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Original research

A study on 107 patients with acute mesenteric ischemia over 30 years

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ABSTRACT

Acute mesenteric ischemia (AMI) is a life threatening cause of acute abdomen. The purpose of this study is to define risk factors that predict the adverse outcome of AMI and to present our experience in the last 30 years. Hospital records and clinical data of 107 patients undergoing surgical intervention for AMI during the last 30 year period were reviewed and clinical outcomes as well as factors influencing mortality were analyzed. Mesenteric arterial thrombosis, arterial embolism and nonocclusive mesenteric ischemia (NOMI) were the cause of AMI in 68 (63.6%), 28 (26%), and 11 patients (10.2%), respectively. Abdominal pain was the most common presenting symptom (90.6%). Peritonitis was observed in 96 patients (89.7%) and 24 patients (22.4%) were in shock. Abdominal ultrasonography was performed in 46 patients (42%), abdominal CT angiography in 36 patients (33%) and mesenteric angiography in 12 patients (10.5%). All patients were operated and 11 (10%) patients underwent a second-look operation. Bowel resection was necessary in 101 patients (93.4%) during the initial operation and in seven patients (6.5%) during the second-look operation. The hospital mortality was 55.1%. Mortality was mainly due to multiorgan failure (43%). Diabetes mellitus, use of digoxine and antiplatelet drugs, duration of the symptoms until before surgery, existence of shock, low levels of the pH and bicarbonate and relaparotomy were found to be negative predictors of the perioperative mortality. The use of total parenteral nutrition and CT angiography was found to be a protective factor against mortality. A high index of suspicion with prompt diagnostic evaluation with CT angiography may reduce time prior to surgical intervention which may lead to improved patient survival.

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1. Introduction

AMI is a life threatening cause of acute abdomen requiring early surgical intervention. In recent years, the mortality of AMI has been reported to vary from 60% to 90% in spite of the improved fluid management, respiratory care and nutritional support.^{1–6}

AMI occurs when mesenteric vessels occlude due to mesenteric arterial, venous thrombosis, arterial embolism and nonocclusive mesenteric ischemia (NOMI).^{6–8} The occlusion of the intestinal vessels causes the impairment of microcirculation of the intestine with the activation of the endothelium, monocytes, leukocytes and platelets.^{2,3,9,10} Activated inflammatory cells produce many mediators such as cytokines, platelet –activating factor, nitric oxide, leukotriene and toxic free oxygen radicals. Leukocyte adhesion, platelet aggregation and nitric oxide increase the damage of the intestinal microcirculation.^{2,3,9} Toxic free oxygen radicals which damage the cell membrane through lipid peroxidation cause an

increase in capillary permeability.¹⁰ The damaged intestinal microcirculation with increased capillary permeability leads to the bacterial translocation and this translocation may play an important role in the development of sepsis resulting in multiple organ failures and death.^{3,9,11}

NOMI is defined as a low cardiac output state associated with diffuse mesenteric vasoconstriction. Mesenteric vasoconstriction occurs in response to hypovolemia, myocardial infarction, congestive heart failure, aortic insufficiency, renal or hepatic disease, abdominal or cardiac surgery and vasopressor treatment.^{1–5} Vasoactice drugs such as digoxine, enteral nutrition in intensive care units, multiple trauma are the other rare causes of NOMI.^{2,3,6}

The key points for recovery from this condition are early diagnosis, resection of the infarcted bowel, restoration of the blood flow, second-look laparotomy if necessary and supportive intensive care.^{2–4,12,13} Most patients with AMI are admitted to the emergency department with severe abdominal pain and signs of peritonitis.^{3,4} These patients with AMI are usually elderly and they usually have a complex medical history. AMI has no specific symptoms or pathognomonic clinical findings.¹² Physical examination cannot differentiate bowel ischemia from necrosis.^{2,12} Therefore the

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diagnosis of AMI with mesenteric infarction may be delayed resulting in high mortality. That may be the reason why in the last years the mortality rate has not changed much except in a few limited series.^{1–6,8,14}

The aim of this study was to present our thirty year experience about the preoperative and postoperative prognostic factors of early mortality in AMI and thus help the clinician in his/her therapeutic decision making.

2. Patients and methods

The clinical records of all the patients who underwent surgical treatment for AMI due to arterial embolism, thrombosis nonocclusive mesenteric ischemia (NOMI) between April 30, 1980 and April 30, 2010, were examined. Patient's demographics, comorbidity, physical findings, laboratory test results, and diagnostic imaging reports were reviewed. Initial admission laboratory test results were taken into account. The data from operative records postoperative complications, second-look laparotomies, mortality (in-hospital mortality after surgical treatment) and hospital stay were recorded. Based on clinical, radiological, surgical and histological findings patients were classified as having mesenteric emboli, arterial or venous thrombosis or a NOMI.^{8,15}

3. Statistical analysis

Data were analyzed using statistical package SPSS 13.01 for windows (SPSS, Chicago, IL, USA, serial number 9069728). The chisquared test or Fisher's test was used to compare categorical data. For the parametric distribution of values, Student *t*-test or analysis of variance (ANOVA) was used to compare the mean values of two or more groups. For the nonparametric variables, the Kruskal–Wallis test or Mann–Whitney *U* test was used to compare the median values of the response variable. A stepwise logistic regression analysis was performed. The test results were considered significant at a *P* value of less than 0.05.

4. Results

Sixty eight patients were men (63.6%) and 39 were women (36.4). The mean \pm SD age was 69 \pm 8 with the range of 39–92 years. Superior mesenteric artery (SMA) thrombosis was the most common form of AMI and was seen in 68 patients (68.6). 28 patients (%26.2) had SMA embolism, and 11 patients (10.2%) had NOM I (Table 1).

Demographic data and concomitant illness related mortality are given in Table 1. Diabetes mellitus, the use of antiplatelet drugs such as aspirin, dipyridamole, clopidogrel in our country and digoxin were related with perioperative mortality. Ninety seven patients (90.6%) were admitted to the emergency department with acute abdominal pain. Presence of shock and interval between the beginning of symptoms and the onset of the operation were statistically associated with mortality (Table 2). 96 patients (89.7%) had signs of peritonitis during the initial abdominal examination. Other symptoms and physical findings are summarized in Table 2. Preoperative laboratory findings related to mortality rate are shown in Table 2. pH and bicarbonate levels were significantly associated with mortality.

All our patients with NOMI were followed in intensive care units by departments of cardiology and anaesthesiology. These patients underwent laparotomy since acute abdomen could not be excluded.

We performed plain abdominal radiography on 90 patients (84.3%) and abdominal ultrasonography on 46 patients (42%). Since 1999 abdominal CT angiography was done in 36 patients (33%) and mesenteric angiography was performed on 12 patients (10.5%).

Laparotomy was performed on all patients, of which 11 were taken a second-look and underwent resection. Surgical procedures are shown in Table 3. Bowel resection was performed in 101

Table 1

Demographics, and comorbidity, of our AMI patients and the causes of AMI. AMI; acute mesenteric ischemia, SMA; superior mesenteric artery.

	Total no (%)	Alive (%)	Dead (%)	Р
Number of patients	107 (100%)	48 (44.9)	59 (55.1)	0.03
Sex				
Men	68 (63.6)	30 (44.1)	38 (55.9)	NS
Women	39 (36.4)	17 (45)	22 (55)	NS
Mean age ^a	69 ± 8	68 ± 8	70 ± 9	NS
Hypertension	82 (76.6)	36 (43.9)	46 (56)	NS
Chronic lung disease	31 (28.1)	15 (48.3)	16 (51.7)	NS
Peripheric vascular disease	39 (36.4)	15 (38.5)	24 (61.5)	NS
Diabetes mellitus	12 (11.4)	3 (25)	9 (75)	0.04
Atrial fibrillation	84 (78.5)	27 (53.6)	57 (46.4)	NS
Congestive heart failure	75 (70)	33 (44)	42 (56)	NS
Coronary arterial diseases	46 (40.2)	21 (45.6)	25 (54.4)	NS
Renal failure	6 (5)	3 (50)	3 (50)	NS
Antiplatelet drugs ^b	74 (69.1)	20 (27)	54 (73)	0.04
Digoxin	56 (52.3)	16 (28.5)	40 (71.5)	0.03
Causes of AMI				
SMA thrombosis	68 (63.6)	32 (47)	36 (53)	NS
SMA embolus	28 (26.2)	15 (53.6)	13 (44.4)	NS
Non-occlusive mesenteric	11 (10.2)	1 (9)	10 (91)	0.45
Ischemia	. ,	. ,	. ,	

NS = no significant.

^a Mean \pm standard deviation.

^b Aspirin, dipyridamole, clopidogrel.

patients (93.4%). Laparotomy was performed on the rest of the patients (six patients) but bowel resection was not done due to the massive gastrointestinal necrosis. End colostomy was performed in one patient. The type of anastomosis, whether stapled or handmade had no effect on mortality.

59 patients died in the postoperative period due to the AMI. Therefore the operative mortality was 55.1%. The mortality rate was 53% in patients with thrombosis, 44.4% in patients with arterial embolism and 91% in patients with NOMI. The type of ischemia was

Table 2

Presenting symptoms, physical findings, laboratory results and between survivors and non-survivors.

	Total no (%)	Alive (%)	Dead (%)	Р
Duration of symptoms	21 ± 5	14 ± 6	36 ± 7	0.05
prior to surgery (h ^a)				
Abdominal pain	97(90.6)	40(84.2)	57(96.5)	NS
Nausea and vomiting	52(48.5)	26(57.1)	26(44.4)	NS
Abdominal distention	28(26.1)	12(25.7)	16(27.5)	NS
Hematemesis	11(10.2)	5(11.4)	6(8.6)	NS
Rectal bleeding	13(12.1)	7(14.2)	6(8.6)	NS
Diarrhea	14(13)	7(14.2)	7(10.3)	NS
Fever	16(14.9)	8(17.1)	8(13.7)	NS
Shock	24(22.4)	4(8.5)	20(34.4)	0.04
Peritonitis	96(89.7)	48(100)	48(79.3)	NS
CT angiography	36(33.6)	12(33.3)	24(66.6)	0.04
Body temperature, °C ^a		36.8 ± 0.8	36.9 ± 1.2	NS
Pulse rate, per min ^a		95 ± 21	108 ± 24	NS
Systolic blood pressure,		129 ± 13	107 ± 14	0.03
mmHg ^a				
Laboratory results ^a				
Hemoglobin, g/dl		12.4 ± 2.4	12.7 ± 3	NS
WBC, mm ³		$\textbf{17,800} \pm \textbf{4400}$	$19{,}549\pm6400$	NS
BUN, mg/dl		32 ± 6	41 ± 7	NS
Creatinine, mg/dl		1.7 ± 0.3	2 ± 0.3	NS
ALT, U/L		46 ± 34	112 ± 52	NS
Amylase, U/L		118 ± 30	280 ± 68	NS
CPK, U/L		425 ± 478	1200 ± 680	NS
Albumin, g/dl		$\textbf{3.2}\pm\textbf{0.3}$	3.1 ± 0.4	NS
PH		$\textbf{7.42} \pm \textbf{0.14}$	$\textbf{7.18} \pm \textbf{0.13}$	0.03
Bicarbonate, mmol/L		$\textbf{20.9} \pm \textbf{2.1}$	10 ± 3	0.04

^a Mean \pm standard deviation.

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