DOI: 10.17816/PED9541-46

SURGICAL TREATMENT COMBINATION OF GASTROESOPHAGEAL REFLUX DISEASE AND CELIAC TRUNK COMPRESSION SYNDROME

© D.I. Vasilevsky, S.G. Balandov, A.M. Ignashov, K.A. Anisimova, L.I. Davletbaeva

Pavlov First Saint Petersburg State Medical University, Ministry of Healthcare of the Russian Federation, Saint Petersburg, Russia

For citation: Vasilevsky DI, Balandov SG, Ignashov AM, et al. Surgical treatment combination of gastroesophageal reflux disease and celiac trunk compression syndrome. *Pediatrician (St. Petersburg)*. 2018;9(5):41-46. doi: 10.17816/PED9541-46

Received: 21.08.2018 Accepted: 12.10.2018

The combination of gastroesophageal reflux disease and the celiac trunk compression syndrome is well known and reflected in the literature comorbidity. In some articles a possible pathogenetic relationship of these diseases was assumed. A common factor in the development of gastroesophageal reflux and the s celiac trunk compression syndrome can be disorganization of the motor function of the digestive tract on the background of their chronic ischemia. However there is no the confirmation or the refutation of this hypothesis. The various clinical aspects of the combination of both diseases insufficiently developed. The modern concept of treating gastroesophageal reflux disease involves conservative therapy. Surgical treatment of this illness is justified only in the not-effective pharmacological cases. Unfortunately, refractory gastroesophageal reflux symptoms noted in 15-40% of cases during the drug medication. The only effective option to eliminate the celiac trunk compression syndrome is the surgical restoration of the full-value blood flow along this vessel. The need to complement the celiac trunk decompression with antireflux reconstruction in comorbidity cases remains a controversial issue and requires further studies. In the article presents an analysis of the results of examination and treatment of 84 patients with a combination of the celiac trunk compression syndrome and gastro-esophageal reflux disease. The factors predetermined or determined the ineffectiveness of drug therapy for gastroesophageal reflux in that comorbidity were identified. The tactics of treatment the gastroesophageal reflux disease the patients with the background of the celiac trunk compression syndrome, was developed and justified.

Keywords: celiac trunk compression syndrome; gastroesophageal reflux disease; combination; treatment; surgical treatment; spiral computed tomography; ultrasonic duplex scanning; impedance of pH-metry.

ХИРУРГИЧЕСКОЕ ЛЕЧЕНИЕ СОЧЕТАНИЯ ГАСТРОЭЗОФАГЕАЛЬНОЙ РЕФЛЮКСНОЙ БОЛЕЗНИ И СИНДРОМА КОМПРЕССИИ ЧРЕВНОГО СТВОЛА

© Д.И. Василевский, С.Г. Баландов, А.М. Игнашов, К.А. Анисимова, Л.И. Давлетбаева

ФГБОУ ВО «Первый Санкт-Петербургский государственный медицинский университет им. акад. И.П. Павлова» Минздрава России, Санкт-Петербург

Для цитирования: Василевский Д.И., Баландов С.Г., Игнашов А.М., и др. Хирургическое лечение сочетания гастроэзофагеальной рефлюксной болезни и синдрома компрессии чревного ствола // Педиатр. – 2018. – Т. 9. – № 5. – С. 41–46. doi: 10.17816/PED9541-46

Поступила в редакцию: 21.08.2018 Принята к печати: 12.10.2018

Сочетание гастроэзофагеального рефлюкса и синдрома компрессии чревного ствола известно и отражено в литературе. В отдельных работах указывается на возможную патогенетическую взаимосвязь данных заболеваний. Общим фактором развития желудочно-пищеводного заброса при синдроме компрессии чревного ствола может быть дезорганизация моторной функции верхних отделов пищеварительного тракта на фоне их хронической ишемии. Однако подтверждения или опровержения данной гипотезы до настоящего времени не получено. Немногочисленные исследования сочетания гастроэзофагеального рефлюкса и синдрома компрессии чревного ствола практически не касаются вопросов патогенеза. Недостаточно разработанными остаются и различные клинические аспекты комбинации обоих заболеваний. Современная концепция лечения желудоч-

но-пищеводного заброса предполагает проведение консервативной терапии. Хирургическое лечение считается показанным только при неэффективности данного подхода. Однако рефрактерное к медикаментозной коррекции течение гастроэзофагеальной рефлюксной болезни отмечается в 15–40 % случаев. Единственным эффективным способом устранения синдрома компрессии чревного ствола является хирургическое восстановление полноценного кровотока по этому сосуду. Однако необходимость дополнения декомпрессии чревного ствола антирефлюксной реконструкцией при сочетании данного заболевания с желудочно-пищеводным забросом остается спорным вопросом и требует дальнейшего изучения. В статье представлен анализ результатов обследования и лечения 84 пациентов с сочетанием синдрома компрессии чревного ствола и гастроэзофагеальной рефлюксной болезни, находившихся в ПСПбГМУ им. И.П. Павлова с 2011 по 2015 г. На основании проведенного исследования выявлены факторы, предрасполагающие или определяющие неэффективность медикаментозной терапии желудочно-пищеводного заброса при подобном сочетании заболеваний. Выделены основные симптомы, характерные для сочетания обоих заболеваний. Разработана и обоснована тактика лечения пациентов с гастроэзофагеальной рефлюксной болезнью, развившейся на фоне синдрома компрессии чревного ствола.

Ключевые слова: синдром компрессии чревного ствола; гастроэзофагеальная рефлюксная болезнь; сочетание; лечение; хирургическое лечение; спиральная компьютерная томография; ультразвуковое дуплексной сканирование; импеданс pH-метрия.

INTRODUCTION

Gastroesophageal reflux disease is defined as a complex of symptoms and complications that develop as a result of regurgitation of gastric contents into the esophagus.

The prevalence of gastroesophageal reflux disease in the adult population of Russia is 14%, which corresponds to indices of other economically-developed countries [7].

In the pathogenesis of the disease, the impairment of barrier mechanisms of the gastroesophageal transition, the inhibition of esophageal clearance (primarily propulsive movements), and the increase in pressure in the stomach and duodenum are most important. Decreased production or altered chemical properties of saliva, reduced mucus production by the glands of the esophagus, increased sensitivity of the esophageal epithelium, and various other reasons may have certain significance in the development of this pathology. The complex interactions of all these factors determine the diversity of clinical and organic manifestations of the disease [1, 3, 6].

Modern understanding of celiac axis compression syndrome is a complex set of clinical symptoms resulting from the reduction of blood flow in a particular vessel and the development of chronic ischemia of the abdominal organs [2, 5].

The most common cause of compression stenosis of the celiac axis is compression by the median arcuate ligament of the diaphragm. It is believed that this variant of anatomical structure is found in 5%–20% of people and can be transmitted by autosomal dominant mode of inheritance. Another mechanism of disease development may be compression of the artery by altered elements of the celiac plexus. Other rarer factors of the occurrence of this pathological condition are also possible [4, 5, 10, 11].

In the general population, celiac axis compression syndrome is found in 0.4% of people. The significant discrepancy in the frequency of occurrence of anatomical prerequisites for the development of the disease and its clinical manifestations suggests the presence of additional, probably physiological conditions leading to its expression. The individual features of the collateral blood supply to the abdominal cavity from other great vessels are most obvious but practically difficult to study [2, 13].

The possibility of the combination of gastroesophageal reflux disease and celiac axis compression syndrome is known and has been reported to some extent in the literature. However, the clinical issues of this complex condition have scarcely been studied to date. In separate studies, a possible inter-relation of some factors of the pathogenesis of both diseases has been reported. The most obvious and important common mechanism for the development of celiac axis compression syndrome and gastroesophageal reflux may be a disorder of the motor-evacuation function of the upper digestive tract due to chronic ischemia [12].

A modern strategy for the treatment of gastroesophageal reflux disease involves conservative therapy. Surgical elimination of gastroesophageal reflux is considered justified only if the pharmacological treatment is ineffective or the patient is intolerant of medication [3, 6].

Refractoriness of the disease symptoms to drug therapy has been reported in 15%–40% of cases and may have several causes. One of the possible factors

of such a course of gastroesophageal reflux is thought to be persistent severe dysfunction of motor activity of the esophagus, stomach, and duodenum. Modern therapeutic medicine does not currently have treatment options to effectively influence such a variant of the disease course [3].

The only way to treat the celiac axis compression syndrome, which is quite radical, is the surgical removal of occlusion of this vessel. Endovascular technology has become widespread over the past two decades, and it used to restore blood flow in a wide variety of diseases. However, it does not achieve the desired effect in case of compression stenosis of the celiac axis [5, 8, 9].

The combination of gastroesophageal reflux disease and celiac axis compression syndrome found in clinical practice, together with the lack of generally accepted ideas about the strategy and methods of treatment of such a combination of pathological conditions served as the basis for a more substantive study of the problem.

MATERIALS AND METHODS

A retrospective analysis was performed of 84 patients with a combination of celiac axis compression syndrome and gastroesophageal reflux disease treated from 2011 to 2015 at the clinic of Departmental Surgery of the Academician I.P. Pavlov First St. Petersburg State Medical University.

There were 30 men (35.7%) and 54 women (64.3%) with an average age of 32.8 years.

The diagnosis of celiac axis compression syndrome was based on the clinical manifestations of the disease (abdominal pain aggravated after eating or physical exercise), data of ultrasound duplex scanning, and spiral computer angiography of visceral vessels. In some cases, a direct aortography was required.

The diagnosis of gastroesophageal reflux disease was based on the major symptoms of the disease (heartburn, belching, dysphagia, odynophagia) and the results of esophagogastroscopy and impedance-pH monitoring of the esophagus. If necessary, histological examination of biopsy specimens of the esophageal mucosa was performed.

RESULTS

The first stage of treatment was an eight-week course of conservative therapy. This included proton pump inhibitors, antacids, and prokinetics prescribed to all 84 patients with a proven combination of both diseases to eliminate the manifestations of gastroesophageal reflux.

There were 29 patients (34.5%) with effective drug control of gastroesophageal reflux symptoms, and surgical treatment was not considered for these patients.

Esophagogastroscopy revealed no morphological changes in 18 of 29 cases, and the presence of gastroesophageal reflux was confirmed based on 24-h impedance-pH monitoring of the esophagus. In 11 patients, there were slight changes in the mucous membrane of the esophagus (esophagitis stages A and B according to the Los Angeles classification).

Based on duplex scanning and computer angiography, the mean degree of celiac stenosis in patients with susceptible drug therapy of gastroesophageal reflux disease was 55.4%.

In 55 (65.5%) patients, conservative therapy did not eliminate the manifestations of gastroesophageal reflux. A similar variant of the course of the disease was considered as an indication to perform anti-reflux reconstruction.

In esophagoscopy, 50 (90.1%) of 55 patients with continuing manifestations had erosive changes in the esophageal mucosa, including 12 (21.8%) with severe changes (stages C and D). Two (3.6%) patients were diagnosed with gastric Barrett metaplasia, and no organic changes were observed in three (5.5%) patients during esophagogastroscopy.

The average degree of celiac axis stenosis in patients with gastroesophageal reflux refractory to drug therapy was 70.1%, which was significantly higher than that in patients with conservatively-treatable disease variants ($p < 0.0001$).

Decompression of the celiac axis was performed in all 84 patients with a combination of the diseases. In all patients, surgery was performed with the traditional approach to reduce the risk of uncontrolled bleeding from the celiac axis or aorta during manipulation.

Surgery in patients with gastroesophageal reflux unresponsive to drug therapy was supplemented with an anti-reflux reconstruction “short floppy” R. Nissen.

The choice of method was determined by its proven good outcome in the elimination of manifestations of gastroesophageal reflux disease and the ability to perform the procedure in patients with any type of disorder of esophageal motor activity.

There were no intraoperative complications and deaths. Four (7.3%) patients had mild functional dysphagia, which resolved on its own by the fourth week of the postoperative period. In 11 (13.1%) patients, signs of hypokinetic dyskinesia of the stomach and symptoms of flatulence were found after elimination of the celiac axis compression syndrome and

gastroesophageal reflux disease, requiring conservative treatment.

Based on the analysis of the approaches used to provide medical care to patients with a combination of celiac axis compression syndrome and gastroesophageal reflux disease, an algorithm for selecting therapeutic tactics was developed (Fig. 1).

Long-term results were evaluated in 78 of 84 patients. Among them, 29 underwent isolated decompression of the celiac axis, and 49 underwent the same procedure in combination with fundoplication "short floppy" R. Nissen.

There were no clinical manifestations of celiac axis compression syndrome and gastroesophageal reflux in 67 patients (85.9%).

According to the results of ultrasound duplex scanning, 75 of 78 patients (96.2%) had normalization of the anatomical and hemodynamic parameters of the celiac axis six months after surgery. In three (3.8%) patients, residual stenosis of the

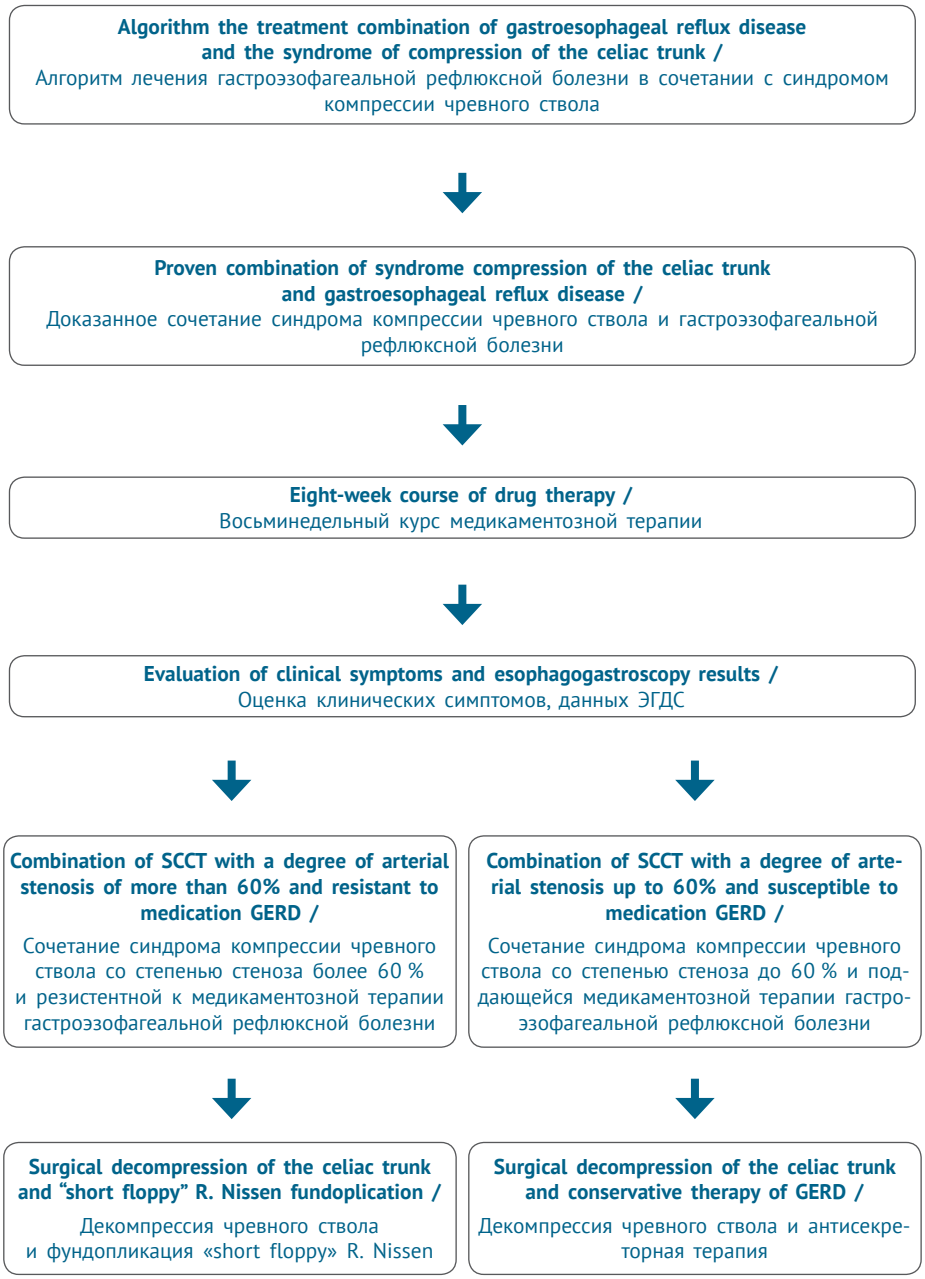

Fig. 1. Algorithm the treatment combination of gastroesophageal reflux disease (GERD) and the syndrome of compression of the celiac trunk (SCCT)

Рис. 1. Алгоритм лечения гастроэзофагеальной рефлюксной болезни в сочетании с синдромом компрессии чревного ствола

celiac axis (up to 30% of the vessel lumen) without clinical manifestations was reported. Occlusion that required revision and isolation of the celiac axis from cicatricial adhesions was found in two patients (2.6%).

Gastroesophageal reflux recurrence was observed in four of 49 patients (8.2%) who underwent anti-reflux surgery. Three patients received effective conservative therapy, and one underwent repeated fundoplication.

With isolated decompression of the celiac axis in 26 of 29 patients (89.6%), no clinical manifestations of gastroesophageal reflux during drug therapy were observed.

CONCLUSION

In gastroesophageal reflux disease developed with celiac axis compression syndrome, conservative treatment is often ineffective, which may be due to a significant reduction in blood flow (>60%) in the visceral arteries. This serves as an indication for the simultaneous surgical treatment of both diseases.

For manifestations of gastroesophageal reflux that develop with celiac axis compression syndrome with the degree of stenosis up to 60%, drug therapy can be effective, and surgical treatment should be limited to isolated decompression of the celiac axis.

The optimal method of simultaneous surgical treatment of the celiac axis compression syndrome and gastroesophageal reflux disease refractory to drug therapy is celiac axis decompression and anti-reflux reconstruction "short floppy" R. Nissen.

The proposed algorithm for the treatment of gastroesophageal reflux disease and celiac axis compression syndrome achieved effective control of clinical manifestations of both pathological conditions in the majority (85.9%) of patients.

REFERENCES

1. Барретт Д.М. Гастроэзофагеальная рефлюксная болезнь и гастрит // Острая абдоминальная патология / Под ред. Д. Клайна, Л. Стед. – М.: БИНОМ, 2014. – С. 90–96. [Barrett DM. Gastroezofageal'naya reflyuksnaya bolezn' i gastrit. In: Ostraya abdominal'naya patologiya. Ed by D.M. Cline, L.M. Stead. Moscow: BINOM; 2014. P. 90-96. (In Russ.)]
2. Белякин С.А., Кохан Е.П., Мироненко Д.А. Хроническая абдоминальная ишемия. – М.: БИНОМ, 2014. [Belyakin SA, Kokhan EP, Mironenko DA. Khronicheskaya abdominal'naya ishemiya. Moscow: BINOM; 2014. (In Russ.)]
3. Василевский Д.И., Кулагин В.И. Хирургическое лечение гастроэзофагеальной рефлюксной болезни. – М.: СИМК, 2015. [Vasilevskiy DI, Kulagin VI. Khirurgicheskoe lechenie gastroezofageal'noy reflyuksnoy bolezni. Moscow: SIMK; 2015. (In Russ.)]
4. Власова М.И. Хирургическая анатомия чревного сплетения и обоснование техники декомпрессии чревного ствола при его экстравазальном стенозе: Автореф. дис. ... канд. мед. наук. – СПб., 2000. [Vlasova MI. Khirurgicheskaya anatomiya chrevnogo spleteniya i obosnovanie tekhniki dekompressii chrevnogo stvola pri ego ekstravazal'nom stenoze. [dissertation] Saint Petersburg; 2000. (In Russ.)]
5. Игнашов А.М. Клиника, диагностика и хирургическое лечение стеноза чревного ствола: Автореф. дис. ... д-ра мед. наук. – М.; Л., 1981. [Ignashov AM. Klinika, diagnostika i khirurgicheskoe lechenie stenoza chrevnogo stvola. [dissertation] Moscow; Leningrad; 1981. (In Russ.)]
6. Кубышкин В.А., Шумкина Л.В. Гастроэзофагеальная рефлюксная болезнь // Абдоминальная хирургия. Национальное руководство: краткое издание / Под ред. И.И. Затевахина. – М.: ГЭОТАР-Медиа, 2016. – С. 334–346. [Kubyshkin VA, Shumkina LV. Gastroezofageal'naya reflyuksnaya bolezn'. In: Abdominal'naya khirurgiya. Natsional'noe rukovodstvo: kratkoe izdanie. Ed by I.I. Zatevakhin. Moscow: GEOTAR-Media; 2016. P. 334-346. (In Russ.)]
7. Лазебник Л.Б., Машарова А.А., Бордин Д.С., и др. Многоцентровое исследование «Эпидемиология гастроэзофагеальной рефлюксной болезни в России» (МЭГРЕ): первые итоги // Экспериментальная и клиническая гастроэнтерология. – 2009. – № 6. – С. 4–12. [Lazebnik LB, Masharova AA, Bordin DS, et al. Mnogotsentrovoe issledovanie "Epidemiologiya gastroezofageal'noy reflyuksnoy bolezni v Rossii" (MEGRE): pervye itogi. *Eksp Klin Gastroenterol*. 2009;(6):4-12. (In Russ.)]
8. Семенов Д.Ю., Поташов Л.В., Цибин А.Ю., Ребров А.А. Опыт применения эндовидеохирургии при лечении больных компрессионным стенозом чревного ствола // Вестник хирургии им. И.И. Грекова. – 2008. – Т. 167. – № 5. – С. 130. [Semenov DY, Potashov LV, Tsibin AY, Rebrov AA. Opyt primeneniya endovideokhirurgii pri lechenii bol'nykh kompressionnym stenozom chrevnogo stvola. *Vestnik khirurgii imeni I.I. Grekova*. 2008;167(5):130. (In Russ.)]
9. Baccari P, Civilini E, Dordoni L, et al. Celiac artery compression syndrome managed by laparoscopy. *J Vasc Surg*. 2009;50(1):134-139. doi: 10.1016/j.jvs.2008.11.124.
10. Dunbar JD, Molnar W, Beman FF, Marable SA. Compression of the celiac trunk and abdominal angina. *Am J Roentgenol Radium Ther Nucl Med*. 1965;95(3):731-744. doi: 10.2214/ajr.95.3.731.

11. Harjola PT. A Rare Obstruction of the Coeliac Artery. Report of a Case. *Ann Chir Gynaecol Fenn*. 1963;52:547-550.
12. di Libero L, Varricchio A, Tartaglia E, et al. Laparoscopic treatment of celiac axis compression syndrome (CACS) and hiatal hernia: Case report with bleeding complications and review. *Int J Surg Case Rep*. 2013;4(10):882-885. doi: 10.1016/j.ijscr.2013.06.021.
13. Rongies-Kosmol M, Jakimowicz T. Celiac artery compression syndrome. Mini-review. *Acta Angiol*. 2015;21(1):21-24. doi: 10.5603/aa.2015.0005.

◆ Information about the authors

Dmitrij I. Vasilevsky — MD, PhD, Dr Med Sci, Associate Professor, Department of Faculty Surgery. Pavlov First Saint Petersburg State Medical University, Ministry of Healthcare of the Russian Federation, Saint Petersburg, Russia. E-mail: vasilevsky1969@gmail.com.

Stanislav G. Balandov — Surgeon, Head, Surgical Department No. 2. Pavlov First Saint Petersburg State Medical University, Ministry of Healthcare of the Russian Federation, Saint Petersburg, Russia. E-mail: stasbal@gmail.com.

Anatoly M. Ignashov — MD, PhD, Dr Med Sci, Professor, Department of Faculty Surgery. Pavlov First Saint Petersburg State Medical University, Ministry of Healthcare of the Russian Federation, Saint Petersburg, Russia. E-mail: a.m.ignashov@yandex.ru.

Kristina A. Anisimova — Surgeon of the Surgical Department No. 2. Pavlov First Saint Petersburg State Medical University, Ministry of Healthcare of the Russian Federation, Saint Petersburg, Russia. E-mail: anisimova-k-a@mail.ru.

Leysan I. Davletbaeva — Resident of the Department of Hospital Surgery No. 2 with a Clinic. Pavlov First Saint Petersburg State Medical University, Ministry of Healthcare of the Russian Federation, Saint Petersburg, Russia. E-mail: davletbaeva-leysan-i@yandex.ru.

◆ Информация об авторах

Дмитрий Игоревич Василевский — д-р мед. наук, доцент, кафедра факультетской хирургии. ФГБОУ ВО «Первый Санкт-Петербургский государственный медицинский университет им. акад. И.П. Павлова» Минздрава России, Санкт-Петербург. E-mail: vasilevsky1969@gmail.com.

Станислав Георгиевич Баландов — врач-хирург, заведующий, хирургическое отделение № 2. ФГБОУ ВО «Первый Санкт-Петербургский государственный медицинский университет им. акад. И.П. Павлова» Минздрава России, Санкт-Петербург. E-mail: stasbal@gmail.com.

Анатолий Михайлович Игнашов — д-р мед. наук, профессор, кафедра факультетской хирургии. ФГБОУ ВО «Первый Санкт-Петербургский государственный медицинский университет им. акад. И.П. Павлова» Минздрава России, Санкт-Петербург. E-mail: a.m.ignashov@yandex.ru.

Кристина Александровна Анисимова — врач-хирург, хирургическое отделение № 2. ФГБОУ ВО «Первый Санкт-Петербургский государственный медицинский университет им. акад. И.П. Павлова» Минздрава России, Санкт-Петербург. E-mail: anisimova-k-a@mail.ru.

Лейсан Индусовна Давлетбаева — ординатор, кафедра госпитальной хирургии № 2 с клиникой. ФГБОУ ВО «Первый Санкт-Петербургский государственный медицинский университет им. акад. И.П. Павлова» Минздрава России, Санкт-Петербург. E-mail: davletbaeva-leysan-i@yandex.ru.