J Gastrointest Surg (2014) 18:1642–1647
DOI 10.1007/s11605-014-2579-0

ORIGINAL ARTICLE

Clinical Outcomes and Prognostic Factors After Surgery for Non-Occlusive Mesenteric Ischemia: a Multicenter Study

Takafumi Yukaya · Hiroshi Saeki · Kenji Taketani · Koji Ando · Satoshi Ida · Yasue Kimura · Eiji Oki · Mitsuhiro Yasuda · Masaru Morita · Ken Shirabe · Yoshihiko Maehara

Received: 9 January 2014 / Accepted: 11 June 2014 / Published online: 3 July 2014

Abstract
Background To date, no large-scale study has been undertaken to understand the clinical features of non-occlusive mesenteric ischemia (NOMI) after surgery. We thus performed a multicenter investigation to clarify the clinical outcomes and prognostic factors of NOMI.
Patients and Methods Clinical databases from 22 Japanese facilities were reviewed for evaluation of patients who received surgery for NOMI between 2004 and 2012. NOMI patients (n=51) were divided into two groups: group I (n=28) consisted of patients who survived, and group II (n=23) consisted of patients who did not survived. Prognostic factors were compared between the two groups.
Results NOMI surgery represented 0.04 % of the total number of operations performed in this time period. The overall mortality rate for NOMI surgery was 45 %. Hemodialysis was a significant negative prognostic factor (p=0.027). Preoperative elevation of transaminases, potassium, and white blood cell count, as well as metabolic acidosis and colon ischemia was poor prognostic factors. The mean Physiological and Operative Severity Score for the enumeration of Mortality and Morbidity (POSSUM) score of group I versus group II was 54.5±3.6 and 85.2±4.1, respectively (p<0.001).
Conclusions Currently, NOMI surgery has a 45 % mortality rate. POSSUM scores can be used to predict the clinical outcome of patients who receive NOMI surgery.

Keywords Non-occlusive mesenteric ischemia · Acute mesenteric ischemia · Prognostic factor · Clinical features

Introduction

Non-occlusive mesenteric ischemia (NOMI) consists of intestinal ischemia and/or necrosis in the absence of an organic obstruction within the main trunk of the mesenteric artery or vein.[1] It is currently thought that NOMI is caused by mesenteric vasoconstriction.[2] NOMI has been reported to be the cause of 10 to 30 % of all cases of acute mesenteric ischemia.[2,3] Until recently, NOMI has had a dismal prognosis, with mortality rates between 70 and 90 %.[2,3] NOMI is associated with age, diabetes mellitus, hypertension, and altherosclerosis,[3,4] suggesting that it is a problem of aging.

Selective mesenteric angiography is considered the gold standard for diagnosing acute mesenteric ischemia,[5] and the American Gastroenterological Association has established guideline for the diagnosis and treatment of acute mesenteric ischemia.[6] However, debate regarding angiography and arterial infusion therapy for NOMI exists,[3,7] and the role of surgical treatment for NOMI is controversial.[3,6–9]

Current understanding of NOMI is based on a small number of clinical reports of patients with miscellaneous forms of acute mesenteric ischemia. The largest study to date on patients with a definitive NOMI diagnosis was performed by Ward et al.[9] who reported on 34 patients with NOMI. The aim of this study was to use a multicenter approach to clarify the clinical outcomes and prognostic predictive factors of NOMI.

T. Yukaya (✉) · H. Saeki · K. Taketani · K. Ando · S. Ida · Y. Kimura · E. Oki · M. Yasuda · M. Morita · K. Shirabe · Y. Maehara
Department of Surgery and Science, Graduate School of Medical Sciences, Kyushu University, 3-1-1 Maidashi, Higashi-ku, 812-8582 Fukuoka, Japan
e-mail: t-yukaya@surg2.med.kyushu-u.ac.jp

Patients and Methods

A retrospective chart review was performed on all patients who underwent surgery at the Department of Surgery and Science, Graduate School of Medical Sciences, Kyusyu University, and 21 related facilities between April 2004 and September 2012. During this period, 114,224 operations (including 12,388 emergency operations) were performed by the 22 institutes. Among them, 51 operations were performed on NOMI patients (0.04 %).

The diagnosis of NOMI was based on operative findings. A definitive diagnosis of NOMI requires the absence of an organic obstruction of the blood vessels distributed in the necrotic intestinal region, segmented discontinuous intestinal ischemic changes, and necrosis.[9–11]

In this study, physicians were asked to fill out a survey form consisting of the following items: patient background [gender, age, underlying disease, surgical division (emergency or scheduled)], laboratory findings at the time of the decision to proceed to surgery, metabolic acidosis upon admission, preoperative hypotension, portal venous gas detected by CT scan, range of ischemic lesion, POSSUM score (predictive mortality rate), additional postoperative treatments (such as prostaglandin E1, continuous hemodiafiltration, polymyxin B-immobilized column direct hemoperfusion, anticoagulant therapy, nitrovasodilators, and octreotide), and prognosis.

NOMI patients were classified into two groups: group I (n=28) consisting of patients who survived to discharge and group II (n=23) consisting of patients who did not. Multiple clinical factors were compared between the two groups.

POSSUM stands for Physiological and Operative Severity Score for the enumeration of Mortality and Morbidity.[12] It was developed by Copeland et al.[12] in 1991 and has since been applied to a number of surgical groups including orthopedics, vascular surgery, head and neck surgery, and GI/colorectal surgery. The POSSUM mortality equation is calculated as follows: $\ln [R / (1 - R)] = -7.04 + (0.13 \times \text{physiological score}) + (0.16 \times \text{operative severity score})$, where R is the predicted risk of mortality.[12]

Statistically significant differences were determined using Fisher's exact test and t test. A p value less than 0.05 was considered to be statistically significant. Receiver operator characteristic (ROC) curves and the corresponding area under the curve (AUC) were used to evaluate how the prediction model performed on the study data.

Results

Characteristics and Symptoms of NOMI Patients

A comparison of patient characteristics is shown in Table 1. Males accounted for 51.0 % of all NOMI patients. The median patient age was 78 years (19–94 years), with 19 patients in their 80s. The overall mortality rate was 45 % (n=23). There were no significant differences in gender or age between the two groups.

Hypertension was the most frequent comorbid condition (49 %), followed by cardiovascular disease (47 %) and renal failure (14 %). Four patients were taking digitalis (8 %). There were no significant differences in comorbid conditions (hypertension, cardiovascular disease) or medications between the two groups. However, group II contained significantly more patients on hemodialysis (p=0.027).

Several presenting symptoms were identified. Abdominal pain was the presenting symptom in 30 patients (59 %) and loss of consciousness in 9 (18 %). Seven patients (14 %) were sedated and thus had no presenting complaint. Hematemesis/bloody stool and abdominal distention were present in each

Table 1 Comparison of clinical characteristics between group I and group II

Factors	All (n =51)	Group I (n=28)	Group II (n=23)	p value
Average age	78	75.5±2.3	76.1±2.6	0.856
Gender (Male/Female)	26/25	16/12	10/13	0.404
Comorbid conditions				
Hypertension	25 (49)	14 (50)	11 (50)	0.551
Diabetes mellitus	10 (20)	5 (18)	5 (22)	0.500
Cardiovascular disease	24 (47)	16 (57)	8 (35)	0.970
Ischemic heart disease	11 (22)	7 (25)	4 (17)	0.621
Atherosclerosis	7 (14)	6 (21)	1 (4)	0.112
Arrhythmia	7 (14)	6 (21)	1 (4)	0.112
Congestive heart failure	6 (12)	4 (14)	2 (9)	0.854
Hemodialysis	7 (14)	1 (4)	6 (27)	0.027
Medications				
Digitalis	4 (8)	1 (4)	3 (13)	0.234
Diuretics	10 (20)	9 (32)	1 (4)	0.015

Data listed for comorbid conditions and medications are total number (%)

Group I patients who survived to discharge, *group II* patients who did not survive to discharge

four patients. Finally, 16 patients (32 %) reported no abdominal symptoms.

Laboratory and Radiological Findings of NOMI Patients

A comparison of laboratory findings is shown in Table 2. Aspartate aminotransferase, alanine aminotransferase, potassium, and white blood cell count were higher in group II than those in group I ($p<0.05$).

Metabolic acidosis was present in 29 % of the patients in group I and in 72 % of the patients in group II ($p=0.017$; Table 3). Only one patient underwent angiography, whereas 46 patients underwent computed tomography for preoperative diagnosis. No patient required CT angiography. Portal venous gas was detected in 62 % in group I but in only 30 % of the patients in group II ($p=0.033$). Extensive ischemia (small intestine and colon) was observed in 25 % of the patients in group I and in 61 % of patients in the group II ($p=0.010$). No significant differences were observed in the incidence of preoperative hypotension, the operation time, or the amount of blood lost between the two groups.

Surgery Performed for NOMI

The surgeries performed on the NOMI patients are listed in Table 4. Thirty-two patients (63 %) underwent bowel resection with enterostomy, and eight patients (16 %) underwent intestinal resection with reconstruction. Exploratory laparotomy was performed for 11 patients, 4 of whom (8 %) had no evidence of necrosis and 7 of whom (14 %) had massive necrosis requiring resection. Three patients underwent a second operation for additional bowel resection, because ischemic progression was suspected. Median operation time was 152.6±12.6 min, and median blood loss was 837.7±397.8 mL.

Additional Postoperative Treatments and POSSUM Score

Additional postoperative treatments for NOMI patients are shown in Table 5. Ten patients were treated with prostaglandin E1, nine with continuous hemodiafiltration, three with polymyxin B-immobilized column direct hemoperfusion, and three with anticoagulation therapy. Prostaglandin E1 is a vasodilator; it was used to prevent vasospasm. The first line therapy upon suspicion of NOMI has been angiography and continuous administration of vasodilators, prostaglandin E1, and papavelin. Mitsuyohi et al. reported that high dose intravenous administration of PGE1 was effective in NOMI.[3] Continuous hemodiafiltration was used for remove of inflammatory cytokines and renal replacement.[13] Direct hemoperfusion with polymyxin B-immobilized column was used for remove of endotoxin.[14] The percent of NOMI patients who underwent additional postoperative treatment was 61 % in group I and 26 % in group II ($p=0.014$).

The mean POSSUM scores of groups I and II were 54.5±3.6 and 85.2±4.1, respectively ($p<0.001$; Table 3). All patients with a POSSUM score over 90 were in group II. Furthermore, 22 of the 25 patients with a POSSUM score under 76.1 were in group I. Group I contained a higher percentage of patients with POSSUM scores between 76.1 and 90 who had received additional postoperative treatment ($p=0.024$; Table 6). The treatments were as follows: one patient was treated with anticoagulant therapy, one was treated with direct hemoperfusion with polymyxin B-immobilized column,

Table 2 Comparison of preoperative laboratory findings between group I and group II

Factors		All (n =51)	Group I (n=28)	Group II (n=23)	p value
Serum aspartate aminotransferase	(IU/L)	39 (12–6715)	60±190	738±215	0.011
Serum alanine aminotransferase	(IU/L)	28 (6–996)	28±41	237±46	<0.001
Serum total bilirubin	(mg/dL)	0.8 (0.2–6.8)	1.5±0.5	1.2±0.5	0.300
Serum creatine phosphokinase	(IU/L)	105 (3.4–84106)	970±2,900	8,700±3,300	0.083
Serum blood urea nitrogen	(mg/dL)	32 (12–94)	35.6±3.7	39.7±4.2	0.235
Serum creatinine	(mg/dL)	1.4 (0.4–11.7)	1.7±0.4	2.9±0.5	0.071
Serum albumin	(g/dL)	2.9 (1.3–4.5)	3.0±0.2	2.7±0.2	0.081
Serum sodium	(Eq/dL)	137 (120–155)	137±1.1	138±1.3	0.364
Serum potassium	(Eq/dL)	4.0 (2.2–7.6)	3.9±0.2	4.6±0.2	0.014
White cell count	(/μL)	9,500 (1,490–52,000)	9,700±1,800	16,100±2,000	0.011
Hemoglobin	(mg/dL)	11.8 (7.2–18.1)	11.5±0.4	12.2±0.5	0.152
Hematocrit	(%)	35.4 (21–58)	33.0±1.4	36.1±1.6	0.237
Platelets	($\times 10^4$/μL)	16.6 (1.2–36.7)	19.5±1.7	15.5±2.0	0.074

Data listed for all patients are mean values (range). Data listed for group I and group II are mean values±standard deviation

Group I patients who survived to discharge, *group II* patients who did not survive to discharge

Table 3 Comparison of clinical findings between group I and group II

Factor	All	Group I	Group II	*p* value
Metabolic acidosis	17/32 (53)	4/14 (29)	13/18 (72)	0.017
Postoperative hypotension	20/50 (40)	8/27 (30)	12/23 (52)	0.179
Portal venous gas	22/46 (48)	16/26 (62)	6/20 (30)	0.033
Colon ischemia	21/51 (41)	7/28 (25)	14/23 (61)	0.010
Bowel resection >1 m	35/51 (69)	17/28 (61)	18/23 (78)	0.149
POSSUM score (n=47), mean±SD	68.3±3.5	54.5±3.6	85.2±4.1	<0.001
Duration of operation (min; n=51), mean±SD	153±13	149±17	158±19	0.365
Blood count (mL; n=51), mean±SD	838±398	320±530	1,470±590	0.070

Data listed are total number (%)

SD standard deviation, *group I* patients who survived to discharge, *group II* patients who did not survive to discharge

and one was treated with prostaglandin E1 and continuous hemodiafiltration.

Discussion

Acute mesenteric ischemia, including mesenteric arterial embolism, mesenteric arterial thrombosis, NOMI, and mesenteric venous thrombosis, has a poor prognosis with a high in-hospital mortality rate (59–93 %).[2] NOMI is a particularly poorly understood condition marked by progressive intestinal ischemia leading to infarction, sepsis, and death in a high proportion of patients.

NOMI appears to occur secondary to cardiac disease, diabetes mellitus, and chronic dialysis-dependent renal failure.[2,4,14] In this study, 49 % of NOMI patients suffered from hypertension, 47 % from cardiovascular disease, 20 % from diabetes mellitus, and 14 % from dialysis-dependent renal disease. According to previous reports, digitalis is an additional risk factor for NOMI,[3,15,16] perhaps because it induces vasoconstriction and thus increases resistance in peripheral splanchnic vessels. In this study, 8 % of NOMI patients were on digitalis therapy.

Conventional angiography is regarded as the gold standard imaging method in patients with acute mesenteric ischemia.[3,5,6] However, NOMI often occurs in patients with poor or unstable systemic conditions, and angiography may not be possible in many of these patients due to its complexity and invasiveness.[2,17] Catheter angiography is invasive and difficult to perform, so its use is limited to select centers.[2,3,17] Indeed, Bender et al.[18] reported that none of their sample population received angiograms. In this study, only one patient underwent angiography. Hence, angiography is not the primary method for NOMI diagnosis in clinical practice. Mitsuyoshi et al.[3] reported the usefulness of multidetector-row computed tomography for the diagnoses of NOMI. In this study, computed tomography was the primary imaging modality used for NOMI diagnosis.

Histopathologic detection of hemorrhagic and necrotic changes is required for definite diagnosis of NOMI.[12] Unfortunately, pathological examination was not available for 11 study patients who received exploratory laparotomy. In these cases, we used macroscopic findings from the laparotomy to definitively diagnose NOMI. Furthermore, although 40 patients underwent bowel resection, a pathological evaluation of the resected specimen was available for only 25 patients. The findings from all 25 patients met the pathological criteria of a NOMI diagnosis.

Ischemic colitis represents the most common form of gastrointestinal ischemia. Many previous reports have not distinguished NOMI from ischemic colitis. Witternberg et al.[19]

Table 4 Comparison of operative procedures between group I and group II

Operative procedure	All	Group I (n=28)	Group II (n=23)
Intestinal resection with enterostomy	30 (59)	18	12
Intestinal resection with reconstruction (with diverting enterostomy)	2 (4)	1	1
Intestinal resection with reconstruction (without diverting enterostomy)	8 (16)	7	1
Exploratory laparotomy (no findings of intestinal necrosis)	4 (8)	2	2
Exploratory laparotomy (findings of intestinal necrosis)	7 (14)	0	7

Data listed are total number (%)

Group I patients who survived to discharge, *group II* patients who did not survive to discharge

Table 5 Comparison of additional postoperative treatment between group I and group II

Additional postoperative treatment	All (n=51)	Group I (n=28)	Group II (n=23)
Patients who received treatment	23	17 (74)	6 (26)*
Prostaglandin E1	10	7	3
CHDF	9	5	4
PMX	3	2	1
Anticoagulant therapy	3	3	0
Nitrovasodilator	1	1	0
Octreotide	1	1	0
Patients who did not receive treatment	28	11 (39)	17 (61)*

Data listed are total number (%)

CHDF continuous hemodiafiltration, *PMX* polymyxin B-immobilized column direct hemoperfusion, *group I* patients who survived to discharge, *group II* patients who did not survive to discharge

*There was significant difference in postoperative mortality between patients who did or did not receive additional postoperative treatment (p=0.01)

reported differences in the incidence of the underlying vascular etiologies of the two major categories of primary ischemic disease of the bowel. NOMI is a disease primarily of the superior mesenteric artery distribution, whereas ischemic colitis is a disease primarily of the inferior mesenteric artery distribution. We thus excluded cases of bowel ischemia isolated to areas of the colon supplied by the inferior mesenteric artery.

Hemodialysis is a known risk factor for NOMI, because patients with end-stage renal disease have many risk factors for mesenteric ischemia.[7] However, the prognostic impact of hemodialysis on patients who undergo surgery for NOMI has not been evaluated. Our results demonstrate that hemodialysis is a negative prognostic factor for NOMI patients who receive surgery.

Among the 46 patients who underwent computed tomography, portal venous gas was detected in 22 (48 %). Portal venous gas may be found in a variety of conditions.[20] Portal venous gas resulting from bowel ischemia has been shown to be a poor prognostic factor, with an associated mortality rate of 75–90 %.[21] Surprisingly, in our study, patients with portal venous gas had a significantly better prognosis. This finding may be explained by the fact that patients with portal venous gas were diagnosed with severe intestinal necrosis and underwent immediate surgery.

Table 6 Comparison of survival rates between patients who did or did not receive additional postoperative treatment

POSSUM score	With postoperative treatment (n=22)	Without postoperative treatment (n=25)	p value
90≦ (n=11)	0/4 (0)	0/7 (0)	–
76.1≦ and <90 (n=11)	3/3 (100)	1/8 (13)	0.024
<76.1 (n=25)	13/15 (87)	9/10 (90)	0.802
Average POSSUM score	66.9±4.9	69.5±5.2	0.360

Percentages are in parentheses

The area affected by ischemic bowel can range from a few decimeters up to the entire small intestine and colon. Sotriadis J et al.[22] reported that patients with isolated right colon ischemia had a worse outcome than patients with ischemia involving other colon regions. We found that patients with extensive bowel involvement (extending from the small intestine to the colon) had a poorer prognosis. Aliosmanoglu et al.[23] reported that acute mesenteric ischemia involving both the colon and the small intestine resulted in a higher mortality. The high mortality rate in these patients may be due to vasoconstriction of the inferior and superior mesenteric artery territories.

In some cases of NOMI, ischemia progresses after surgery, requiring a second-look surgery to be performed.[8] Ward et al.[8] reported that aggressive re-exploration and delayed intestinal anastomosis improved survival of NOMI patients. In this study, 31 patients (59 %) underwent bowel resection with enterostomy (without anastomosis), 10 patients (20 %) with anastomosis (2 patients with diverting enterostomy), and 3 patients underwent second-look surgery. It can be difficult for surgeons to determine whether to create an anastomosis or an enterostomy during NOMI operations. We found no complications associated with anastomosis formation, suggesting that surgeons appropriately judged the most suitable operative procedure for each patient.

POSSUM scores and the Acute Physiology and Chronic Health Evaluation (APACHE) II are used to evaluate the risk of surgery.[24–26] The POSSUM score is easier to use than APACHE II and has been reported to be superior to APACHE II in predicting mortality in patients admitted to a high-dependency unit after general surgery.[27] To the best of our knowledge, this is the first study to report the prognostic role of the POSSUM score in a series of NOMI patients treated with surgery. We found that a POSSUM score of 76.1 or higher [as determined by the ROC curve (AUC=0.905)] was a predictor of in-hospital mortality.

To date, no published study has focused on additional postoperative treatment of NOMI patients. In this study, patients who received additional postoperative treatments had a better clinical course. However, we found no significant difference in the POSSUM scores of patients who underwent additional postoperative treatment and those who did not (Table 6). Hence, additional postoperative treatment of NOMI patients has the potential to improve prognosis, especially among patients with POSSUM scores between 76.1 and 90.

Patients with NOMI commonly receive intra-arterial infusions of papaverine after surgery. However, angiography is difficult to perform, and access is limited to select centers; hence, intra-arterial infusions of vasodilators can be difficult.

Mituyoshi et al. reported that continuous intravenous PGE1 administration resolved the spasm and narrowing of the superior mesenteric artery in NOMI patients. In our study, ten patients received continuous intravenous PGE1 administration. A larger study is required to understand the efficacy of continuous intravenous PGE1 administration for additional postoperative treatment of NOMI.

Our study is the largest study to date to evaluate the clinical features and prognostic factors of NOMI. Based on our findings, we conclude that the POSSUM score can be used to predict the outcome of NOMI patients who undergo surgery. Surgery followed by additional postoperative treatment may improve the prognosis of NOMI. It is difficult and impractical to use randomized controlled trials to determine the usefulness of surgery for NOMI, and retrospective studies from multiple institutions may be required to provide sufficient data. Our findings offer useful information for determining the treatment strategy for NOMI.

Acknowledgments We wish to thank the following institutions for participating in the investigation: Iizuka Hospital, Imari Arita Kyoritsu Hospital, Eikou Hospital, Oita Medical Center, Oita Prefecture Hospital, Oita Red Cross Hospital, Onga Hospital, Kitakyushu Municipal Medical Center, Kakizoe Hospital, National Kyushu Cancer Center, Saiseikai Karatsu Hospital, Saiseikai Fukuoka General Hospital, Saiseikai Yahata General Hospital, Shinnakama Hospital, Nakatsu Municipal Hospital, Hita Central Hospital, Hiroshima Red Cross Hospital & Atomic Bomb Survivors Hospital, Fukuoka City Hospital, National Fukuoka-Higashi Medical Center, Steel Memorial Yawata Hospital, Beppu Medical Center, Matsuyama Red Cross Hospital, and Munakata Medical Association Hospital.

References

1. Ende N. Infarction of the bowel in cardiac failure. N Engl J Med. 1958;258:879–881.
2. John AS, Tuerff SD, Kerstein MD. Nonocclusive mesenteric infarction in hemodialysis patients. J Am Coll Surg 2000; 190:84–88.
3. Mitsuyoshi A, Obama K, Shinkura N, Ito T, Zaima M. Survival in nonocclusive mesenteric ischemia: Early diagnosis by multidetector row computed tomography and early treatment with continuous intravenous high-dose prostaglandin E1. Ann Surg 2007;246:229–235
4. Quiroga B, Verde E, Abad S, Vega A, Goicoechea M, Reque J, López-Gómez JM, Luño J. Detection of patients at high risk for non-occlusive mesenteric ischemia in hemodialysis. J Surg Res. 2013;180:51–55.
5. AGA technical review on intestinal ischemia. Gastroenterology 2000;118:954–968.
6. American Gastroenterological Association medical position statement: guidelines on intestinal ischemia. Gastroenterology 2000;118:951–953.
7. Brandt LJ, Boley SJ. Nonocclusive Mesenteric Ischemia. Annu Rev Med 1991;42:107–117
8. Boley SJ, Sprayrgen S, Sieglman SS, Vieth FJ. Initial results from an aggressive roentgenological approach to acute mesenteric ischemia. Surgery 1977;82:848–855
9. Ward D, Vernava AM, Kaminski DL, Ure T, Peterson G, Garvin P, Arends TW, Longo WE. Improved Outcome by Identification of High-Risk Nonocclusive Mesenteric Ischemia, Aggressive Reexploration, and Delayed Anastomosis. Am J Surg 1995;170: 577–581.
10. Heer FW, Silen W, French SW. Intestinal gangrene without apparent vascular occlusion. Am J Surg. 1965;110:231–238.
11. Fogarty JT, Fletcher SW. Genesis to nonocclusive mesenteric ischemia. Am J Surg. 1966;111:130–137.
12. Copeland GP, Jones D, Walters M. POSSUM: a scoring system for surgical audit. British Journal of Surgery 1991;78(3):355–60.
13. Hirasawa H, Oda S, Nakamura M, Watanabe E, Shiga H, Matsuda K. Continuous hemodiafiltration with a cytokine-adsorbing hemofilter for sepsis. Blood Purif. 2012;34(2):164–70.
14. Sato H, Oshima K, Arakawa K, Kobayashi K, Yamazaki H, Suto Y, Takeyoshi I. Direct hemoperfusion with a polymyxin B-immobilized cartridge in intestinal warm ischemia reperfusion. World J Gastroenterol. 2008 ;14(35):5436–41.
15. Howard TJ, Plaskon LA, Wiebke EA, Wilcox MG, Madura JA. Nonocclusive mesenteric ischemia remains a diagnostic dilemma. Am J Surg. 1996;171:405–408.
16. Trompeter M, Brazda T, Remy CT, Vestring T, Reimer P. Non-occlusive mesenteric ischemia: etiology, diagnosis, and interventional therapy. Eur Radiol. 2002;12(5):1179–1187.
17. Lock G, Scholmerich J. Nonocclusive mesenteric ischemia. Hepatogastroenterology. 1995;42:234–239.
18. Bender JS, Ranter LE, Magnuson TH, Zenilman ME. Acute abdomen in the hemodialysis patient population. Surgery 1995;117:494–497.
19. Wittenberg J, Athanasoulis CA, Williams LF, Paredes S, O'sullvian P, Brown B. Ischemic colitis. Radiology and pathophysiology. Am J Roentgenol Radium Ther Nucl Med. 1975;123(2):287–300.
20. Clinical features and management of hepatic portal venous gas: four case reports and cumulative review of the literature. Arch Surg 2001;136 (12) 1410–1414.
21. Liebman PR, Patten MT, Manny J, Benfield JR, Hechtman HB. Hepatic-portal venous gas in adults: Etiology, pathophysiology and clinical significance. Ann Surg 1978;187: 281–287.
22. Sotiriadis J, Brandt LJ, Behin DS, Southern WN. Ischemic Colitis Has a Worse Prognosis When Isolated to the Right Side of the Colon. Am J Gastroenterol 2007;102(10):2247–2252.
23. Aliosmanoglu I, Gil M, Kapan M, Arikanoglu Z, Taskesen F, Basol O, Aldemir M. Risk factors effecting mortality in acute mesenteric ischemia and mortality rates: a single center experience. Int Surg 2013;98:76–81.
24. Knaus WA, Draper EA, Wagner DP, Zimmerman JE. APACHE II: A severity of disease classification system. Critical Care Medicine 1985;13:818–829.
25. Hsu HP, Shan YS, Hsieh YH, Sy ED, Lin PW. Impact of Etiologic Factors and APACHE II and POSSUM Scores in Management and Clinical Outcome of Acute Intestinal Ischemic Disorders after Surgical Treatment. World J Surg 2006;30: 2152–2162
26. Mohil RS, Bhatnagar D, Bahadur L Rajneesh, Dev DK, Magan M. POSSUM and P-POSSUM for risk-adjusted audit of patients undergoing emergency laparotomy. Br J Surg 2004;91 500–503.
27. Jones DR, Copelans GP, de Cossart L. Comparison of POSSUM with APACHE II for prediction of outcome from a surgical high dependency unit. Br J Surg 1992;79:1293–1296.