## ORIGINAL ARTICLE

# **Clinical Factors and Outcomes in Patients with Acute Mesenteric Ischemia in the Emergency Department**

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**Background:** The purpose of this study was to determine the initial clinical characteristics of acute mesenteric ischemia and identify variables associated with adverse outcomes in the emergency department (ED).

**Methods:** The charts of 124 consecutive patients with surgically and pathologically identified acute mesenteric ischemia from September 1990 to September 2000 were reviewed retrospectively to obtain data about demographics, initial clinical presentations, predisposing diseases, previous medications, laboratory tests, and common findings on computed tomography scans with contrast. Only patients admitted through the ED and treated on medical or surgical wards were enrolled.

**Results:** Mean patient age was 71.1 years (range, 25–100 years). The overall mortality rate was 50%. There were no significant differences in gender, underlying disease, previous medication, initial signs and symptoms, and causes of mesenteric infarction, between survivors and non-survivors. Univariate analysis demonstrated that older age, bandemia, hepatic and renal impairment, hyperamylasemia, metabolic acidosis, hypoxia, intramural pneumatosis, and septic syndrome, were more frequent in patients who died than in those who survived (p < 0.05). Logistic regression identified the following variables as independent predictors of death: old age (odds ratio, OR, 1.077; 95% confidence interval, Cl, 1.013, 1.146; p = 0.02); bandemia (OR, 3.894; 95% Cl, 1.160, 13.074; p = 0.03); elevated serum aspartate aminotransferase (AST; OR, 4.532; 95% Cl, 1.274, 16.122; p = 0.02); increased blood urea nitrogen (BUN; OR, 7.219; 95% Cl, 1.166, 44.696; p = 0.03); and metabolic acidosis (OR, 6.604; 95% Cl, 1.804, 24.171; p < 0.01).

**Conclusion:** A high index of suspicion and aggressive diagnostic imaging can facilitate early diagnosis and improve outcomes for patients with acute mesenteric ischemia. Risk stratification showed that elderly patients with metabolic acidosis, bandemia, or elevated AST and BUN had a poor prognosis. Greater therapeutic intervention is advocated to reduce mortality in high-risk patients with acute mesenteric ischemia. [*J Chin Med Assoc* 2005;68(7):299–306]

Key Words: emergency department, mesenteric ischemia, outcomes

## Introduction

Acute mesenteric ischemia is an uncommon but devastating emergency, occurring mostly in elderly patients and with an overall mortality rate ranging from 60% to 90%.<sup>1-3</sup> Factors associated with this high mortality include atypical clinical presentations (subacute nature of symptom progression),<sup>4</sup> lack of

predisposing disease, and diagnostic difficulties leading to delayed surgical intervention.<sup>5,6</sup> A high index of suspicion for the disease and an aggressive diagnostic approach are essential for early identification and surgical intervention to increase survival<sup>7,8</sup> and reduce the risk of malpractice claims.<sup>9</sup> The primary role of emergency physicians in evaluating acute abdominal pain is to distinguish benign abdominal disorders from

\*Correspondence to: Dr. David Hung-Tsang Yen, Department of Emergency Medicine, Taipei Veterans General Hospital, 201, Section 2, Shih-Pai Road, Taipei 112, Taiwan, R.O.C. E-mail: hjyen@vghtpe.gov.tw • Received: December 22, 2004 • Accepted: April 4, 2005 ischemia-related syndromes.<sup>10</sup> Further, the clinical presentations associated with a high risk of mortality in patients with acute mesenteric ischemia should be identified to provide appropriate risk stratification in the emergency department (ED). The purpose of this study was to demonstrate such initial clinical presentations and investigate prognostic risk factors in ED patients with acute mesenteric ischemia.

#### Methods

#### Study population

Cases of acute mesenteric ischemia were first identified from a computer search for hospital discharge codes (International Classification of Diseases) of 557.0 and 557.9. The diagnosis of mesenteric infarction was made by surgical intervention and later confirmed by histologic results. Only patients admitted through the ED and treated on medical or surgical wards were enrolled. Eight patients with mesenteric ischemia secondary to mechanical obstruction, adhesions, or volvulus were excluded, as were patients less than 14 years of age. Data about demographics, initial clinical presentations, predisposing diseases, previous medications, laboratory tests, and the common findings on computed tomography (CT) scans<sup>11</sup> with contrast were evaluated in 124 patients classified into 2 groups according to outcome: survival or death.

#### Statistical analysis

Categoric variables were compared between groups using the Chi-squared or Fisher's exact test for qualitative data. The Mann-Whitney non-parametric U-test or t test was used to analyze quantitative data. Multiple logistic regression analyses were applied to determine the independent variables predictive of patient outcomes. The Hosmer-Lemeshow goodness-of-fit test and the likelihood ratio test for model selection were performed. When applicable, tests were 2-tailed. A p value of less than 0.05 was considered statistically significant.

#### Results

From September 1990 to September 2000, 124 patients (104 males and 20 females) with acute mesenteric ischemia, identified by surgical intervention and pathologic confirmation, were enrolled. The initial ED and admission medical records of all patients were reviewed.

Mean patient age was 71.1 years (range, 25–100 years) (Figure 1), with 99 patients (79.8%) older than



**Figure 1.** Age distribution in 124 patients with acute mesenteric infarction (mean age,  $71.1 \pm 12.0$  years).

65 years. Table 1 lists demographic data, major underlying disorders, medications, and causes for all patients with acute mesenteric ischemia. Patients who died were significantly older than those who survived (74.3 vs 67.9 years; p < 0.01). Hypertension (29.8% of patients), arrhythmias (27.4%), and cerebral vascular diseases (19.4%) were the most common underlying conditions (Table 1). No statistically significant differences were evident between survivors and non-survivors regarding major predisposing disorders, medications used, and causes of acute mesenteric ischemia.

The mean duration of signs and symptoms before ED visit was shorter (but not significantly) in survivors than non-survivors  $(54.0 \pm 102.2 \text{ hr vs } 62.6 \pm 82.4 \text{ m})$ hr); and corresponding mean durations of operation after ED visit were also not significantly different  $(36.0 \pm 98.0 \text{ hr vs } 53.8 \pm 100.9 \text{ hr})$  (Table 2). Significantly more survivors than non-survivors had a history of abdominal pain at clinical presentation (91.9% vs 74.2%; p = 0.02), and systolic blood pressure was significantly greater in survivors than non-survivors (129.5 vs 114.8 mmHg; *p* < 0.01). In laboratory tests, leukocytosis (white blood cell count >  $10,800/\text{mm}^3$ ) was noted in 80 patients (64.5%), and band-form shifting in 64 (51.6%). Septic syndrome and metabolic acidosis were noted significantly less frequently, and significantly lower values were documented for bandemia, blood urea nitrogen (BUN), creatinine, aspartate aminotransferase (AST), alanine aminotransferase, and amylase, in survivors than nonsurvivors ( $p \le 0.03$ ) (Table 2).

In radiologic studies, plain abdominal films revealed abnormal findings in 16 of 74 patients (21.6%). These abnormal findings included bowel-wall thickening (n = 12), intestinal loop or ileus (7), and pneumatosis intestinalis (2). Fifty-six patients (45.2%) underwent

Table 1. Comparison of demographic characteristics,	major underlying disorders,	and causes of acute	mesenteric ischemia
between groups			

	Number (%) of patients		
	Survivors $(n = 62)$	Non-survivors ( $n = 62$ )	Ρ
Age, yr*	67.9 ± 13.7	74.3 ± 9.1	< 0.01
Male/female	54/8	50/12	NS
Underlying disease			
Hypertension	20 (32.3)	17 (27.4)	NS
Arrhythmias	16 (25.8)	18 (29.0)	NS
Cerebral vascular diseases	11 (17.7)	13 (21.0)	NS
Diabetes mellitus	9 (14.5)	10 (16.1)	NS
Coronary artery disease	12 (19.4)	4 (6.5)	0.06
Renal disease <sup>†</sup>	4 (6.5)	12 (19.4)	0.06
Congestive heart failure	5 (8.1)	4 (6.5)	NS
Previous myocardial infarction	4 (6.5)	4 (6.5)	NS
Malignancy	4 (6.5)	3 (4.8)	NS
Hematologic disease	2 (3.2)	1 (1.6)	NS
Valvular heart disease	5 (8.1)	1 (1.6)	NS
Systemic lupus erythematosus	1 (1.6)	0 (0)	NS
Medications			
Antiplatelet	10 (16.1)	4 (6.5)	NS
Anticoagulant	5 (8.1)	0 (0)	0.06
Anti-angina	11 (17.7)	6 (9.7)	NS
Causes of acute mesenteric ischemia			
Superior mesenteric artery embolus	23 (37.1)	26 (41.9)	NS
Non-occlusive mesenteric infarction	17 (27.4)	15 (24.2)	NS
Superior mesenteric artery thrombosis	8 (12.9)	8 (12.9)	NS
Mesenteric venous thrombosis	6 (9.7)	6 (9.7)	NS
Unknown	8 (12.9)	7 (11.3)	NS

\*Mean  $\pm$  standard deviation; <sup>†</sup>chronic renal insufficiency or failure. NS = not significant.

#### Table 2. Comparison of initial clinical presentations, laboratory results, and hospital stays between groups

	Number (%) of patients		n
	Survivors ( $n = 62$ )	Non-survivors ( $n = 62$ )	٣
Duration of signs and symptoms before ED visit, hr	* 54.0 ± 102.2	62.6 ± 82.4	NS
Duration of operation after ED visit, hr*	36.0 ± 98.0	53.8 ± 100.9	NS
Signs and symptoms			
Abdominal pain	57 (91.9)	46 (74.2)	0.02
Vomiting	32 (51.6)	22 (35.5)	NS
Abdominal distension	9 (14.5)	15 (24.2)	NS
Hypotension	4 (6.5)	11 (17.7)	NS
Diarrhea	15 (24.2)	9 (14.5)	NS
Bloody stools	8 (12.9)	5 (8.1)	NS
Tarry stools	2 (3.2)	4 (6.5)	NS
Coffee-ground vomitus	3 (4.8)	4 (6.5)	NS
Hematemesis	0 (0)	1 (1.6)	NS
Chest pain	0 (0)	1 (1.6)	NS

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able 2. Comparison of initial clinical presentations, laboratory results, and hospital stays between groups			
	Number (%	Number (%) of patients	
	Survivors $(n = 62)$	Non-survivors ( $n = 62$ )	Ρ
Physical findings			
Body temperature, °C*	$36.7 \pm 1.0$	$36.6 \pm 1.4$	NS
Pulse rate, per min*	94.7 ± 24.4	$102.3 \pm 27.1$	NS
Systolic blood pressure, mmHg*	129.5 ± 35.0	114.8 ± 37.2	< 0.01
Abdominal tenderness	59 (95.2)	53 (85.5)	NS
Decreased or absent bowel sounds	37 (59.7)	45 (72.6)	NS
Rebounding pain	39 (62.9)	34 (54.8)	NS
Muscle guarding	20 (32.3)	24 (38.7)	NS
Laboratory results*			
WBC, $mm^3$ ( <i>n</i> = 124)	12,725.7 ± 4,727.4	15,205.3 ± 8,908.0	NS
Band, % $(n = 124)$	$3.1 \pm 9.5$	$14.9 \pm 18.4$	< 0.01
Segment, % ( $n = 124$ )	78.3 ± 15.7	66.7 ± 21.5	< 0.01
Lymphocyte, % ( $n = 124$ )	$13.5 \pm 10.8$	$10.3 \pm 7.8$	NS
Platelet, $\times 10^{3}$ /mm <sup>3</sup> ( <i>n</i> = 124)	226.2 ± 110.6	197.8 ± 112.3	NS
Hct, % $(n = 124)$	39.7 ± 7.8	38.5 ± 8.8	NS
BUN, mg/dL ( $n = 124$ )	33.5 ± 35.3	53.8 ± 42.0	< 0.01
Creatinine, mg/dL ( $n = 124$ )	$2.0 \pm 1.8$	$3.6 \pm 4.1$	< 0.01
LDH, U/L ( $n = 93$ )	323.9 ± 162.0	429.1 ± 520.9	NS
AST, U/L ( $n = 115$ )	45.8 ± 46.9	159.8 ± 409.1	< 0.01
ALT, U/L ( $n = 114$ )	24.6 ± 17.5	79.9 ± 181.6	0.02
ALP, U/L ( $n = 110$ )	82.9 ± 32.8	99.6 ± 62.6	NS
CK, U/L ( $n = 110$ )	505.0 ± 1,359.3	570.5 ± 1,046.2	NS
Amylase, U/L ( $n = 107$ )	$146.1 \pm 116.8$	434.8 ± 569.0	< 0.01
Phosphate, U/L ( $n = 18$ )	$3.3 \pm 1.9$	$4.9 \pm 13.0$	NS
pH(n = 111)	$7.42 \pm 0.08$	$7.32 \pm 0.16$	< 0.01
$PO_{2}$ , mmHg (n = 111)	129.6 ± 108.2	117.2 ± 98.8	NS
$PCO_{2}$ , mmHg (n = 111)	29.9 ± 7.7	$29.5 \pm 11.8$	NS
$HCO_3$ , mmol/L (n = 111)	$19.9 \pm 5.8$	$15.4 \pm 6.7$	< 0.01
$SaO_{2}, \% (n = 111)$	96.3 ± 3.5	94.4 ± 5.2	0.04
CRP, mg/dL ( $n = 26$ )	$7.2 \pm 10.4$	8.8 ± 11.6	NS
PT, sec $(n = 82)$	23.0 ± 34.6	$18.6 \pm 11.9$	NS
aPTT, sec $(n = 85)$	$49.8 \pm 41.3$	46.1 ± 21.8	NS
Sepsis <sup>†</sup>	19 (30.6)	33 (53.2)	0.02
Sepsis-induced hypotension $^{\dagger}$	4 (6.5)	13 (21.0)	0.03
Metabolic acidosis*	8 (12.9)	30 (48.4)	< 0.01

\*Mean  $\pm$  standard deviation; <sup>†</sup>the definitions of sepsis and sepsis-induced hypotension were adopted from reference 26; <sup>†</sup>metabolic acidosis = serum pH < 7.36 and HCO<sub>3</sub> < 24 mmol/L. ALP = alkaline phosphatase; ALT = alanine aminotransferase; aPTT = activated partial thromboplastin time; AST = aspartate aminotransferase;

ALP = alkaline phosphatase; ALT = alanine aminotransferase; aPTT = activated partial thromboplastin time; AST = aspartate aminotransferase; BUN = blood urea nitrogen; CK = creatine phosphokinase; CRP = C-reactive protein; ED = emergency department;  $HCO_3$  = bicarbonate; Hct = hematocrit; LDH = lactate dehydrogenase; NS = not significant;  $PCO_2$  = partial pressure of carbon dioxide in arterial blood;  $PO_2$  = partial pressure of oxygen in arterial blood; PT = prothrombin time;  $SaO_2$  = arterial oxygen concentration; WBC = white blood cell count.

	Number (%) of patients		n
	Survivors ( $n = 25$ )	Non-survivors ( $n = 31$ )	Ρ
Intestinal bowel-wall thickening with or without wall enhancement	nt 20 (80.0)	16 (51.6)	0.03
Intramural pneumatosis	1 (4.0)	14 (45.2)	< 0.01
Mesenteric or portal vein gas	2 (8.0)	7 (22.6)	NS
Mesenteric artery or vein thrombosis	8 (32.0)	6 (19.4)	NS

\*Adapted from reference 11. NS = not significant.

CT scan with intravenous contrast before surgical intervention. Table 3 demonstrates the most common CT imaging findings, including bowel-wall thickening (Figure 2) in 36 patients, intramural pneumatosis (Figure 3) in 15, mesenteric artery or vein thrombosis (Figure 4) in 14, and mesenteric or portal vein gas



**Figure 2.** Computed tomography findings: bowel-wall thickening in jejunal loops in the left abdomen (arrow).

(Figure 5) in 9. CT findings of intramural pneumatosis indicated a poor prognosis (p < 0.01). Fourteen patients (11.3%) underwent angiography, which revealed occlusion of the superior mesenteric artery with an embolus or thrombus (n = 8), and non-occlusive mesenteric vessels with hypoperfusion (6).



**Figure 3.** Computed tomography findings: annular intramural air (intramural pneumatosis) is demonstrated over multiple smallbowel loops in the lower abdomen (arrows).



Figure 4. Computed tomography findings: (A) patency (contrast-enhanced, arrow) of the proximal superior mesenteric artery; (B) intraluminal filling defect, which was found to be thrombosis during surgery, is noted in the distal superior mesenteric artery (arrow).



Figure 5. Computed tomography findings: (A) massive portal vein air is noted in both portal veins (arrow); (B) air was also demonstrated in the superior mesenteric vein and mesenteric vein (arrow).

	Number (%) of patients		n
	Survivors $(n = 62)$	Non-survivors ( $n = 62$ )	٢
Bowel resection	54 (87.1)	41 (66.1)	0.01
Laparotomy only	2 (3.2)	18 (29.0)	< 0.01
Revascularization	2 (3.2)	1 (1.6)	NS
Bowel resection and revascularization	4 (6.4)	1 (1.6)	NS
Laparotomy and revascularization	0 (0)	1 (1.6)	NS

NS = not significant.

Table 4 demonstrates that more survivors than non-survivors underwent bowel resection (87.1% vs 66.1%; p = 0.01). The rate of laparotomy only (i.e. open and closed, without revascularization) was significantly lower in survivors than non-survivors (3.2% vs 29.0%; p < 0.01), because of diffuse bowel gangrene and the catastrophic clinical condition of patients in the latter group.

Independent predictors of mortality, based on multiple logistic backward analyses, were old age (> 65 years; odds ratio, OR, 1.077; 95% confidence interval, CI, 1.013, 1.146; *p* = 0.02), bandemia (OR, 3.894; 95% CI, 1.160, 13.074; *p* = 0.03), elevated AST (OR, 4.532; 95% CI, 1.274, 16.122; *p* = 0.02) and BUN (OR, 7.219; 95% CI, 1.166, 44.696; *p* = 0.03), and metabolic acidosis (OR, 6.604; 95% CI, 1.804, 24.171; *p* < 0.01). Among 124 patients studied, 85 (68.5%) satisfied the criteria of old age, bandemia, elevated AST and BUN, and metabolic acidosis. In 16 of these 85 patients (18.8%) who had concurrent bandemia and elevated AST and BUN levels, the mortality rate was 93.8%. The sensitivity and specificity for predicting mortality were 68.9% and 74.2%, respectively, for bandemia (p < 0.001); 62.1% and 78.9% for elevated AST (*p* < 0.001); 88.5% and 38.3% for elevated BUN (*p* < 0.05); and 53.6% and 85.5% for metabolic acidosis (p < 0.001). The Hosmer-Lemeshow statistic was not significant, indicating little departure from a perfect fit ( $\chi^2 = 4.5$ ; df = 8; p = 0.809). Area under the receiver-operating characteristic (ROC) curve for the model was 0.852 (95% CI, 0.779, 0.925), suggesting good model discrimination (Figure 6).

#### Discussion

Early diagnosis and properly selected surgical interventions in acute mesenteric ischemia can reduce mortality.<sup>7,8</sup> Recognition of risk factors and comorbidities, and evaluation with appropriate



Figure 6. Receiver-operating characteristic (ROC) curve displaying the relationship between sensitivity and specificity for the regression-model analysis of independent variables predicting mortality from acute mesenteric ischemia.

diagnostic modalities, are mandatory for the early diagnosis of mesenteric ischemia in the ED. Patients typically complain of severe periumbilical pain, which is worse than that evident from physical examination.<sup>12,13</sup>

In this study, all enrolled patients had either abdominal pain or distension, which was often accompanied by vomiting (43.5% of patients), gastrointestinal bleeding (20.1%), diarrhea (19.4%), and hypotension (12.1%). Individual positive findings on physical examination, including abdominal tenderness, decreased or absent bowel sounds, or rebounding pain, were apparent in over 50% of patients in this study. However, only 32 patients (25.8%) had a typical presentation plus massive abdominal distension, decreased or absent bowel sounds, rebounding and localized tenderness, and muscle guarding indicating advanced bowel necrosis.<sup>13,14</sup> A high index of suspicion of vascular ischemia of the

bowel was essential to obtain an early diagnosis. In laboratory testing, metabolic acidosis,<sup>15–19</sup> hyperamylasemia,<sup>12</sup> and hyperphosphatemia,<sup>19,20</sup> were proposed as useful diagnostic supports for mesenteric ischemia and infarction. However, no single, abnormal laboratory parameter was identified in more than 90% of patients in this study. Univariate analysis demonstrated that bandemia, hyperamylasemia, hepatic and renal impairment, and metabolic acidosis were more frequently noted in non-survivors than survivors. Thus, our results suggest that the degree of multiple organ responses may be correlated with the severity of mesenteric ischemia or infarction. Any association between clinical presentation and abnormal laboratory results may therefore increase the index of suspicion for the early recognition of acute mesenteric ischemia.

Among diagnostic imaging studies, angiography is the gold standard for diagnosing acute mesenteric ischemia,<sup>21</sup> and must be performed early in patients in whom such ischemia is highly suspected. Plain abdominal imaging has shown very low sensitivity for detecting ischemic bowel disease.<sup>22,23</sup> However, because of its ready availability, improved quality, and similar sensitivity to angiography for detecting mesenteric ischemia,<sup>22</sup> CT has been suggested not only for diagnosing patients with a clinically high suspicion of mesenteric ischemia, but also for ruling out other causes of acute abdomen.<sup>4</sup> Our CT scan results suggest that intramural pneumatosis, an uncommon but more specific finding of infarcted bowel,<sup>11,22,24</sup> was associated with a poor prognosis.

Besides the need to diagnose acute mesenteric ischemia early, before infarction, knowledge of the clinical risk factors that predict prognosis enables emergency physicians to use more aggressive resuscitation and treatment strategies to improve outcomes. Aging was suggested to be an important predictor of adverse outcomes in patients with abdominal pain<sup>25</sup> or mesenteric ischemia,<sup>16</sup> as was also demonstrated in our study in which 99 patients (79.8%) were older than 65 years of age. In a previous report,<sup>16</sup> the proposed significant predictors of mortality in mesenteric infarction were an above-mean level of serum lactate and non-occlusive mesenteric ischemia. We identified several significant variables from our regression model, including old age, metabolic acidosis, bandemia, and elevated AST and BUN levels, that could independently predict an increased risk of death in patients with acute mesenteric ischemia. Based on these objective findings, considerably greater therapeutic intervention should be attempted in the ED in elderly patients with multiple organ dysfunction and acute mesenteric ischemia.

Our study has some limitations. First, data were collected from a retrospective chart review, and some clinical presentations or imaging studies may not have been documented in relevant medical records. Second, we excluded patients with widespread mesenteric infarction who did not undergo surgery because of a critical or retractable clinical condition, and patients with mild mesenteric ischemia who received only medical treatment were not enrolled.

In conclusion, acute mesenteric ischemia is a geriatric abdominal emergency that is associated with high morbidity and mortality if not recognized and treated early. In this study, only 1-quarter of patients had a clinical presentation and physical examination that was typically suggestive of acute mesenteric ischemia. A high index of suspicion and emergency diagnostic imaging, including CT scan or angiography, are mandatory for early diagnosis and favorable outcomes. For risk stratification of patients with acute mesenteric ischemia in the ED, we identified the following significant independent predictors of poor prognosis: aging, bandemia, metabolic acidosis, and elevated levels of AST and BUN. Thus, more intensive therapeutic management and intervention is advocated to improve outcomes in high-risk patients with acute mesenteric ischemia.

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