ambulatory pH monitoring. *Gastroenterology* 1998; 115: 1574-1576
27. Korkut E, Bekta M, Alkan M, Ustün Y, Meco C, Ozden A, Soykan I. Esophageal motility and 24-h pH profiles of patients with heterotopic gastric mucosa in the cervical esophagus. *Eur J Int Med* 2010; 21: 21-4.
28. Helander HF. Physiology and pharmacology of the parietal cell. *Baillieres Clin Gastroenterol* 1988; 2: 539-54.
29. Mangeat P, Gusdinar T, Sahuquet A, Hanzel DK, Forte JG, Magous R. Acid secretion and membrane reorganization in single gastric parietal cell in primary culture. *Biol Cell* 1990; 69: 223-32.
30. Yao X, Forte JG. Cell biology of acid secretion by the parietal cell. *Annu Rev Physiol* 2003; 65: 103-31.
31. Ueno J, Davis SW, Tanakami A, Seo K, Yoshida S, Nishitani H, Irie H, Lu CC. Ectopic gastric mucosa in the upper esophagus: detection and radiographic findings. *Radiology* 1994; 191: 751-3.
32. Flejou JF, Potet F, Molas G, et al. Campylobacter-like organisms in heterotopic gastric mucosa of the upper oesophagus. *J Clin Pathol* 1990; 43: 961.
33. 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.
34. Borhan-Manesh F, Farnum JB. Study of Helicobacter pylori colonisation of patches of heterotopic gastric mucosa (HGM) at the upper esophagus. *Dig Dis Sci* 1993; 38: 142-6.
35. Song ZY, Huang X, Qian KD, Peng JP, Sun AW, Zhang YY, Zhao ZG. Clinical analysis of 39 cases of heterotopic gastric mucosa in the upper esophagus. *Zhonghua Yi Xue Za Zhi* 2005; 85: 244-7.
36. Fearon ER, Vogelstein B. A genetic model for colorectal tumorigenesis. *Cell* 1990; 61: 759-61.
37. Mion F, Lambert R, Partensky C, Cherkaoui M, Berger F. High-grade dysplasia in an adenoma of the upper esophagus developing on heterotopic gastric mucosa. *Endoscopy* 1996; 28: 633-5.
38. Abe T, Hosokawa M, Kusumi T, et al. Adenocarcinoma arising from ectopic gastric mucosa in the cervical esophagus. *Am J Clin Oncol* 2004; 27: 644-5.
39. Noguchi T, Takeno S, Takahashi Y, Sato T, Uchida Y, Yokoyama S. Primary adenocarcinoma of the cervical esophagus arising from heterotopic gastric mucosa. *J Gastroenterol* 2001; 36: 704-9.
40. Klaase JM, Lemaire LC, Rauws EA, Offerhaus GJ, van Lanschot JJ. Heterotopic gastric mucosa of the cervical esophagus: A case of high-grade dysplasia treated with argon plasma coagulation and a case of adenocarcinoma. *Gastrointest Endosc* 2001; 53: 101-4
41. Sauvé G, Croué A, Denez B, Boyer J. High-grade dysplasia in heterotopic gastric mucosa in the upper esophagus after radiotherapy: Successful eradication 2 years after endoscopic treatment by argon plasma coagulation. *Endoscopy* 2001; 33: 732.
42. Lauwers GY, Scott GV, Vauthey JN. Adenocarcinoma of the upper esophagus arising in cervical ectopic gastric mucosa. Rare evidence of malignant potential of so-called "inlet patch". *Dig Dis Sci* 1998; 43: 901-7.
43. Nguyen VX, Le Nguyen VT, Nguyen CC. Appropriate use of endoscopy in the diagnosis and treatment of gastrointestinal diseases: up-to-date indications for primary care providers. *Int J Gen Med* 2010; 3: 345-57.
44. Puttemans NAM, Andre PP, Jamsin SAMJ, Balikdijan DPR, Lustman F. Detection of gastroduodenal ulcers using Technetium-99m-labeled

sucrafate. In: Nuclear Medicine in Gastroenterology. Biersack HJ, Cox PH (eds.). Kluwer Academic Publishers, Dordrecht – Boston 1991, 139-51.

45. Hoshino A, Otuka Y, Nara S, Harihara Y, Konishi T. A case of primary adenocarcinoma of the cervical esophagus arising from the ectopic gastric mucosa. *Esophagus* 2007; 4: 83-6.

**Address for correspondence:**

**Andrzej Marcin Bugaj**

os. Jagiellońskie 3 m. 9

61-224 Poznań, Poland

mobile: +48 501 645 004

e-mail: andrzej.marcin.bugaj@gmail.com

**Submitted:** 23.10.2012

**Accepted:** 14.02.2013