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Catheter Guided Thrombolysis of Acute Mesenteric Embolism in Cases of Paroxysmal Atrial Fibrillation-Review Article

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[1]Mesenteric ischemia is caused by blood flow that is insufficient to meet the metabolic demands of the visceral organs. The severity of ischemia and the type of organ involved depend on the affected vessel and the extent of collateral-vessel blood flow.

Despite advances in the techniques used to treat problems in the mesenteric circulation, the most critical factor influencing outcomes in patients with this condition continues to be the speed of diagnosis and intervention. Although mesenteric ischemia is an uncommon cause of abdominal pain, accounting for less than 1 of every 1000 hospital admissions, an inaccurate or delayed diagnosis can result in catastrophic complications; mortality among patients in whom this condition is acute is 60 to 80%.

Arterial obstruction, the most common cause of mesenteric ischemia, has both acute and chronic forms. Acute mesenteric ischemia constitutes a surgical emergency. It is associated with embolic occlusion in 40 to 50% of cases, with thrombotic occlusion of a previously stenotic mesenteric vessel in 20 to 35% of cases, and with dissection or inflammation of the artery in less than 5% of cases.

More than 90% of cases of chronic mesenteric ischemia are related to progressive atherosclerotic disease that affects the origins of the visceral vessels; treatment in such cases is focused on elective revascularization to avert the risk of complications and death associated with the development of acute ischemia.

[2]Mesenteric venous thrombosis, which accounts for 5 to 15% of cases of mesenteric ischemia, results in impaired venous outflow, visceral edema, and abdominal pain. Its causes include primary or idiopathic thrombosis; however, 90% of cases are related to thrombophilia, trauma, or local inflammatory changes that may include pancreatitis, diverticulitis, or inflammation or infection in the biliary system. Patients typically have a response to anticoagulation in combination with treatment for the underlying local or systemic processes. Surgical intervention is reserved for patients who are critically ill or whose condition is deteriorating; it is rarely required.

[3]The mesenteric circulation is a high-resistance vascular bed in which impaired regional perfusion owing to vasospasm can develop. The resulting ischemia is referred to as nonocclusive mesenteric ischemia. Although the incidence of nonocclusive mesenteric ischemia may be decreasing as awareness of the condition increases and as supportive therapies improve, it accounts for 5 to 15% of all cases of mesenteric ischemia. It is most often associated with cardiac insufficiency or low-flow states that occur after cardiac surgery or because of hypovolemia or heart failure, and it is increasingly identified in patients undergoing hemodialysis. Knowledge of its causes is critical, since misinterpretation of this condition may lead to worsened visceral perfusion and worsened mesenteric ischemia.

The mesenteric circulation is extremely complex. Three primary vessels — the celiac artery, superior mesenteric artery, and inferior mesenteric artery — interconnect through collateral networks between the visceral and nonvisceral circulations. These interconnections ensure that the loss of a single vessel does not lead to catastrophic malperfusion of the viscera.

Table 1. Causes of Altered Mesenteric Circulation.
Atherosclerosis
Arterial embolus
Arterial dissection
Thrombosis
Vasculitis
Mesenteric venous thrombosis
Poor cardiac output leading to low mesenteric flow
Inflammatory or other conditions affecting mesenteric vessels (e.g., pancreatitis, perforated ulcer, tumor)

[1]Reference: Clair DG, Beach JM. Mesenteric Ischemia. **N Engl J Med 2016;374:959-68.**

[4]Patients with acute mesenteric ischemia may initially present with classic "pain out of proportion to examination," with an epigastric bruit. Other patients may have tenderness with palpation on examination owing to peritoneal irritation caused by full thickness bowel injury. In a patient with abdominal pain of acute onset, it is critical to assess the possibility of atherosclerotic disease and potential sources of an embolus, including a history of atrial fibrillation and recent myocardial infarction.

Differentiation between arterial and venous obstruction is not always simple; however, patients with mesenteric venous thrombosis, as compared with those with acute arterial occlusion, tend to present with a less abrupt onset of abdominal pain. Risk factors for venous thrombosis that should be evaluated include a history of deep venous thrombosis, cancer, chronic liver disease or portal-vein thrombosis, recent abdominal surgery, inflammatory disease, and thrombophilia.

[5]Patients with chronic mesenteric ischemia can present with a variety of symptoms, including abdominal pain, postprandial pain, nausea or vomiting (or both), early satiety, diarrhea or constipation (or both), and weight loss. A detailed inquiry into the abdominal pain and its relationship to eating can be enlightening. Abdominal pain 30 to 60 minutes after eating is common and is often self-treated with food restriction, resulting in weight loss and, in extreme situations, fear of eating, or "food fear." Postprandial pain may, however, be associated with other intraabdominal processes, including biliary disease, peptic ulcer disease, pancreatitis, diverticular disease, gastric reflux, irritable bowel syndrome, and gastroparesis.

[6]In the diagnosis of mesenteric vascular disease, duplex ultrasonography has a high degree of reliability and reproducibility, with both a sensitivity and a specificity of 85 to 90%. It is an effective, low-cost tool that is helpful in the assessment of the proximal visceral vessels, although the results can be limited more distally.

Given its 95 to 100% accuracy, computed tomographic angiography (CTA) has become the recommended method of imaging for the diagnosis of visceral ischemic syndromes. Images of the origins and length of the vessels can be obtained rapidly, characterize the extent of stenosis or occlusion and the relationship to branch vessels, and aid in the assessment of options for revascularization.

[7]Catheter angiography, which was previously considered to be the standard method of diagnosis of mesenteric ischemia, has become a component of initial therapy. Angiography with selective catheterization of mesenteric vessels is now used once a plan for revascularization has been chosen. Single or complementary endovascular therapies, including thrombolysis, angioplasty with or without stenting, and intraarterial vasodilation, are then combined to restore blood flow.

Management and Outcome

[8]Endovascular strategies can theoretically restore perfusion more rapidly than can open repair and may thus prevent progression of mesenteric ischemia to bowel necrosis. Although the use of endovascular techniques is becoming more common, the comparative data on the results with the two approaches in patients with acute mesenteric ischemia are insufficient to show a clear advantage of one approach over the other.

Table 2: Acute mesenteric arterial ischemia.

Study	N° patients	Cause occlusion			Symptoms	Arteries involved	Primary treatment		
		Emboli	Thrombosis	Other			Thrombolysis	Thrombectomy	PTA/stent
Lim 2005 [2]	3	0%	33% (1)	66% (2)	Acute abdominal pain 100%, Nausea 33%, Vomiting 33%, Diarrhea 33%, Hematemesis 33%, Melena 33%	SMA	33% (1)	0%	66% (2)
Wyers 2007 [3]	2	0%	100%	0%	NS	SMA	NS	NS	100%
Acosta 2009 [4]	21	48% (10)	52% (11)	0%	NS	SMA	10% (2)	38% (8)	24% (5)
Schermerhorn (2000-2006) 2009 [5]	1857	NS			NS	NS	0%	0%	100%
Block 2010 [6]	42	29% (12)	62% (26)	9% (4)	Abdominal pain, Vomiting, Diarrhea, Hematochezia	SMA	NS		
Bjornsson 2011 [7]	34	82% (28)	18% (6)	0%	Acute abdominal pain 30% (10), Sudden abdominal pain 36% (12), Bloody stools 3% (1), Diarrhea 36% (12), Vomiting 62% (21), Atrial fibrillation 62% (21)	SMA	100%	0%	0%
Arthurs 2011 [8]	56	35%	65%	0%	Abdominal pain 92%, Nausea 69%, Emesis 51%, Bloody diarrhea 31%	SMA 66% SMA+ celiac artery 34%	48%	11%	32%
Ryer* 2012 [9]	11	31% (29)	54% (50)	0-6%	Abdominal pain (91-98%), Abdominal tenderness (58-79%), Diarrhea (38-23%), Nausea (42-38%), Vomiting (36-27%), LGIB (16-13%)	SMA +- celiac artery	67% (2)-25% (2)	0-13%	33% (1)- 62.5% (5)
Beaulieu 2014 [10]	165	NS	NS	NS	Lactic acidosis 11.4%, ARDS 7%, SIRS 5.9%, Hypotension 4.9%	NS	NS		
Jia 2014 [11]	21	NS	NS	NS	Abdominal pain and no evidence of advanced bowel ischemia sign	SMA	0%	100%	0%
Barrera 2015 [12]	9	33%	45%	22%	No peritoneal irritation	Small intestine	18%	NS	NS
Karkkainen 2015 [13]	50	36% (18)	64% (32)	0%	Abdominal pain 94%, Abdominal distension 26%, Diarrhea 48%, Vomiting 56%, Paralytic ileus 20%, GI bleeding 14%, Acute kidney injury 6%, Clinical features of peritonitis 14%	SMA SMA + celiac artery SMA + IMA	0%	40% (20)	60% (30)
Raupach 2016 [14]	37	100%	0%	0%	Abdominal pain Diarrhea, Bloody diarrhea, Nausea, Emesis	SMA	0%	100%	0%

*1990s-2000s

PTA, percutaneous transluminal angioplasty; LPT, laparotomy; LPS, laparoscopy; NS, not specified; SMA, superior mesenteric artery; ET, endovascular therapy; TIA, transient ischemic Attack; GI, gastrointestinal; ARDS, acute respiratory disease syndrome; SIRS, systemic inflammatory response syndrome

Secondary treatment			Technical succ	Clinical succ	Recurrence rate	Complications	Mortality	Lpt/Lps and/or resection		
Thrombolysis	Thrombectomy	PTA/Stent						LPT	LPS	Resection
NS			100% (3)	66% (2)	0%	Ischemic hepatitis 33% (1) Acute renal failure 33% (1) Malnutrition 33% (1)	0%	33% (1)	0%	33% (1)
NS			100%	NS	NS	NS	100%	0%	0%	100%
25% (2)	NS	NS	NS	NS	NS	SMA dissection distal emboli in arterial branch	14% (3)	67% (14)	8% (1)	43% (9)
NS			NS	NS	NS	Acute renal failure 11.4%, Acute myocardial infarction 5%, Cardiac 2.1% Stroke 1%, Peripheral vascular 0.5%, respiratory 1.1% Hemorrhage 2.4%	16%	NS	NS	28%
NS			79%	NS	NS	Groin hematoma (2) Renal embolization (1) Femoral artery occlusion (1)	27%	73% (27)	0%	40.5% (15)
0%	35.2% (12)	8.8% (3)	88%	NS	NS	Bleeding complication 15% (5)	26% (9)	38% (13)	0%	24%(8)
0%	12% (of thrombolysis)	33% (of thrombolysis) 22% (of thrombectomy)	87%	NS	NS	Acute renal failure 27% pulmonary failure 27% Myocardial infarction 2% GI bleeding 7%	36% ET success 50% ET failure	31%	NS	NS
NS			NS	NS	1%	Overall 73-63% Pulmonary 16-32% Renal 12% Neurologic (TIA/stroke) 5-2% GI 7-26%	17%-27%	63% (7)	NS	45% (5)
NS			NS	Survival 39.9%	NS	NS	24.9%	NS	NS	14.4%
28.6% (6)	NS	NS	Cerebral infarction 5% (1) short bowel syndrome 5% (1)	9.5% (2)	29% (6)	NS	24% (5)	-	-	-
NS	33.3% (3/9)	ET+Chir 67% (6)	NS	NS	NS	NS	33.3% (3)	NS	NS	33.3% (3)
NS			88% (44)	NS	NS	Access site bleeding 4% (2), Intra abdominal bleeding 2% (1), New GI bleeding 10% (5), Stroke 2% (1), Myocardial infarction 4% (2) Heart failure 12% (6) Acute kidney injury 8% (4) Pulmonary failure 4% (2)	30%	40%	NS	34%
5% (2)	0%	5% (2)	91.9%	NS	NS	Groin hematoma 5.4% (2) Renal embolization 3% (1) Femoral artery occlusion 3% (1)	27%	73% (27)	NS	40.5% (15)

[2]Reference: **Ierardi AM, Tsetis D, Sbaraini S, et al. The role of endovascular therapy in acute mesenteric ischemia.** *Ann Gastroenterol 2017; 30 (4): 526-533*

In a recent paper [2]Ierardi AM et al analysed the role of endovascular therapy success for acute mesenteric arterial ischemia and found good technical and clinical success rates with fewer complications as mentioned above.

[9]Recent data show that Endovascular strategy may be most appropriate for patients with ischemia that is not severe and those who have severe coexisting conditions that place them at high risk for complications and

death associated with open surgery. Endovascular therapy is a very successful, minimally invasive approach that provides relief of symptoms in up to 95% of patients and has a lower rate of serious complications than open repair. In all cases of mesenteric ischemia, any evidence of peritonitis, stricture, or gastrointestinal bleeding should trigger an exploratory laparotomy to assess for the possibility of bowel necrosis and the need for operation.

[10]Boley et al first proposed an aggressive approach to SMA occlusion with rapid angiography and infusion of intraarterial vasodilators. Simonetti et al first described the use of intraarterial fibrinolysis in acute mesenteric ischemia. There are a limited number of studies evaluating the long-term efficacy of percutaneous interventions in acute mesenteric ischemia, with none reporting the clinical outcomes past 1 year. Moteki et al suggested that, in cases of occlusions at or proximal to the major branches of the SMA, success with thrombolytic therapy is seen with symptoms duration of 5 hours to as long as 60 hours. Mechanical thrombectomy can be helpful, but it carries an additional risk of distal emboli.

Based upon recent evidences [3]Acosta S et al suggested a simplifies algorithm for evaluation and management of acute mesenteric ischemia of SMA occlusion which has very good accuracy and precision.

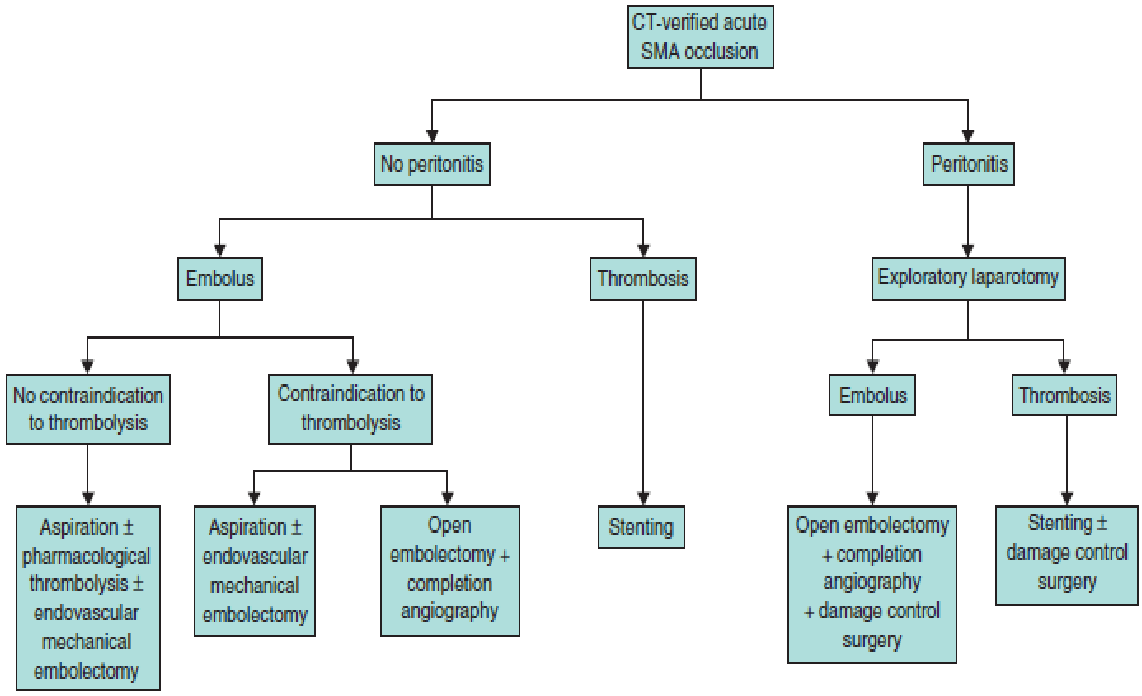

Figure 3 : Algorithm for management of patients with acute superior mesenteric artery (SMA) occlusion. CT, computed tomography.

[3]Reference: Acosta S, Bjorck M. Modern treatment of acute mesenteric ischaemia. BJS 2014; 101: e100–e108.

Table-3 1

Case Reports and Small Series of Thrombolytic Therapy for Acute Superior Mesenteric Artery Thromboembolism

Study	Year	Patient No.	Sex—Age (years)	History	Duration of Presentation (hours) Before Angiography	Bloody Stools	Angiography Site (cm from origin)	Angiography Occlusion
Jamieson *et al* (41)	1979	1	F—78	AF, MI, CAD	14	–	P, 6 cm	T (−)
Theiss *et al* (42)	1982	2	F—77	AF, MI, EE+	<1	NR	D	T (+)
Flickinger *et al* (43)	1983	3	M—60	AF, MI, CHF, CAD	3	NR	P	I
Pillari *et al* (44)	1983	4	M—60	CHF, AF	72	NR	P, 6 cm	(+) (thrombus)
Vujic *et al* (45)	1984	5	F—57	AMI, SMAE, DM	<1	–	P, 3 cm	T (−)
Kohler *et al* (46)	1985	6	M—56	AOD, EE+	_	NR	D	I (thrombus)
Fernandez *et al* (47)	1990	7	M—62	MI, AOD, CI, FVIII	4	+	_	NR
Hillers *et al* (48)	1990	8	M—80	AF, HT, PUD, CAD	<24	+	D	NR
Ramirez *et al* (49)	1990	9	M—50	HT, EE	8	+	P	NR
Rodde *et al* (50)	1991	10	M—70	MI, CAD, EE+	_	NR	D	I
Schoenbaum *et al* (62)	1992	11	M—79	AF, MI, EE, AA	48	+	P	I (thrombus)
		12	F—62	AF, HVD, RF, EE+	_	NR	P	I (thrombus)
		13	F—83	AF, MI, EE	24	NR	D	T (−)
		14	M—63	MI	_	NR	P	T (−) (thrombus)
Bonardelli *et al* (51)	1994	15	M—84	CAD, EE	<1	–	D	NR
Boyer *et al* (52)	1994	16	M—66	AF, EE+	6	+	P, 3 cm	I
McBride & Gaines (53)	1994	17	F—80	AF, HT	6–144	–	P, 5 cm	I
Nathan *et al* (54)	1995	18	F—73	MI, COPD	10	NR	P, 6 cm	I
Turegano *et al* (63)	1995	19	M—62	AF, CM	5	+	P	I
		20	M—68	AF, CI	4	–	D	NR
Gallego *et al* (64)	1996	21	M—65	HT, EE	8	+	P, 2–3 cm	NR
		22	F—69	AF, HT	6	+	D	T
Kwauk *et al* (55)	1996	23	M—66	AF, MI, HT	6	–	D	I
Regan *et al* (56)	1996	24	M—76	MI, HT, HC	3	NR	P	I (thrombus)
Badiola & Scopetta (57)	1997	25	M—82	SMAE, AF, MI, CI	2	NR	D	I
Hirota *et al* (58)	1997	26	M—41	AF	24	NR	P, 4 cm	I
Simo *et al* (65)	1997	27	M—62	AF, CM	5	+	P	Major (T/I)
		28	M—68	AF	18	–	D	Minor (I)
		29	M—77	AF	18	+	D	Minor (I)
		30	M—72	AF	6	–	D	Minor (I)
		31	F—64	HVD	8	+	D	Minor (I)
		32	F—77	AF	14	+	D	Minor (I)
		33	M—82	AF, HVD	8	–	D	Minor (I)
		34	F—76	HVD	6	+	D	Minor (I)
		35	F—79	AF	6	–	P	Major (T/I)
		36	M—76	AF, HVD	7	–	D	Minor (I)
Yamaguchi *et al* (66)	1999	37	M—44	AF, CI	12	2/8 pts	D	T
		38	F—91	AF, MI, EE+	24	"	D	T
		39	F—86	AF	8	"	D	T
		40	M—67	AF, HT, EE	18	"	D	T
		41	M—59	AF, HT	17	"	P	T
		42	F—59	AF, HVD, CI	8	"	D	I
		43	M—69	AF, MI, CI	60	"	P	T
		44	F—76	AF, AA	15	"	P	T
Dominquez *et al* (59)	2000	45	M—NR	AF	6	NR	P, 6 cm	NR
Mellander *et al* (67)	2001	46	M—87	EE	6	–	P few cm	NR
		47	F—78	EE, DM, CML	12	NR	P, 5 cm	NR
Bakarate *et al* (60)	2002	48	M—84	AF	12	NR	P, 7 cm	T (+)

Note.—AA = aortic aneurysm; AD = aortic dissection; AF = atrial fibrillation; AMI = acute mesenteric ischemia; AOD = peripheral arterial occlusive disease; CAD = coronary artery disease; CHF = chronic heart failure; CI = cerebral infarction; CM = cardiomyopathy; CML = chronic myeloid leukemia; COPD = chronic obstructive pulmonary disease; DM = diabetes mellitus; EE = embolic event in extremity; EE+ = embolic event concomitant with acute mesenteric ischemia; FVIII = factor VIII deficiency; HC = hypercholesterolemia; HT = hypertension; HVD = heart valve disease; MI = myocardial infarction; PTA = percutaneous transluminal angioplasty; PUD = peptic ulcer disease; RF = rheumatic fever; SMAE = superior mesenteric artery embolism; SVT = supraventricular tachyarrhythmia; P = SMA occlusion proximal to the middle colic artery; D = SMA occlusion distal to the middle colic artery; T = total occlusion; T (−) = almost total occlusion; (+) = multiple occlusions; I = incomplete/partial occlusion; T/I = total or incomplete occlusion; Y = year; mo = month; d = day; + = bloody diarrhea present; − = bloody diarrhea absent; NR = not reported.

[4]Reference : **Schoots IG, Levi MM, Reekers JA, et al. Thrombolytic Therapy for Acute Mesenteric Artery Occlusion. J Vasc Interv Radiol 2005;** 16:317–329.

Table-4

Outcome of Thrombolytic Therapy for Acute Superior Mesenteric Artery Thromboembolism

Patient No.	Drug	Pain Relief (h)	Technical Success (h)	Complications	Laparotomy; Findings; Procedure	Mortality/Morbidity
1	SK	<4	Yes, 36–72h	No	No	Survived
2	UK	<	Yes, <2h	Bleeding (cath.)	Yes, resection necrosis, 25 cm	Died of MI (3d), no abdominal symptoms
3	SK	<	Yes, 34–60h	No	No	Died of CHF, SVT (7d) no abdominal symptoms
4	SK	<	Yes, 36h	No	No	Survived
5	SK	<<	Yes, <30h	Bloody diarrhea	Yes, exploratory, normal	Survived
6	UK	<2	Yes, <3h	No	No	Survived
7	UK	<18	Yes, <18h	No	No	Survived
8	SK	<60	Yes, 30–60h	No	No	Survived
9	UK	<	Yes, <4h	No	No	Survived
10	UK	<12	Yes, <1h	No	No	Survived
11	UK	NR	Yes, 12–36h	No	No	Survived
12	UK	<hours	Yes, 12–40h	No	No	Survived
13	UK	<2	Yes, 2–24h	No	Yes, mild ischemia, no resection	Survived
14	UK	<2	Yes, <10h	No	Yes, necrosis, resection 60 cm	Survived
15	UK	<1	Failed, <1h	in 2h > abdominal pain	Yes, embolectomy, resection necrosis 20 cm	Survived
16	rtPA	NR	Yes, 20–48h	No	Yes, exploratory 72h, normal	Survived
17	SK	NR	Yes, 5–17h	No	No	Survived
18	SK rtPA	=, >	Partial <1h	Multiple emboli, abdominal pain, 24 h shock	Yes, resection of 2 non-perforated ischemic bowel segments	Died of shock (5d) non-perforated necrosis
19	UK	<½	Yes, 14–24h	No	No	Survived
20	UK	<½	Yes <15h	No	No	Survived
21		<4				
22	UK	4–16	Yes, 4–16h	No	No	Survived
	UK	<4	Yes, 4–12h	Bloody diarrhea, cerebral embolism	No	Survived
23	SK	<<	Yes, <18h	Renal embolism	No	Survived, died of MI (2 mo)
24	UK	<<	Yes, 20–48h	No	Yes, exploratory laparoscopy 24h	Survived
25	UK	<6	Yes + partial	Hematuria, hematoma (catheter)	No	Survived
26	UK	NR	Yes, 1–10h	No	No	Survived
27	UK	<1	Yes, 12–24h	No	No	Survived
28	UK	<1	Yes, 9–15h	No	No	Survived
29	UK	<1	Yes, 6–18h	No	No	Survived
30	UK	<1	Yes, 12–18h	No	No	Survived
31	UK	<1	Yes, 4–12h	No	No	Survived
32	UK	6	Yes, 24–29h	No	Yes, resection necrosis, 40 cm at 72h	Survived, abscess iliac fossa (1 mo), fistula (6 mo)
33	UK	<1	Yes	No	Yes, normal	Died of shock (20d), no abdominal symptoms
34	UK	>15	Partial	Infusion was stopped at 15h, >abdominal pain	Yes, resection necrosis, second look 24h	Survived
35	UK	>8	Yes	Infusion was stopped at 8h, >abdominal pain	Yes, resection necrosis, second look 24h	Survived
36	UK	<1	Yes, 12–20h	No	No	Survived
37	UK	NR	Yes	No	No	Survived
38	UK	NR	Yes	No	No	Survived
39	UK	NR	Failed	Shower emboli	No	Died of shock (1d)
40	UK	NR	Yes	No	Yes, atheroma	Survived, SMA bypass (1 mo)
41	UK	NR	Yes	No	Yes, normal	Survived
42	UK	NR	Yes	No	No	Survived, leg emboli
43	UK	NR	Yes	No	No	Survived, died of MI (1 mo)
44	UK	NR	Failed	Extravasation	Yes, resection necrosis	Survived, died of CI (1 mo)
45	UK	<5	Yes, 5–10h	No	No	Survived
46	rtPA	<3	Yes, 3–8h	No	No	Survived
47	UK	<hours	Yes, reocclusion	Reocclusion after 8h, successful thrombolysis, + thrombosis leg (32h)	No	Survived
48	UK	<1	Yes, 6–18h	No	Yes, exploratory 48h, normal, shock developed, 2nd exploratory 96h, normal	Survived

Note.—SK = streptokinase; UK = urokinase; rtPA = recombinant tissue plasminogen activator; < = decrease (within)/less than; << = rapid decrease (duration was not reported); > = increase (within); y = year; mo = month; d = day; h = hour; NR = not reported. Technical (angiographic) success was defined as resolution of superior mesenteric artery occlusion. Occlusion of the superior mesenteric artery was distinguished in proximal and distal occlusion, defined as thromboembolism located proximal or distal to the middle colic artery. Resolution of the occlusion was defined as post-procedural, normal angiographic morphology of the vessel or minimal presence of clot remnants without compromising arterial flow. Procedure-related complications were scored separately. Clinical success was defined by angiographically demonstrated resolution of the occlusion, disappearance of abdominal pain during thrombolytic therapy, absence of peritoneal signs at physical examination, treatment without surgical intervention, and hospital discharge. Overall survival was defined as hospital discharge, with or without additional surgical intervention.

[4]Reference : Schoots IG, Levi MM, Reekers JA, et al. Thrombolytic Therapy for Acute Mesenteric Artery Occlusion. J Vasc Interv Radiol 2005; 16: 317-329.

[4]Schoots IG et al reviewed in this paper small case series of different endovascular and surgical therapies for acute arterial mesenteric ischemia and concluded that those presenting early without complications of bowel ischemia have good endovascular outcome comparable to surgical therapy.

Table 5 Published series of acute mesenteric ischemia

First author	*Publication year*	*No. of patients*	*Mortality rate*
Foley	2000	21	24%
Mamode	1999	57	81%
Newman	1998	98	60%
Urayama	1998	34	35%
Klempnauer	1997	90	66%
Voltolini	1996	47	72%
Konturek	1996	28	96%
Ward	1995	34	45%
Deehan	1995	43	70%
Levy	1990	62	40%
Batellier	1990	65	51%
Bapat	1990	20	40%
Finucane	1989	32	69%
Sitges-Serra	1988	83	71%
Wilson	1987	102	92%
Lazaro	1986	23	27%
Andersson	1984	60	82%
Sachs	1982	30	77%
Krausz	1978	40	78%
Kairaluoma	1977	44	70%
Boley	1977	30	46%
Smith	1976	23	91%
Singh	1975	32	81%
Ottinger	1967	136	92%
Total		1234	69%

[5]Reference : Park WM, Gloviczki P, Cherry KJ, et al. **Contemporary management of acute mesenteric ischemia, Factors associated with survival.** J Vasc Surg 2002; 35: 445-52.

[5]Park WM et al in their paper looked into prognostic factors after endovascular or surgical therapies for acute mesenteric ischemia and concluded that older age, nonocclusive mesenteric ischemia, late presentations all have adverse outcomes.

Table 6 : Case series mortality - Asia

Author	**Number of patients**	**Mortality (%)**
Li X *et.al*	40	50
Zhang Z *et.al*	42	15.3
Haghighi PH *et.al*	105	50.4
Alhan E *et.al*	107	55.1
Aliosmanoglu *et.al*	98	42.1
Eris C *et.al*	52	67

[6]Reference : *Stephen E, Sarfaraz ZK, Abdelhedy I, et al.* **Acute Mesenteric Ischemia: The What, Why, and When?.** Indian J Vasc Endovasc Surg 2016;3:24-8.

Only few papers have come about Asian population on mesenteric ischemia. [6]Stephen E et al reviewed Asian population small case series related articles and found similar characteristics and presentations of acute mesenteric ischemia compared to their western counterparts except that outcomes are slightly poorer due to lack of awareness and late presentations with most of the people land into chronic form of mesenteric ischemia.

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