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OPTIMIZATION OF DIAGNOSIS AND SURGICAL APPROACH IN ACUTE MESENTERIC ARTERY THROMBOSIS: A PROGNOSTIC SCALE FOR INTESTINAL VIABILITY AND A DIFFERENTIATED TREATMENT ALGORITHM




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ЎТКИР МЕЗЕНТЕРИАЛ АРТЕРИЯЛАР ТРОМБОЗИДА ТАШХИС ВА ЖАРРОҲЛИК ТАКТИКАСИНИ ОПТИМАЛЛАШТИРИШ: ИЧАК ҲАЁТИЙЛИГИНИ БАШОРАТЛАШ ШКАЛАСИ ВА ДИФФЕРЕНЦИАЛ ДАВОЛАШ АЛГОРИТМИ

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ОПТИМИЗАЦИЯ ДИАГНОСТИКИ И ХИРУРГИЧЕСКОЙ ТАКТИКИ ПРИ ОСТРОМ ТРОМБОЗЕ МЕЗЕНТЕРИАЛЬНЫХ АРТЕРИЙ: ПРОГНОСТИЧЕСКАЯ ШКАЛА ЖИЗНЕСПОСОБНОСТИ КИШЕЧНИКА И ДИФФЕРЕНЦИРОВАННЫЙ АЛГОРИТМ ЛЕЧЕНИЯ

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Резюме. *Мақсад: ичак ҳаётийлигини башоратлаш шкаласи ва дифференциал диагностик-даволаш алгоритмини ишлаб чиқиш орқали ўткир мезентериал қон айланиши бузилиши (ЎМҚАБ) бўлган беморларни ташхислаш ва жарроҳлик даволаш натижаларини яхшилаш. Материал ва усуллар: 2013–2024 йилларда Самарқанддаги иккита клиник марказда ўтказилган ретроспектив-проспектив қиёсий когорт тадқиқотга ЎМҚАБ бўлган 165 нафар бемор киритилди: назорат гуруҳи (n=82, анъанавий тактика) ва асосий гуруҳ (n=83, янги алгоритм). Таққослаш гуруҳини бошқа этиологияли «ўткир қорин» синдроми бўлган 60 нафар бемор ташкил этди. Лаборатор маркерлар, контрастли компьютер томографияси (КТ), 118 та резекция препаратининг морфологик ва иммуногистокимёвий текшируви, ROC-таҳлил, bootstrap валидацияли кўп омилли логистик регрессия ва Kaplan–Meier таҳлили қўлланилди. Натижалар: томир окклюзиясини D-димер (AUC 0,846; чегара 1540 нг/мл), ишемик шикастланиш чуқурлигини эса лактат (AUC 0,874; чегара 3,8 ммоль/л) энг яхши акс эттирди; КТнинг сезгирлиги 94,5%, спецификлиги 88,3% ни ташкил этди. Беш компонентли шкала (ичак девори контраст кучайишининг йўқлиги, лактат >3,8 ммоль/л, перитонеал белгилар, pH <7,31, ЛДГ >485 ХБ/л) ≥6 балл чегарасида қайтмас некрозни 86,1% сезгирлик ва 84,2% спецификлик билан башорат қилди (AUC 0,910; bootstrap бўйича тузатилган AUC 0,892). Алгоритм жорий этилгач, 30 кунлик леталлик 59,8% дан 39,8% гача (p=0,010), тирик қолганлар орасида калта ичак синдроми 54,5% дан 26,0% гача (p=0,008) камайди, бир йиллик яшовчанлик 30,5% дан 51,8% гача ошди (p=0,006); асосий гуруҳнинг тузатилган OR кўрсаткичи 0,49 (95% ИО 0,25–0,96) бўлди. Хулоса: прогностик шкала ва дифференциал алгоритм (диффуз перитонит бўлмаганда аввал реваскуляризация, ичак ҳаётийлигини такрорий баҳолаш, ≥6 баллда босқичли операция) кўп тармоқли шифохонада ЎМҚАБни даволаш натижаларини яхшилайди.*

Калит сўзлар: *ўткир мезентериал ишемия; мезентериал артериялар тромбози; ичак некрози; прогностик шкала; реваскуляризация; ичак резекцияси; калта ичак синдроми; D-димер; лактат; APACHE II.*

Abstract. *Aim: to improve the diagnosis and surgical treatment of acute mesenteric circulatory disorder (AMCD) by developing a prognostic scale for intestinal viability and a differentiated diagnostic-and-treatment algorithm. Materials and methods: a retrospective–prospective comparative cohort study was carried out at two clinical centres in Samarkand (2013–2024) and included 165 patients with AMCD: a control group (n=82, conventional tactics) and a main group (n=83, the new algorithm). Sixty patients with an acute abdomen of other aetiology formed a diagnostic comparison group. Laboratory markers, contrast-enhanced computed tomography (CT), morphological and immunohistochemical examination of 118 resection specimens, ROC analysis, multivariable logistic regression with bootstrap validation and*

Kaplan–Meier analysis were used. Results: vascular occlusion was best reflected by D-dimer (AUC 0.846; threshold 1540 ng/mL) and the depth of ischaemic injury by lactate (AUC 0.874; threshold 3.8 mmol/L); CT sensitivity was 94.5% and specificity 88.3%. The five-component scale (absence of bowel-wall contrast enhancement, lactate >3.8 mmol/L, peritoneal signs, pH <7.31, LDH >485 U/L) at a cut-off of ≥6 points predicted irreversible necrosis with 86.1% sensitivity and 84.2% specificity (AUC 0.910; bootstrap-corrected AUC 0.892). After implementation of the algorithm, 30-day mortality fell from 59.8% to 39.8% (p=0.010) and short bowel syndrome among survivors from 54.5% to 26.0% (p=0.008), while one-year survival rose from 30.5% to 51.8% (p=0.006); the adjusted OR for the main group was 0.49 (95% CI 0.25–0.96). Conclusion: the prognostic scale and the differentiated algorithm (revascularization first in the absence of diffuse peritonitis, repeated viability assessment, staged surgery at a score of ≥6 points) improve the outcomes of AMCD in a multidisciplinary hospital.

Keywords: *acute mesenteric ischaemia; mesenteric artery thrombosis; intestinal necrosis; prognostic scale; revascularization; bowel resection; short bowel syndrome; D-dimer; lactate; APACHE II.*

Acute disturbance of mesenteric circulation is among the most severe conditions encountered in emergency abdominal surgery: pooled international data show short-term mortality of about 60%, and arterial occlusion – thrombosis or embolism – accounts for the majority of cases [1, 6, 8].

The principal driver of unfavourable outcome is late diagnosis. Reliance on the signs of established necrosis – abdominal distension, absent peristalsis, peritoneal signs – means diagnosing the disease at a stage when the bowel can no longer be salvaged, whereas the early sign of pain disproportionate to physical findings is characteristic of the stage of reversible ischaemia. The absence of an objective means of distinguishing reversible ischaemia from transmural necrosis produces two opposite errors: preservation of non-viable bowel with ongoing necrosis, or resection of viable segments with resultant short bowel syndrome [2, 5].

International guidance on acute mesenteric ischaemia has been updated twice in recent years. The World Society of Emergency Surgery guidelines reinforced the need for immediate contrast-enhanced computed tomography whenever the diagnosis is suspected and endorsed endovascular revascularization as the first choice for arterial occlusion where sufficient expertise is available [1]. The 2025 clinical practice guidelines of the European Society for Vascular Surgery, succeeding the 2017 edition, consolidated 102 recommendations on the management of arterial, venous and non-occlusive mesenteric disease [3, 6]. The multicentre prospective AMESI study, which enrolled 705 patients at 32 hospitals and confirmed the diagnosis in 418, found a median interval of 24 hours from symptom onset to hospitalization and 6 hours from hospitalization to diagnosis, with nearly half of patients dying during the index admission; operation without revascularization remained the predominant approach [7]. A systematic review and meta-analysis of incidence and outcomes across 163 studies estimated pooled short-term mortality at 59.6% (95% CI 55.5–63.6%), confirming that the geographic spread of reported incidence figures more likely reflects case-ascertainment completeness than a true difference in disease frequency [8].

Systematic reviews of candidate biomarkers — ischaemia-modified albumin, interleukin-6, procalcitonin and intestinal fatty-acid-binding protein — have consistently concluded that no single marker possesses sufficient accuracy in isolation, while studies of marker combinations remain scarce. Experience from dedicated intestinal-ischaemia units indicates that outcome is shaped more by the organization of multidisciplinary emergency care than by the surgical technique itself, and that a stepwise strategy combining revascularization with staged, on-demand resection improves survival compared with resection-first approaches [2, 4, 5]. Nonetheless, an instrument for distinguishing preoperative reversible ischaemia from transmural necrosis, threshold values of biomarkers for the different clinical forms, and the adaptation of the specialized-centre model to resource-limited multidisciplinary hospitals remain open questions. These gaps define the rationale for the present study.

The burden described in this cohort is not unique to the study region: pooled international data indicate a mean incidence of roughly six cases per 100,000 person-years and around five admissions per 10,000 hospitalizations, with short-term mortality that has fallen only modestly, from about 69% before the year 2000 to about 55% thereafter, despite four decades of technical progress in imaging and endovascular therapy [8]. This plateau in outcome, occurring against a background of clear improvement in diagnostic technology, is itself evidence that the rate-limiting step lies not in the availability of tests but in how quickly and consistently they are translated into a structured operative decision — the specific gap that the present study set out to close for a multidisciplinary, non-specialized hospital setting.

Aim of the study: to improve the results of diagnosis and surgical treatment of patients with acute mesenteric circulatory disorder through the development of a prognostic scale for intestinal viability and a differentiated diagnostic-and-treatment algorithm suitable for a multidisciplinary hospital setting.

Table 1. Sex, age and severity of condition on admission

Characteristic	Control, n = 82	Main, n = 83	p
Male sex	46 (56.1%)	48 (57.8%)	0.823
Female sex	36 (43.9%)	35 (42.2%)	0.823
Age, mean ± SD, years	67.8 ± 11.4	66.9 ± 10.9	0.606
Age range, years	38–89	36–87	–
Age ≥ 70 years	38 (46.3%)	34 (41.0%)	0.486
ASA class ≥ III	75 (91.5%)	74 (89.2%)	0.617
APACHE II, mean ± SD, points	18.7 ± 6.1	17.9 ± 5.8	0.389
SOFA, Me [Q1; Q3], points	7 [5; 10]	7 [4; 9]	0.348

Note: ASA – American Society of Anesthesiologists physical status; SD – standard deviation; Me [Q1; Q3] – median and interquartile range.

The objectives were: (1) to characterize the incidence, aetiological structure and clinical course of AMCD and to identify the causes of discrepancy between primary and final diagnosis; (2) to determine, separately and with calculation of threshold values, the diagnostic value of laboratory indices and contrast-enhanced computed tomography for detecting vascular occlusion and for assessing the depth of ischaemic bowel-wall injury; (3) to develop, on the basis of clinical, laboratory, radiological and morphological criteria, a prognostic scale for irreversible intestinal necrosis and to perform its internal validation; (4) to substantiate a differentiated surgical tactic and to evaluate, by multivariable analysis, its effect on the immediate and long-term results of treatment.

Materials and methods. *Study design and setting*. The work was carried out at two clinical bases — the multidisciplinary clinic of Samarkand State Medical University and the Samarkand branch of the Republican Scientific Centre of Emergency Medical Care. It is an open comparative cohort study with a retrospective and a prospective component, covering the period from 2013 to 2024, with follow-up of survivors continued through 31 December 2025. In total, 187 case records of patients with suspected AMCD were reviewed; 22 observations were excluded according to pre-specified exclusion criteria (incomplete records, death within 24 hours before any diagnostic work-up, or a final diagnosis unrelated to mesenteric circulation), leaving a clinical cohort of 165 patients. An additional 60 patients admitted with an acute abdomen of other aetiology were examined and included in a diagnostic comparison group, bringing the total diagnostic array to 225 observations, used to calculate the specificity of the diagnostic criteria under a four-field design. The primary endpoint was 30-day mortality; secondary endpoints were the length of resected bowel, the incidence of short bowel syndrome, the relaparotomy rate, and one-year survival. The study protocol was approved by the Local Ethics Committee of Samarkand State Medical University (Protocol No. 12, 18 December 2018).

Patients and grouping. The clinical cohort was divided according to the period of hospitalization: a control group of 82 patients (49.7%) treated according to conventional tactics before the algorithm was implemented, and a main group of 83 patients (50.3%) managed according to the developed algorithm. The comparability of the two groups at baseline was verified across sex and age distribution, physical status by the American Society of Anesthesiologists (ASA) classification, and severity scores on the APACHE II and SOFA scales (Table 1).

No significant between-group differences were found for any of the recorded baseline parameters, which supports the validity of subsequent outcome comparisons. Patients older than 60 years accounted for 124 observations (75.2%) and patients older than 70 years for 72 (43.6%); ASA class III or higher was recorded in 149 patients (90.3%), characterizing the cohort as predominantly elderly and burdened with comorbidity: coronary artery disease in 94 patients (57.0%), arterial hypertension in 112 (67.9%), and atrial fibrillation in 60 (36.4%).

Diagnostic methods. Clinical evaluation included history-taking with clarification of the time of pain onset, assessment of pain intensity on a visual-analogue scale, detection of peritoneal signs, and grading of the severity of the condition by the ASA classification and by the APACHE II and SOFA scales. Laboratory evaluation comprised serial measurement of lactate, D-dimer, procalcitonin, C-reactive protein, leukocyte count, lactate dehydrogenase and acid-base status at six time points — on admission and at 6, 12, 24 and 72 hours and on day 7. Instrumental evaluation included plain abdominal radiography; ultrasonography with Doppler assessment of the visceral arteries (Mindray DC-70, 2.5–5.0 MHz convex and 5–12 MHz linear probes); contrast-enhanced computed tomography on a 64-slice scanner (Siemens SOMATOM Definition AS) using 80–100 mL of iohexol (350 mg iodine/mL) at 4–5 mL/s, with arterial-phase acquisition at 25–30 s and portal-venous-phase acquisition at 60–70 s delay, 0.6 mm collimation and 1.0 mm reconstruction; catheter angiography (Siemens Artis zee floor); and endoscopic examination of the colon. Diagnostic laparoscopy was used where indicated.

Morphological evaluation was performed on 118 of 121 resection specimens and on 29 autopsy materials, stained with haematoxylin and eosin, van Gieson and Mallory trichrome, and periodic acid–Schiff. The depth of ischaemic injury was graded on an adapted Chiu–Park scale (grades 0–6). Immunohistochemistry for CD31 and CD34 was performed on 24 specimens and for Caspase-3 on 20 specimens to characterize microvascular density and apoptotic activity.

Statistical analysis. Data were processed in IBM SPSS Statistics 26.0, MedCalc 20.218, GraphPad Prism 10.1.0 and the R 4.3.2 environment. Normality was assessed by the Shapiro–Wilk test; between-group comparisons used the Student t-test or Mann–Whitney U-test for continuous variables and Pearson's chi-square test or Fisher's exact test for categorical variables, as appropriate. Diagnostic accuracy was evaluated by receiver-operating-characteristic (ROC) analysis with calculation of the area under the curve (AUC), 95% confidence intervals, and the Youden index for optimal cut-off selection; AUCs were compared by the DeLong method. Independent predictors were identified by multivariable logistic regression, with model calibration assessed by the Hosmer–Lemeshow test and internal validation performed by bootstrap resampling (1000 iterations). Survival was analysed by the Kaplan–Meier method with the log-rank test, and correlations were quantified by the Spearman rank correlation coefficient. A two-sided p-value < 0.05 was considered statistically significant throughout.

Results. *Incidence, diagnostic delay and stage at admission.* Between 2013 and 2024, 78,462 emergency surgical admissions were recorded at the two clinical bases, of which 165 (0.210%, or 2.10 per 1000 admissions) were attributable to acute mesenteric circulatory disorder. Implementation of the developed algorithm was associated with a marked reduction of the pre-hospital interval and a corresponding shift toward earlier clinical stages (Table 2).

The median interval from pain onset to admission decreased from 19.5 [12.0; 31.0] to 13.0 [7.0; 23.0] hours ($p < 0.001$): the proportion of patients presenting after more than 24 hours fell from 39.0% to 19.3%, while the proportion presenting within the first six hours rose from 8.5% to 18.1%. The clinical stage at admission changed accordingly — the share of patients arriving with infarction and diffuse peritonitis fell from 45.1% to 31.3%, and the share at the stage of ischaemia without necrosis rose from 13.4% to 22.9% ($p = 0.126$ for stage distribution). The reduction of the pre-hospital interval reflects a change in referral practice and in the vigilance of primary-care physicians and was treated as an independent confounder when interpreting between-group differences in outcome.

Aetiological structure and diagnostic errors. Aetiological analysis confirmed the predominance of arterial thrombosis — 85 observations (51.5%) — followed by embolism in 51 patients (30.9%), venous thrombosis in 15 (9.1%) and the non-occlusive form in 14 (8.5%); the between-group comparison of aetiological structure gave $p = 0.957$, confirming comparability (Table 3).

Discrepancy between the primary and final diagnosis was recorded in 58 patients (35.2%), disproportionately in the control group — 39 patients (47.6%) versus 19 (22.9%) in the main group ($\chi^2 = 11.02$, $p = 0.001$; odds ratio 3.06, 95% CI 1.57–5.96). The most frequent erroneous diagnosis was acute intestinal obstruction (19 observations), followed by acute pancreatitis (12) and acute cholecystitis or gastroenteritis (six each). The two-fold reduction of the error rate between periods was achieved mainly by shifting verification to the pre-operative stage: the proportion of patients whose diagnosis was confirmed by contrast-enhanced computed tomography rose from 34.1% to 75.9%, while the proportion confirmed only at intraoperative revision fell from 58.5% to 15.7%.

Embolic sources were identified in 49 of 51 patients with embolism, with atrial fibrillation predominating (44 observations, 86.3%); of these patients, 29 (65.9%) had not been prescribed anticoagulation, 7 (15.9%) had irregular intake, and therapeutic anticoagulation at admission was documented in only 4 (9.1%).

Table 2. Interval from symptom onset to hospitalization and clinical stage on admission

Indicator	Control, n = 82	Main, n = 83	Total
Interval from symptom onset			
Up to 6 hours	7 (8.5%)	15 (18.1%)	22
6–12 hours	16 (19.5%)	25 (30.1%)	41
12–24 hours	27 (32.9%)	27 (32.5%)	54
More than 24 hours	32 (39.0%)	16 (19.3%)	48
Clinical stage on admission			
Ischaemia without necrosis	11 (13.4%)	19 (22.9%)	30
Infarction without diffuse peritonitis	34 (41.5%)	38 (45.8%)	72
Infarction with diffuse peritonitis	37 (45.1%)	26 (31.3%)	63

Table 3. Form of mesenteric circulatory disorder and structure of primary diagnostic errors

Indicator	Control	Main	Total	Share, %
Arterial thrombosis	42	43	85	51.5
Embolism	26	25	51	30.9
Venous thrombosis	8	7	15	9.1
Non-occlusive form	6	8	14	8.5
Incorrect primary diagnosis	39	19	58	35.2
including intestinal obstruction	13	6	19	11.5
including acute pancreatitis	8	4	12	7.3
including acute cholecystitis	4	2	6	3.6

Among 85 patients with acute arterial thrombosis, stenosis of 50% or more at the origin of the superior mesenteric artery was found in 68 (80.0%), coeliac trunk stenosis in 32 (37.6%), and involvement of all three visceral vascular territories in 11 (12.9%), whereas a pre-existing diagnosis of chronic mesenteric ischaemia had been established in only 9 patients (10.6%).

Clinical presentation across disease stages. Abdominal pain was present in 161 patients (97.6%). The classic warning sign of pain disproportionate to physical findings was documented in 71 patients overall (43.0%), but its frequency fell steeply with disease progression — from 80.0% at the stage of reversible ischaemia to 12.7% at the stage of peritonitis — while signs of established necrosis (abdominal distension, absent peristalsis, muscular guarding, peritoneal signs, tachycardia, hypotension, fever) increased in the opposite direction. Sudden pain onset distinguished the arterial forms from venous thrombosis and the non-occlusive form: 77.9% versus 20.0% and 28.6%, respectively ($p < 0.001$). Because reliance on the signs of established necrosis inevitably means diagnosing the disease at a stage when the bowel can no longer be preserved, while the early warning sign is present chiefly during the reversible-ischaemia window, these findings underline the need for an objective, laboratory- and imaging-based trigger for early suspicion rather than reliance on the classical clinical triad.

Laboratory dynamics and the differentiated diagnostic value of biomarkers. Admission lactate values were similar between eventual survivors and non-survivors ($p = 0.414$); the divergence between the two trajectories became apparent from the sixth hour and persisted through day 7, a pattern mirrored by lactate dehydrogenase (admission values 486 [351; 721] vs 472 [338; 698] U/L; $p = 0.671$). Admission values were compared with 30-day outcome to weigh their prognostic contribution (Table 4).

ROC analysis showed that the relative informativeness of each marker changed with the diagnostic task at hand. For distinguishing AMCD from an acute abdomen of other aetiology, D-dimer ranked first, with an AUC of 0.846 at a threshold of 1540 ng/mL (sensitivity 81.2%, specificity 76.7%). For distinguishing transmural necrosis from reversible ischaemia, lactate ranked first (AUC 0.874; threshold 3.8 mmol/L; sensitivity 83.3%, specificity 80.7%), followed by pH (0.855) and lactate dehydrogenase (0.851), while D-dimer fell to fifth place (0.807). In practical terms, D-dimer is well suited to selecting patients for computed tomography because it flags the occlusive event itself, but it does not characterize the depth of wall injury, a task for which the metabolic markers are more informative.

Diagnostic accuracy of contrast-enhanced computed tomography. Contrast-enhanced CT was performed in 91 patients of the clinical cohort (28 in the control group, 63 in the main group) and in all 60 patients of the comparison group. A direct sign of occlusion — a filling defect within the arterial lumen — was identified in 72 patients (79.1%), while indirect signs of bowel-wall and mesenteric injury were less frequent and appeared later in the disease course. Diagnostic accuracy, calculated under a four-field design against the comparison group, is summarised in Table 5.

Table 4. Admission laboratory indices in relation to 30-day outcome

Indicator	Survivors, n = 83	Non-survivors, n = 82	p
Lactate, mmol/L	3.0 [2.0; 4.2]	6.3 [4.1; 8.7]	<0.001
D-dimer, ng/mL	1980 [1210; 3210]	4210 [2580; 6870]	<0.001
Procalcitonin, ng/mL	0.71 [0.22; 2.10]	3.84 [1.10; 9.20]	<0.001
C-reactive protein, mg/L	61 [32; 108]	139 [74; 218]	<0.001
Leukocytes, ×10⁹/L	14.1 ± 4.1	19.0 ± 5.1	<0.001
Lactate dehydrogenase, U/L	361 [277; 495]	648 [438; 892]	<0.001
pH	7.35 ± 0.06	7.25 ± 0.09	<0.001

Note: data are presented as Me [Q1; Q3] or mean ± SD.

Table 5. Diagnostic accuracy of contrast-enhanced computed tomography

Indicator	Value	Indicator	Value
True positive	86	Sensitivity	94.5%
False positive	7	Specificity	88.3%
True negative	53	Positive predictive value	92.5%
False negative	5	Negative predictive value	91.4%
Overall accuracy	92.1%	AUC (95% CI)	0.914 (0.865–0.963)

Three of the five false-negative results were attributable to inherent limitations of the method — distal occlusion of a small branch, and an early non-occlusive form without demonstrable wall change. Comparison of the CT-estimated length of affected bowel with the intraoperative findings in 74 patients showed complete agreement in 63 observations (85.1%), underestimation in 8 and overestimation in 3, confirming that full intraoperative revision of the bowel remains mandatory irrespective of the tomographic conclusion. Morphological examination of 118 specimens gave quantitative expression to the effect of time: the rank correlation between the depth of injury and the interval to admission was 0.61 ($p < 0.001$), grade 5–6 injury on the Chiu–Park scale did not occur among patients admitted within six hours and reached 43.2% among those admitted after more than 24 hours. Vascular density by CD34 immunostaining fell from 41.2 ± 8.1 to 21.6 ± 6.3 per mm² with increasing injury grade, while the proportion of Caspase-3-positive cells rose from 18.7 ± 6.2% to 46.9 ± 10.4%, confirming progressive, irreversible microcirculatory and cellular injury in parallel with delay.

Development of a prognostic scale for irreversible intestinal necrosis. Irreversible transmural or other non-viable necrosis, confirmed morphologically or at intraoperative revision, was defined as the modelled outcome and was recorded in 108 patients. Univariable analysis screened clinical, laboratory and radiological signs; seventeen reached a significance level below 0.10 and were entered into the multivariable model, with regression coefficients converted into integer points proportional to their magnitude (Table 6).

Five predictors retained independent prognostic significance: absence of bowel-wall contrast enhancement (adjusted odds ratio 5.37; $p = 0.003$), lactate above 3.8 mmol/L (4.44; $p = 0.001$), peritoneal signs (3.71; $p = 0.007$), pH below 7.31 (3.39; $p = 0.007$) and lactate dehydrogenase above 485 U/L (2.80; $p = 0.018$). The model constant was −3.12, and the resulting scale ranges from 0 to 10 points, combining one radiological sign of impaired wall perfusion, two metabolic indices, one marker of cellular breakdown and one clinical sign present in the early hours after admission. The optimal cut-off, determined by the maximum Youden index, was 6 points; the frequency of necrosis rose from 14.3% at a total score of 0–2 points to 96.2% at 8–10 points.

The diagnostic accuracy of the scale at its adopted cut-off was benchmarked against the single best-performing marker (Table 7).

Table 6. Multivariable logistic model and point score of predictors of irreversible intestinal necrosis

Predictor	β	Adjusted OR	95% CI	p	Points
Absence of bowel-wall contrast enhancement	1.68	5.37	1.77–16.29	0.003	3
Lactate > 3.8 mmol/L	1.49	4.44	1.91–10.34	0.001	2
Peritoneal signs	1.31	3.71	1.44–9.58	0.007	2
pH < 7.31	1.22	3.39	1.39–8.27	0.007	2
Lactate dehydrogenase > 485 U/L	1.03	2.80	1.19–6.59	0.018	1

Note: model constant –3.12; total score 0–10 points.

Table 7. Diagnostic accuracy of the scale at a cut-off of 6 points or higher

Indicator	Value	Indicator	Value
Sensitivity	86.1%	Positive likelihood ratio	5.45
Specificity	84.2%	Negative likelihood ratio	0.17
Positive predictive value	91.2%	AUC (95% CI)	0.910 (0.861–0.959)
Negative predictive value	76.2%	Bootstrap-corrected AUC	0.892
Overall accuracy	85.5%	Hosmer–Lemeshow p	0.632

The area under the curve for the scale (0.910) exceeded that of the best single marker, lactate (0.874), confirming the value of the multivariable approach. The clinical meaning of the scale is best expressed by its negative likelihood ratio of 0.17: a total score below six points reduces the odds of irreversible necrosis roughly six-fold, providing objective support for a bowel-preserving strategy with repeated viability assessment after revascularization. The Hosmer–Lemeshow p-value of 0.632 indicated good agreement between predicted and observed frequencies, the calibration slope was 0.94 with an intercept of −0.03 (no systematic bias), and bootstrap validation over 1000 resamples yielded a corrected AUC of 0.892, confirming that the model's performance was not an artefact of overfitting.

Differentiated surgical tactics and revascularization. Surgical intervention was performed in 154 patients — 78 in the control group and 76 in the main group; 11 patients with venous thrombosis or the non-occlusive form were managed conservatively. The interval from admission to the start of operation decreased from 6.8 [3.9; 11.6] to 3.7 [1.8; 6.4] hours ($p < 0.001$), and the proportion of operations begun within the first six hours rose from 43.6% to 68.4% ($p = 0.002$). Repeated intraoperative viability assessment — after warming of the bowel loops, novocaine blockade of the mesenteric root, and restoration of blood flow — was performed in 61 patients (17 in the control group, 44 in the main group). The appearance of the bowel improved in 40 patients, the planned volume of resection was reduced in 30, and resection was avoided altogether in 9; the median reduction of the anticipated resection length was 50 [30; 75] cm, with a maximum of 140 cm, meaning that forgoing repeated assessment would have led to removal of viable bowel of this length.

Revascularization was carried out in 65 patients — 22 in the control group and 43 in the main group — by open techniques in 55 and endovascular techniques in 18 patients, including 8 hybrid procedures that combined both approaches. The time from the start of the operation to restoration of blood flow decreased from 76 [61; 94] to 52 [39; 68] minutes ($p < 0.001$). Fogarty catheter thromboembolectomy was performed in 40 patients, with complete technical success in 85.0%; the overall rate of vascular complications was 27.3% in the control group versus 9.1% in the main group. Endovascular treatment was performed in 18 patients of the main group: main-trunk flow was restored in 16 (88.9%), complete angiographic success was achieved in 14 (77.8%), and laparotomy after the endovascular stage was still required in 11 (61.1%).

The effect of revascularization on the volume of bowel that had to be removed was assessed by comparing patients with and without restoration of blood flow (Table 8).

The mean resection length fell from 187 ± 79 to 123 ± 62 cm ($p < 0.001$), and the proportion of resections exceeding 200 cm fell from 48.6% to 19.1% ($p = 0.001$). The 64-cm mean difference corresponds to bowel preserved through restoration of blood flow and largely explains the between-group difference in the incidence of short bowel syndrome discussed below. The method of completing the operation was determined by haemodynamic stability and confidence in the viability of the proximal and distal bowel ends: the proportion of delayed anastomoses rose from 7.9% to 31.0%, and anastomotic leakage occurred in 10 of 59 patients (16.9%) after primary anastomosis versus 2 of 23 (8.7%) after a delayed anastomosis. A laparostomy was fashioned in 29 patients, and a programmed relaparotomy was performed in 30, during which additional resection was required in 13 — bowel that would have been left non-viable had the operation been completed in a single stage. Mortality was 34.5% with the staged tactic versus 57.6% with single-stage completion ($p = 0.030$).

Treatment outcomes. The structure of postoperative complications and treatment outcomes, compared between groups, served as the final measure of the algorithm's effect (Table 9).

Thirty-day mortality fell from 59.8% to 39.8% ($p = 0.010$), corresponding to a relative risk of 0.67, an absolute risk reduction of 20.0 percentage points and a number needed to treat of 5 to prevent one death. One-year survival rose from 30.5% to 51.8% ($p = 0.006$; log-rank $\chi^2 = 8.47$; hazard ratio for the main group, 0.62). Sepsis fell from 53.7% to 37.3% ($p = 0.042$), multiple organ failure from 57.3% to 41.0% ($p = 0.032$), and short bowel syndrome among survivors from 54.5% to 26.0% ($p = 0.008$); ICU stay shortened from 6 [3; 11] to 4 [2; 8] days ($p = 0.018$) and mechanical ventilation duration from 48 [18; 108] to 29 [12; 72] hours ($p = 0.031$). Multiple organ failure and septic shock together accounted for 52 of 82 deaths (63.4%).

Table 8. Extent of intestinal resection in relation to revascularization

Resection length	With revascularization, n = 47	Without revascularization, n = 74	p
Up to 100 cm	18 (38.3%)	14 (18.9%)	–
100–200 cm	20 (42.6%)	24 (32.4%)	–
More than 200 cm	9 (19.1%)	36 (48.6%)	0.001
Resection length, mean ± SD, cm	123 ± 62	187 ± 79	<0.001

Table 9. Comparison of endpoints between groups

Indicator	Control, n = 82	Main, n = 83	p
30-day mortality	49 (59.8%)	33 (39.8%)	0.010
One-year survival	25 (30.5%)	43 (51.8%)	0.006
Sepsis	44 (53.7%)	31 (37.3%)	0.042
Multiple organ failure	47 (57.3%)	34 (41.0%)	0.032
Short bowel syndrome among survivors	18/33 (54.5%)	13/50 (26.0%)	0.008
Resection length, mean ± SD, cm	186 ± 79	134 ± 68	<0.001
ICU stay, Me [Q1; Q3], days	6 [3; 11]	4 [2; 8]	0.018
Ventilation duration, Me [Q1; Q3], hours	48 [18; 108]	29 [12; 72]	0.031

Note: ICU – intensive care unit.

Table 10. Univariable association of predictors with 30-day mortality

Predictor	OR	95% CI	p
APACHE II ≥ 20 points	5.24	2.66–10.31	<0.001
Vasopressor support	5.15	2.56–10.36	<0.001
Lactate > 3.8 mmol/L	4.91	2.46–9.81	<0.001
Peritonitis on admission	4.87	2.47–9.61	<0.001
pH < 7.31	4.28	2.13–8.59	<0.001
Resection > 200 cm	3.72	1.82–7.60	<0.001
Hospitalization later than 12 hours	3.16	1.65–6.05	0.001
Age ≥ 70 years	2.12	1.14–3.94	0.017
Membership of the main group	0.44	0.24–0.82	0.010
Revascularization performed	0.34	0.18–0.66	0.001

Independent predictors of 30-day mortality. To isolate the independent contribution of the implemented algorithm, the association of candidate predictors with 30-day mortality was first examined by univariable analysis and then adjusted by multivariable regression (Table 10).

Ten characteristics were associated with mortality; the strongest were an APACHE II score of 20 points or more (odds ratio 5.24), the need for vasopressor support (5.15), lactate above 3.8 mmol/L (4.91) and peritonitis on admission (4.87), while membership of the main group (0.44) and revascularization (0.34) were protective. After adjustment for the time to hospitalization, age, disease stage, APACHE II severity, lactate level and the fact of revascularization, membership of the main group retained an independent protective effect on 30-day mortality, with an adjusted odds ratio of 0.49 (95% CI 0.25–0.96; p = 0.038), and revascularization retained an adjusted odds ratio of 0.48 (95% CI 0.23–0.99; p = 0.047). Age of 70 years or older lost its association with outcome once severity of condition was accounted for (p = 0.116), indicating that age alone should not be used as grounds for withholding active treatment. With 82 events and 11.7 events per predictor, the model built on the 165-patient cohort achieved an area under the curve of 0.856 (95% CI 0.798–0.914), a Hosmer–Lemeshow p-value of 0.679, and a bootstrap-corrected area of 0.837, confirming that the observed benefit of the algorithm is unlikely to be explained by the measured confounders alone.

Taken together, the diagnostic and surgical components of the algorithm form a single decision pathway: patients are first separated according to the presence of diffuse peritonitis; in its absence, revascularization is performed first, followed by repeated viability assessment; when diffuse peritonitis is present, laparotomy with resection is performed directly, and the point score of the prognostic scale determines whether the operation is completed in one stage or as a staged procedure with programmed relaparotomy. This sequence shortened the time to operation from 6.8 to 3.7 hours, reduced the mean resection volume from 186 ± 79 to 134 ± 68 cm and the incidence of short bowel syndrome among survivors from 54.5% to 26.0%, decreased 30-day mortality from 59.8% to 39.8%, and increased one-year survival from 30.5% to 51.8%.

This stepwise sequence operationalizes the statistical findings presented above into a bedside instrument that requires only tests already available in a standard multidisciplinary hospital — a coagulation panel, a blood-gas analyser, and contrast-enhanced computed tomography — without dependence on a dedicated intestinal-ischaemia unit.

Discussion. The present findings confirm, on a two-centre cohort of 165 patients, that outcome in acute mesenteric circulatory disorder is governed less by the technical performance of the operation than by the timeliness and objectivity of the decision that precedes it.

Table 11. Decision steps of the differentiated diagnostic-and-treatment algorithm

Step	Trigger	Action
1. Early suspicion	Pain disproportionate to physical findings; sudden onset in a patient with atrial fibrillation, atherosclerosis or a hypercoagulable state	Immediate lactate and D-dimer; do not await plain radiography, which carries no diagnostic value
2. Occlusion screening	D-dimer above 1540 ng/mL or persistent clinical suspicion	Urgent contrast-enhanced multiphase CT; ultrasonography with Doppler where CT is not immediately available
3. Depth-of-injury scoring	Confirmed or strongly suspected occlusion	Calculate the five-component prognostic scale (wall enhancement, lactate, peritoneal signs, pH, LDH)
4. Operative strategy – no diffuse peritonitis	Scale total below 6 points	Revascularization first (open, endovascular or hybrid), then repeated viability assessment before any resection
5. Operative strategy – diffuse peritonitis or scale ≥ 6	Established transmural necrosis or extensive contamination	Laparotomy with resection; staged completion with laparostomy and programmed relaparotomy at 24–48 hours
6. Anastomosis decision	Haemodynamic instability or uncertain bowel-end viability	Delay anastomosis to the relaparotomy stage rather than complete it primarily

Note: CT – computed tomography; LDH – lactate dehydrogenase.

The baseline mortality of 59.8% recorded in the control group sits close to the pooled short-term mortality of 59.6% reported by a systematic review and meta-analysis of 163 studies [8] and to the roughly half of patients who died during the index admission in the multicentre AMESI cohort [7], indicating that the historical arm of this study is representative of contemporary international practice rather than an unusually poor comparator.

Against this backdrop, the reduction to 39.8% achieved after implementation of the differentiated algorithm, and its persistence as an independent protective effect after adjustment for severity of condition, age, timing and revascularization (adjusted OR 0.49), places the observed benefit in the range that specialized intestinal-ischaemia units have reported through the introduction of structured multidisciplinary care rather than through a single new operative manoeuvre [2, 4, 5].

The diagnostic component of the study reinforces, and adds quantitative detail to, a point already made by systematic reviews of candidate biomarkers: no single laboratory index is sufficiently accurate in isolation. The present data go one step further by showing that the informativeness of a given marker depends on the diagnostic task being asked of it. D-dimer performed best at detecting the occlusive event itself (AUC 0.846), which is consistent with its role as a marker of thrombus formation and fibrinolysis, but it was a comparatively weak indicator of the depth of bowel-wall injury, for which lactate, pH and lactate dehydrogenase were superior. This separation of tasks — "is a vessel occluded" versus "how deep is the injury" — offers a practical rule for ordering and interpreting tests in a resource-limited multidisciplinary hospital that cannot rely on a battery of novel biomarkers: an elevated D-dimer should trigger urgent contrast-enhanced computed tomography, while the metabolic panel should guide the choice between a bowel-preserving and a resection-first strategy once occlusion is confirmed.

The five-component prognostic scale integrates exactly these two diagnostic tasks with a clinical sign and a marker of established necrosis, and its area under the curve (0.910, bootstrap-corrected 0.892) exceeded that of the best single marker (lactate, 0.874) by a margin that is unlikely to be due to chance alone, given the tight confidence interval and the favourable Hosmer–Lemeshow calibration. Mechanistically, the scale's performance is coherent with the morphological findings: the correlation between the Chiu–Park grade and the time to admission ($\rho = 0.61$), the fall in CD34-positive microvascular density, and the rise in Caspase-3-positive cells with increasing injury grade together describe a time-dependent progression from reversible ischaemia to apoptosis-mediated, and ultimately necrotic, transmural injury — a progression that the scale's components (perfusion, metabolic acidosis, lactate, cytolysis and peritoneal irritation) are individually positioned to detect at different points along the same pathway. The negative likelihood ratio of 0.17 at the chosen cut-off gives the scale direct clinical utility: a score below six points reduces the odds of irreversible necrosis roughly six-fold and provides an evidence-based trigger for repeated viability assessment after revascularization rather than immediate resection, addressing directly the two opposite errors —

unnecessary resection and retained non-viable bowel — that motivated this study.

The surgical results echo the international shift, formalized in the current World Society of Emergency Surgery and European Society for Vascular Surgery guidance, toward revascularization-first strategies wherever technically feasible [1, 3, 6]. In the present series, revascularization halved the mean resected bowel length (123 vs 187 cm) and, together with repeated intraoperative viability assessment, avoided resection altogether in 9 of 61 re-examined patients while allowing a reduction of the planned resection in a further 30 — findings that translate directly into a lower incidence of short bowel syndrome among survivors (26.0% vs 54.5%). The staged approach, with laparostomy and programmed relaparotomy reserved for patients with a high necrosis score or extensive peritoneal contamination, lowered mortality relative to single-stage completion (34.5% vs 57.6%), a pattern broadly consistent with reports favouring damage-control and open-abdomen strategies in severe peritonitis of mesenteric origin. Taken together, these results support a management philosophy, already advocated by dedicated intestinal-ischaemia centres, in which the operation is not a single irreversible act but a staged process built around repeated reassessment of bowel viability.

Strengths and limitations of the study. The principal strengths of this work are the two-centre design covering more than a decade of practice, the prospective collection of laboratory and imaging data at six standardized time points in the main group, the morphological and immunohistochemical corroboration of the clinical and biomarker findings, and the use of multivariable modelling with bootstrap internal validation rather than unadjusted before-after comparison. Several limitations should be considered when interpreting these results. First, the control arm was drawn from an earlier calendar period, so secular improvements in general critical care, imaging availability and pre-hospital referral — partially captured here as a reduction in the time to admission — cannot be fully separated from the specific effect of the algorithm, although the persistence of an independent protective effect after multivariable adjustment for these very factors mitigates this concern. Second, the study was conducted at two affiliated centres in a single region, and external validation of the prognostic scale in an independent cohort, ideally across several institutions with differing case-mix, is required before the scale can be recommended for routine use elsewhere. Third, although the multivariable models were adequately powered by conventional events-per-variable criteria and were internally validated by bootstrap resampling, the number of deaths (82) limits the number of covariates that could be examined simultaneously, and residual confounding by unmeasured factors cannot be excluded. Fourth, the economic and workflow implications of implementing round-the-clock contrast-enhanced computed tomography and a structured relaparotomy programme in resource-limited hospitals were not formally assessed and merit dedicated study. Notwithstanding these limitations, the consistency of the diagnostic findings with the international literature, the biological plausibility supplied by the morphological and immunohistochemical data, and the magnitude and independence of the mortality benefit together support the clinical relevance of the proposed scale and algorithm for multidisciplinary hospitals without a dedicated intestinal-ischaemia unit.

Conclusions. Acute mesenteric circulatory disorder accounts for 0.210% of emergency surgical admissions, with arterial thrombosis (51.5%) and embolism (30.9%) predominating; atrial fibrillation without adequate anticoagulant protection is the leading remediable aetiological factor (anticoagulants had not been prescribed or were taken irregularly in 36 of 44 patients with the cardioembolic form). The discrepancy between the primary and final diagnosis reaches 47.6% and stems from reliance on the signs of established necrosis rather than on the early warning sign of pain disproportionate to physical findings, which is present in 80.0% of patients at the stage of ischaemia but in only 12.7% at the stage of peritonitis.

Contrast-enhanced computed tomography detects mesenteric circulatory disorder with 94.5% sensitivity and 88.3% specificity (AUC 0.914). Laboratory indices serve two distinct diagnostic tasks: vascular occlusion is best reflected by D-dimer at a threshold of 1540 ng/mL (AUC 0.846), and the depth of ischaemic injury by lactate at a threshold of 3.8 mmol/L (AUC 0.874), with no single marker reaching an AUC of 0.90.

The developed five-component scale (absence of bowel-wall contrast enhancement, lactate above 3.8 mmol/L, peritoneal signs, pH below 7.31, lactate dehydrogenase above 485 U/L) predicts irreversible intestinal necrosis at a cut-off of six points with 86.1% sensitivity, 84.2% specificity and an AUC of 0.910, retained at 0.892 after internal validation, outperforming any single marker and consistent with the morphological correlation between injury depth on the Chiu–Park scale and the time to hospitalization ($\rho = 0.61$; $p < 0.001$).

A differentiated surgical tactic — revascularization in the absence of diffuse peritonitis, repeated viability assessment after restoration of blood flow, and a staged approach when the scale total is six points or higher — reduces 30-day mortality from 59.8% to 39.8% and increases one-year survival from 30.5% to 51.8%, retaining an independent effect on outcome after adjustment for the time to hospitalization, age, disease stage, severity of condition, lactate level and the fact of

revascularization (adjusted odds ratio 0.49; 95% CI 0.25–0.96; p = 0.038).

Practical recommendations. In any multidisciplinary hospital with round-the-clock access to a clinical laboratory and a computed tomography scanner, pain disproportionate to physical findings should prompt immediate measurement of lactate and D-dimer and, when either is abnormal, urgent contrast-enhanced computed tomography, rather than a period of clinical observation.

The five-component prognostic scale should be calculated for every patient with confirmed or strongly suspected vascular occlusion; a total below six points supports a bowel-preserving strategy with revascularization and repeated viability assessment, while a total of six points or higher supports primary resection.

In the absence of diffuse peritonitis, revascularization (open, endovascular or hybrid, according to local expertise and vessel anatomy) should precede the decision on resection; viability should be reassessed after warming of the bowel and restoration of blood flow before any resection margin is finalized.

When the prognostic scale total is six points or higher, or when peritoneal contamination is extensive, a staged approach with laparostomy and a programmed relaparotomy at 24–48 hours should be preferred over single-stage completion of the operation.

Patients with atrial fibrillation should be systematically evaluated for anticoagulant eligibility and adherence, since inadequate anticoagulation was the dominant remediable factor behind the cardioembolic form of the disease in this cohort.

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ОПТИМИЗАЦИЯ ДИАГНОСТИКИ И ХИРУРГИЧЕСКОЙ ТАКТИКИ ПРИ ОСТРОМ ТРОМБОЗЕ МЕЗЕНТЕРИАЛЬНЫХ АРТЕРИЙ: ПРОГНОСТИЧЕСКАЯ ШКАЛА ЖИЗНЕСПОСОБНОСТИ КИШЕЧНИКА И ДИФФЕРЕНЦИРОВАННЫЙ АЛГОРИТМ ЛЕЧЕНИЯ

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Резюме. *Цель: улучшить результаты диагностики и хирургического лечения больных с острым нарушением мезентериального кровообращения путем разработки прогностической шкалы жизнеспособности кишечника и дифференцированного лечебно-диагностического алгоритма. Материал и методы: в ретроспективно-проспективное сравнительное когортное исследование, проведенное в 2013–2024 гг. в двух клинических центрах Самарканда, включены 165 больных с острым нарушением мезентериального кровообращения: контрольная группа (n=82, традиционная тактика) и основная группа (n=83, новый алгоритм). Группу сравнения составили 60 пациентов с синдромом «острого живота» другой этиологии. Использованы лабораторные маркеры, компьютерная томография (КТ) с контрастным усилением, морфологическое и иммуногистохимическое исследование 118 резекционных препаратов, ROC-анализ, многофакторная логистическая регрессия с bootstrap-валидацией и анализ Kaplan–Meier. Результаты: окклюзию сосуда лучше всего отражал D-димер (AUC 0,846; порог 1540 нг/мл), глубину ишемического поражения – лактат (AUC 0,874; порог 3,8 ммоль/л); чувствительность КТ составила 94,5%, специфичность – 88,3%. Пятикомпонентная шкала (отсутствие контрастного усиления стенки кишки, лактат >3,8 ммоль/л, перитонеальные симптомы, pH <7,31, ЛДГ >485 Ед/л) при пороге ≥6 баллов прогнозировала необратимый некроз с чувствительностью 86,1% и специфичностью 84,2% (AUC 0,910; после bootstrap-коррекции 0,892). После внедрения алгоритма 30-дневная летальность снизилась с 59,8% до 39,8% (p=0,010), частота синдрома короткой кишки среди выживших – с 54,5% до 26,0% (p=0,008), однолетняя выживаемость возросла с 30,5% до 51,8% (p=0,006); скорректированное OR для основной группы составило 0,49 (95% ДИ 0,25–0,96). Заключение: прогностическая шкала и дифференцированный алгоритм (реваскуляризация в первую очередь при отсутствии разлитого перитонита, повторная оценка жизнеспособности кишки, этапная операция при сумме ≥6 баллов) улучшают результаты лечения острого нарушения мезентериального кровообращения в многопрофильном стационаре.*

Ключевые слова: *острая мезентериальная ишемия; тромбоз мезентериальных артерий; некроз кишечника; прогностическая шкала; реваскуляризация; резекция кишечника; синдром короткой кишки; D-димер; лактат; APACHE II.*