Original Article

PROGRESSIVE FAILURE AFTER GASTRIC AND DUDEON RESECTION

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Abstract

The scientific literature reviews on the origin of anemia and the development of anemia after gastric and duodenal resection, the state of advanced hyperchromic anemia, both experimental and clinical observations on the possibility of developing forms of anemia are given in this article.

Keywords: gastric, duoden, resection, anemia, surgical treatment.

INTRODUCTION

As we know from the history of medicine, there are many recommendations for the prevention and treatment of anemia caused after gastric ulcer and duodenal resection operations, which have been proven in scientific literature. We will touch on some of them below. In experimental studies, pernicious anemia develops when a significant part of the stomach is removed, because the anti-anemic factor of Castle, which is associated with the origin of anemia, is also produced in the pyloric and cardiac glands [1]. Partial resection of Bruner's glands of the stomach may not lead to advanced anemia because it does not completely eliminate the anti-anemic factor. The same thing was observed when the entire stomach of pigs was removed, hypochromic anemia at first and hyperchromic megalocytic anemia after 2-3 years. Some authors associate the origin of the antianemic factor with certain sections of the stomach: entrance to the stomach, lower and pyloric part [2, 3]. Dedihen observed 164 patients with anemia after gastric resection for 15 years, mostly of the hypochromic type, but rarely of the hyperchromic type.

Also, according to Billrot I, the percentage of anemia after resection is the smallest, and after Billrot II - especially when modified according to Pollna - the percentage increases significantly, which the author attributes to the faster passage of food through the stomach calculates [4].

Some have pointed to four case descriptions of true pernicious anemia after resection to support the possibility of developing pernicious anemia after gastrectomy. When observing 25 patients who underwent gastric resection, anemia was present in almost all cases, often hypochromic, rarely hyperchromic. In one case, a transition from hypochromic to hyperchromic anemia was observed [5].

Gastric ulcer affects 8-15% of adults in many countries. They are mostly people of working age. The highest level of duodenal ulcer appears in 30-40 years, stomach ulcer in 50-60 years. Men suffer from duodenal ulcer 4-10 times, and gastric ulcer 6-27 times more than women [7, 8, 9]. According to the received statistics, only 10% of patients with stomach ulcers have the opportunity to be treated regularly with adequate drugs, as well as diet and nutrition [10].

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Access this article online

Quick Response Code:

Website: www.pnrjournal.com

DOI: 10.47750/pnr.2022.13.04.154

How to cite this article: I B Makhamatibrokhimovich, I B Uktamovich, Q S Nomonovich, PROGRESSIVE FAILURE AFTER GASTRIC AND DUDEON RESECTION, J PHARM NEGATIVE RESULTS 2022;13: 1113-1117.

A gradual aging of the contingent of patients with peptic ulcer is noted. However, the study of data from foreign literature does not allow to observe a clear trend. Many authors have shown in their scientific experiments about the increase in the number of complex forms of gastric ulcer [11, 12, 13, 14, 15]. However, a number of researchers have noted a decrease in the number of punctured and bleeding wounds in recent years [16, 17, 18, 19, 20]. Other authors report the relative stability of these indicators [21, 22]. Gastric ulcer pathogenesis is complex and has many factors. Currently, the infectious factor is of great importance. Many authors agree that Helicobacter pylori infection plays a leading role in the etiology and pathogenesis of gastroduodenal ulcers [23, 24, 25]. As a proof, there are observations showing faster and more effective repair of wound defects in patients who received eradication therapy [6, 26]. However, the etiological role of Helicobacter pylori in the development of gastric ulcer and gastric cancer is not proven [27]. According to information, 70% of Helicobacter pylori carriers do not develop ulcers during their lifetime, and artificial infection does not cause ulcers. According to the literature, the percentage of duodenal ulcer is 20-38%, and gastric ulcer is 40-56% [28, 29]. At the same time, microbiological research results show an increase in the number of antibiotic-resistant strains, which may be the result of the widespread introduction of eradication treatment regimens [30]. In this regard, the question arises about the expediency of empirical therapy against Helicobacter in the treatment of gastric ulcer. According to the recommendations of the Maastricht IV (Maastricht IV) international reconciliation conference held in 2010, it is stated that treatment of Helicobacter pylori infection should be carried out only when it is detected [31, 32, 33].

Currently, the role of non-steroidal anti-inflammatory drugs (NSAID) in the development of gastric ulcer is of great importance. NSAID also have direct toxic effects on the gastric mucosa and duodenum, as well as indirect cyclooxygenase blockade [34, 35]. In this direction, the following special terms have appeared: " NSAID - gastropathy", "NSAID related ulcer", "NSAID gastropathy" includes erosions and ulcers of the stomach and / or duodenum. Damage to the mucous membrane of the upper gastrointestinal tract with the development of life-threatening complications such as gastrointestinal bleeding and perforation is known in science. The risk of developing gastroduodenal ulcers with the use of NSAID is especially high in elderly patients. 34.6% of acute gastroduodenal bleeding is caused by NSAID. The use of selective cyclooxygenase-2 inhibitors in combination with proton pump inhibitors or misoprostol is recommended to reduce the risk of NSAID gastropathy [36]. Dysfunction of the autonomic nervous system plays a certain role in the pathogenesis of gastric ulcer. It is noted that the increased tone of the vagus nerve in patients with duodenal ulcer causes hypermotility and high acidity of gastric secretion. An increase in the activity of the sympathetic nervous system contributes to a decrease in gastric motility and creates the necessary conditions for the development of gastric ulcer. The functional imbalance of the autonomic nervous system is mainly formed under the influence of mental factors.

One of the pathogenetic factors of gastric ulcer development is chronic duodenal obstruction. Chronic duodenal obstruction occurs in the sub- and decompensation phase. Atrophy of the stomach epithelium and a decrease in mucus production are observed. Chronic duodenal obstruction is found in less than 50% of patients with duodenal ulcer and less than 20% of patients with peptic ulcer. NSAID aggravates gastric ulcer, but is not the direct cause of its development. Many authors recognize the leading role of acid-peptic factor in the pathogenesis of duodenal ulcer [27, 37, 38].

Research data from different years show that there is a direct relationship between the genetically determined increase in the secretion of pepsinogen I and the high acidity of gastric juice [39, 40, 41, 42]. V.M. Lobankov and M.N. Kambalov studied gastric ulcers in twins and concluded that genetic predisposition determines the development of the disease more than the influence of lifestyle and exogenous factors [43].

According to other authors, the majority of operations for stomach ulcers (87.2 - 96.8%) are performed as an emergency and urgent for its complications [17, 44]. M.I. Kuzin et al. (2002) believe that the following absolute indications for surgical treatment of gastric ulcer are distinguished: wound perforation, heavy and repeated bleeding, impaired evacuation function. Yu.B. Martov et al. (1995) cite as relative indications for surgery the penetration of gastric and duodenal ulcers into adjacent organs, as well as the presence of Johnson type II or III ulcers. According to the authors' observations, pyloric ulcers are difficult to treat and are usually treated with stenosis formation. The simultaneous presence of ulcers in the duodenum and stomach indicates a serious violation of the patency of the duodenum, which requires surgical treatment. Many authors believe that all patients with gastric ulcer complicated by duodenal cancer should undergo surgery [27, 38]. The use of anti-ulcer drugs allows successful conservative treatment of compensated duodenal cancer.

One of the absolute indications for surgery is persistent bleeding from stomach and duodenal ulcers or its recurrence in the hospital [17, 29]. The possibility of endoscopic hemostasis is considered ineffective in emergency use at the height of bleeding. At the same time, patients with unstable hemostasis are recommended for urgent surgery within 2-24 hours. Various algorithms have been developed to objectively assess the risk of recurrent bleeding [45, 46, 47]. At the same time, many authors agree that urgent surgical intervention at the height of bleeding is recommended only against the background of complete infusion, transfusion and hemostatic therapy, in case of ineffective endoscopic hemostasis or in cases of repeated bleeding [8, 19, 33].

In full conservative treatment, surgical treatment is recommended for chronic stomach ulcers that do not heal for about 3-4 months, as well as when metaplasia and dysplasia are detected at the edge, bottom or scar of a healed ulcer. Continuation of conservative treatment and dynamic monitoring in this category of patients is dangerous not only because of the possibility of malignancy of gastric ulcer, but also because of the complexity of differential diagnosis of chronic ulcer and primary ulcerative form of cancer [31]. I.M. According to Selivanova (2006), in 26.5% of patients operated on chronic gastric ulcer, early cancer at the edge of chronic ulcer, 8.2% in the scar, precancerous changes in the gastric mucosa - in 83% [48].

A number of authors believe that it is necessary to expand the indications for surgical treatment of peptic ulcer with the ineffectiveness of complete antisecretory and antibacterial therapy. Planned surgical treatment of patients with a long history of peptic ulcer disease prevents the development of complications after the disease, such as bleeding, perforation, penetration, and duodenal cancer. Suturing of a perforated ulcer of the duodenum or patients who experienced ulcer bleeding, as well as in the absence of a permanent effect of conservative treatment carried out for 3 years or more, should be operated on a planned basis. In patients with perforated wound closure, surgical treatment is one of the best indications for wound recurrence [12].

When choosing a surgical method and size for gastric ulcer complications, it is recommended to follow the following criteria: type, localization and size of the ulcer; acid production rate; morphofunctional condition of esophagus, stomach and duodenum; dynamics of pathomorphological changes in the gastric mucosa. The choice of the surgical method depends on the level of gastric secretion, the tendency to develop functional disorders, the patient's condition, anatomical and morphological conditions. Many authors believed that gastric resection is a suitable option for peptic ulcer. In order to adequately suppress acid production in gastric ulcers, it is recommended that the distal resection of the stomach be at least 2/3, and with duodenal ulcers at least ¾. In the case of type I ulcers, it is stated in the scientific literature that it is appropriate to resect ½ part of the stomach in order to ensure the sufficient length of its tail region [33, 42].

Some authors note the advantages of gastric resection according to Billrot I compared to Billrot II and show the possibility of performing it in any localization of the wound. However, when it is complicated by large and large postbulbar ulcers, especially when they penetrate into the hepatoduodenal ligament and the pancreas, the surgeon is forced to perform gastric resection according to the second Billrot method. Roux-en-Y distal gastric resection combined with trunk vagotomy is considered the most effective operation for low, giant, penetrating duodenal ulcers. M.I. Kuzin et al (2002) recommend selective proximal vagotomy without pyloroplasty as the standard operation for duodenal ulcers unresponsive to conservative treatment [6].

Suturing is the most effective operation of choice for a perforated duodenal ulcer. It has been found that when patients with a perforated wound are sutured, they can reduce the number of recurrences from 62% to 19% when they are followed up and treated completely. Plastic closure of a perforated wound with a larger omentum reduces the risk of failure and postoperative mortality even in patients with a giant ulcer (more than 2 cm in diameter), while gastric resection shows a significantly higher number of complications and mortality. Recurrence of the wound occurs in 36.3 - 64.6% of cases, repeated surgical treatment is required in 21.5 - 54.1% of patients. Analyzing the long-term results of 986 patients with perforated ulcers, it was found that the highest probability of recurrence occurred approximately 1 year after the wound was sutured. In this regard, the authors recommend monitoring and prophylactic treatment for this category of patients for 1 year after surgery. According to the results of the study, one of the reasons for the recurrence of wounds after suturing is considered to be residual ligatures. In this regard, it is recommended to use absorbable sutures when closing a punctured wound [9].

The experimentally developed and clinically introduced method of closing a perforated wound with a two-level suture without touching the mucosa without plastic tension. Suturing of a perforated duodenal ulcer should be completed with a proximal vagotomy to prevent its recurrence. Such work is carried out in patients with chronic wounds with stable hemodynamics within 24 hours from the time of perforation.

According to other researchers, eradication therapy after ulcer closure is more effective and safer than vagotomy in preventing recurrence. Many modern surgeons adhere to the tactics of a differential approach to closing perforated wounds, taking into account the diameter of the wound, its localization and the stage of peritonitis. Thus, they recommend that gastric resection for a perforated ulcer should be performed according to strict guidelines and under appropriate conditions. As absolute indicators, large, giant and callous ulcers, bilateral localization ulcers, re-perforation of a previously sutured wound, as well as two or more complications of a gastric ulcer (bleeding, penetration) are indicated [32].

Gastric resection is an indication for cutting the wound with pyloroplasty and vagotomy in case of giant, mobile or penetrating ulcer, clear ulcer infiltrate, inability to rule out a gastric tumor, as well as the release of ligatures during suturing. In recent years, laparoscopy has been successfully used in the treatment of patients with perforated ulcers. It has been proven in practice that this modern method allows to reduce the invasiveness and the number of postoperative complications, to accelerate the physical and social rehabilitation of patients. For laparoscopic closure of perforated wounds, he proposed the use of spring-loaded metal clamps of an original design, tested in an experiment

and successfully used in the treatment of 26 patients. E.A. Baranov (2008) performs laparoscopic vagotomy and mini-access pyloroplasty with wound excision to treat a complex duodenal ulcer [50].

When bleeding from a stomach ulcer, the main operation is a gastric resection. After gastric resection for bleeding peptic ulcer, the number of relapses is half that after organ-sparing interventions (2.9% and 6.2%, respectively). At the same time, performing radical operations on a patient with severe wound bleeding poses a great risk. The number of complications after emergency gastric resection is 2 times higher, and mortality is 4 times higher after vagotomy with pyloroplasty. Recommends vagotomy with pyloroplasty in the treatment of duodenal bleeding. According to the authors, this operation is technically simpler, less traumatic, and is accompanied by less death than gastrectomy [51]. Complications of gastrectomy M. Merenda et al. (2011) distinguish the following groups of complications after gastric resection: general (cardiopulmonary failure, cardiac arrhythmia, exudative pleurisy, psychosis) and surgery. Among surgical complications, the authors distinguish abdominal (anastomotic insufficiency, acute pancreatitis) and those associated with wound infection. In the early postoperative period after gastric resection, abdominal complications include: bleeding, insufficiency of anastomoses and duodenal sutures, intestinal obstruction, impaired evacuation from the gastric cavity, postoperative pancreatitis. Early complications of gastrectomy consider the most severe insufficiency of the duodenum according to Billrot II, as well as evacuation disorders caused by edema and spasm of the anastomosis. Duodenal insufficiency after gastrectomy according to Billrot II is 1-10% [52].

After gastric resection and emergency gastrectomy for peptic ulcer, anastomotic insufficiency develops in 4.5 - 7.7%. Complications of the early postoperative period include: bleeding from the vessels of the stomach wall and small omentum, necrosis of the small curvature, acute complicated gastric ulcer, bleeding from the lower part of the penetrating wound, gastric atony, dysphagia. E.A. According to Baranova (2008), after vagotomy with urgently performed pyloroplasty, gastrostasis is 5.9 - 6.7%, postoperative pneumonia - 3.6%, wound suppuration - 5.1%, Recurrence of duodenal ulcer bleeding - 3%, suture - 07%, pyloroplasty failure - 0.73% of patients were found in research [53]. According to foreign authors, about 4% of organ preservation operations are complicated by failure of pyloroplasty sutures [54].

Conclusion

Thus, the prevalence of peptic ulcer determines its social importance. The development of this disease is facilitated by a combination of various factors, including infectious, medicinal, neurovegetative, humoral.

The initial factor for vegetative disorders can be severe experiences, shocks, anxiety and excitement regularly experienced by the patient.

Disruption of gastric evacuation function is the most frequent among the initial complications of vagotomy. It can be said that the duration of gastrostasis in patients who underwent surgery for bleeding and perforation of the wound does not depend on the pyloroplasty method, but is determined by the degree of vagotomy and the spread of peritonitis.

Improvement of the technique of performing primary operations with gastric ulcer complications, creation of safe biological materials for operations in conditions of impaired blood supply to the walls of the gastrointestinal tract, development of methods for early diagnosis of the duodenum, anastomoses, among the recommended methods the accumulation of clinical experience will determine the future of our further clinical studies.

References

1. Мейленграхт, Центр, м. ж., 19, 1938.
2. Gutze (t, Miinch. med.Woch. 10, 1932.
3. Gutreit, Klin. Woch. 2, 1932.
4. Дедихен, Центр, м. ж. 2, 1935.
5. Гукасян Смотров и Судакова, Клин. мед. 18 — 1, 1940.
6. Кузин, М.И. Язвенная болезнь желудка и двенадцатиперстной кишки / М.И. Кузин, М.А. Чистова // Хирургические болезни: учебник / под ред. М.И. Кузина. – М.: Медицина, 2002. – С. 281–316.
7. Yousaf F, Spinowitz B, Charytan C, Galler M. Pernicious Anemia Associated Cobalamin Deficiency and Thrombotic Microangiopathy: Case Report and Review of the Literature. Case Rep Med.2017:9410727.
8. Григорян, Р.А. Абдоминальная хирургия: в 2 т. Т. 1 / Р.А. Григорян. – М., 2006. – 608 с.
9. WHO. Iron Deficiency Anemia: Assessment, 405 Prevention and Control—A Guide for Programme Managers. 2001. Available online: https://www.who.int/publications/m/item/iron-children-6to23--archived-iron-deficiency-anaemia-assessment-prevention-and-control (accessed on 3 May 2022).
10. Das, A.; Das, M.; Ghosh, S. Impact of nutritional status and anemia on COVID-19: Is it a public health concern? Evidence from National Family Health Survey-4 (2015–2016), India. Public Health 2020, 185, 93–94.
11. Курыгин, А.А. Хирургические заболевания желудка и двенадцатиперстной кишки//Госпитальная хирургия. Руководство для врачей-интернов – СПб.: Лань, 2005. – 896 с.
12. Чернышев, В.Н. Экстренная хирургия осложнений гастродуоденальных язв. Лекции для хирургов при последипломной подготовке – Самара: СамЛюксПринт, 2012. – 110 с.
13. Афендулов, С.А. Лечение прободной язвы – М., 2005. – 166 с.
14. Лобанков, В.М. «Хирургическая эпидемиология» язвенной болезни в период с 1990-го по 2004 год – 2007. – № 1. – С. 50–55.
15. Бородин, Н.А. Послеоперационные осложнения хирургического лечения язвенной болезни желудка и двенадцатиперстной кишки, пути снижения летальности: автореф. дис. … д-ра мед. наук / – Тюмень, 2008 – 43 с
16. Хирургическое лечение перфоративных язв у женщин // Альманах Института хирургии им. А.В. Вишневского. – 2015. – № 2. – С. 410–411.
17. Lassen, A. Complicated and uncomplicated peptic ulcers in a Danish county 1993– 2002: a population-based cohort study // American Journal of Gastroenterology. – 2006. – Vol. 101. – P. 945–953.
18. , P.N. Declining incidence of peptic ulcer but not of its complications: a nation wide study in The Netherlands // Aliment. Pharmacol. Ther. –

2006. – Vol. 23(11). – P. 1587–1593.
19. Зайцев, О.В. Изменение распространенности осложнений язвенной болезни за период 1995-2009 гг. – 2011. – № 4. Режим доступа: http://cyberleninka.ru/article/n/izmenenie-rasprostranennosti-oslozhneniy yazvennoy-bolezni-za-period-1995-2009-gg, свободный. – Загл. с экрана. – (дата обращения: 12.05.2014).
20. Выбор метода резекции желудка в условиях хронической дуоденальной непроходимости // Вестник экспериментальной и клинической хирургии. – 2015. – Т. 3, № 4. – С. 335–338.
21. Acute upper GI bleeding: did anything change? Time trend analysis of incidence and outcome of acute upper GI bleeding between 1993/1994 and 2000 // American Journal of Gastroenterology. – 2003. – Vol. 98. – P. 1494–1499.
22. Admission rates for peptic ulcer in the Trent region, UK, 1972–2000. Changing pattern, a changing disease? // Digestive Liver Disease. – 2004. – Vol. 36. – P. 577–588.
23. Systematic Review of the Epidemiology of Complicated Peptic Ulcer Disease: Incidence, Recurrence, Risk Factors and Mortality // Digestion. – 2011. – Vol. 84, № 2. – P. 102–113.
24. Canoy, D.S. Epidemiology of duodenal ulcer perforation: a study on hospital admissions in Norfolk, United Kingdom // Dig. Liver Dis. – 2002. – Vol. 34, № 5. – P. 322–327.
25. Changes in aetiology and clinical outcome of acute upper gastrointestinal bleeding during the last 15 years // Eur. J. Gastroenterol. Hepatol. – 2004. – Vol. 16. – P.177–182.
26. Денисова, Е.В. Статистический анализ многолетней заболеваемости как метод оценки эффективности эрадикационной терапии в лечении язвенной болезни и ее осложнений – 2012. – № 144. – С. 66–75.
27. Peek, R.M.Jr. Pathophysiology of Helicobacter pylori-induced Gastritis and Peptic Ulcer Disease // American Journal of Medicine. – 1997. – Feb., Vol. 102(2). – P. 200–207.
28. Debas, H.T. Gastrointestinal Surgery Pathophysiology and Management – N.Y.: Springer-Verlag, 2004. – 391 p.
29. Dramatic Decline in Prevalence of Helicobacter pylori and Peptic Ulcer Disease in an Endoscopy-referral Population // The American Journal of Medicine. – 2011. – Vol. 124, № 3. – P. 260–264.
30. Циммерман, Я.С. Нерешенные и спорные проблемы современной гастроэнтерологии – М.: Медпресс-информ, 2013. – 244с.
31. Баранская, Е.К. Язвенная болезнь и инфекция Helicobacter pylori // Русский медицинский журнал. – 2000. – № 1. – С. 8–14.
32. Минушкин, О.Н. Современный взгляд на проблему эрадикации Helicobacter pylori // Практикующий врач. – 2002. – № 1. – С. 52–54.
33. Маев, И.В. Антибиотикорезистентность Helicobacterpylori: от клинического значения до молекулярных механизмов – 2014. – № 2. – С. 34–39.
34. Исаков, В.А. Диагностика и лечение инфекции, вызванной Helicobacter pylori: IV Маастрихтское соглашение // Best Clinical Practice. Русское издание. – 2012. – Вып. 2. – С. 4–23.
35. Вялов, С.С. Язвенная болезнь и Маастрихт-4: внедрение в клиническую практику // Гастроэнтерология. – 2012. – № 6. – С. 16–22.
36. The European Helicobacter Study Group (EHSG). Management of Helicobacter pylori infection – the Maastricht IV/ Florence Consensus Report // Gut. – 2012. – Vol. 61. – P. 646–664.
37. Лапина, Т.П. Гастропатия, индуцированная нестероидными противовоспалительными препаратами: пути решения проблемы // Русский медицинский журнал. – 2009. – Т. 11, № 2. – С. 54–57.
38. Карасева, Г.А. НПВП-индуцированная гастропатия: от понимания механизмов развития к разработке стратегии профилактики и лечения // Медицинские новости. – 2012. – № 8. – С. 21–26.
39. Roth, S.H. Nonsteroidal anti-inflammatory drug gastropathy: new avenues for safety // Clinical Interventions in Aging. – 2011. – № 6. – P.125–131.
40. Ю.Б. Мартов, В.В. Аничкин, С.Г. Подолинский [и др.]. Язвенная болезнь глазами хирурга //– Витебск, 1995. – 269 с.
41. Хавкин, А.И. Современные принципы терапии язвенной болезни – 2005. – № 2. – С. 30– 33.
42. Физиологическое и клиническое значение пепсиногенов желудка // Клиническая медицина. – 2014. – Т. 92, № 3. – С. 26–30.
43. Carvalho de, A.S.T. Peptic ulcer // Journal de Pediatria. – 2000. – Vol. 76, supl. 2. – P. 127–134.
44. Alteration of correlation between serum pepsinogen concentrations and gastric acid secretion after H. pylori eradication // Journal of Gastroenterology. – 2009. – Vol. 44(8). – P. 819–825.
45. The value of serum pepsinogen levels for the diagnosis of gastric diseases in Chinese Han people in midsouth China // BMC Gastroenterology. – 2014. – № 3. – P. 1–6.
46. Лобанков, В.М. Клинические варианты язвенной болезни у близнецов // Вестник хирургической гастроэнтерологии. – 2008. – № 3. – С. 53–55.
47. В.В. Замятин, В.И. Белоконев, В.П. Афанасенко [и др.]. Основные статистические показатели организации помощи больным с язвенной болезнью в Самарской области Материалы III межобластной конф. хирургов Бугуруслан, 2002. – Казань, 2003. – С. 3–15.
48. Евсеев, Е.М. Антисекреторные препараты в неотложной хирургической гастроэнтерологии – М.: Кван, 2009. – 180 с.
49. Корытцев, В.К. Кровотечение из язвы двенадцатиперстной кишки. Прогнозирование течения и лечебная тактика у больных с остановившимся кровотечением: автореф. дис. … д-ра мед. наук / – Самара, 2009. – 40 с.
50. Козлов, А.А. Тактика хирурга в лечении больных с перфорациями хронических пилородуоденальных язв: автореф. дис. … канд. мед. наук – Самара, 2011. – 25 с.
51. Селиванова, И.М. Радикальное хирургическое лечение раннего рака желудка: дис. … канд. мед. наук. – М., 2006. – 133с.
52. M. Merenda, D. Janczak, J. Cianciara [et al.]. Acute Pancreatitis as an Early Complication after Gastric Resection // Polish Journal of Surgery. – 2011. –Vol. 82, № I.12. – P. 645–650.
53. Баранов, Е.А. Непосредственные и отдалённые результаты малоинвазивных эндохирургических вмешательств у больных с осложнённой язвенной болезнью двенадцатиперстной кишки: автореф. дис. … канд. мед. наук. – М., 2008. – 14 с.
54. Pappas, T.N. Historical aspects, anatomy, pathology, physiology, and peptic ulcer disease – Philadelphia: Saunders, 1997. – P. 847–868.