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Narrative Review

Persistent Shock After Source Control in Intra-Abdominal Sepsis: A Structured Critical Review and a Time-Triggered Reassessment Algorithm

João Rêgo Araújo[1]; Amália Cinthia Meneses Rêgo[2]; Irami Araújo Filho[3],[4].

[1]Medical Student, School of Medicine, Universidade Potiguar (UnP), Natal, Rio Grande do Norte, Brazil.

[2]Ph.D.; Full Professor (Biotechnology), Universidade Potiguar (UnP), Natal, Rio Grande do Norte, Brazil.

[3]M.D., Ph.D. in Health Sciences (Universidade Federal do Rio Grande do Norte, UFRN); Ph.D. in Experimental Surgery (Sorbonne University, Paris).

[4]Full Professor, Department of Surgery, Universidade Federal do Rio Grande do Norte (UFRN), Natal, Rio Grande do Norte, Brazil.

Correspondence

Prof. Dr. Irami Araújo Filho

Full Professor, Department of Surgery, Universidade Federal do Rio Grande do Norte (UFRN). Av. Nilo Peçanha, 620 — Petrópolis, Natal — RN, 59012-300, Brazil.

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Abstract

Intra-abdominal sepsis is a frequent and lethal cause of septic shock in critically ill adults, and early, adequate source control is a cornerstone of management. Yet after operative or percutaneous source control, many patients remain in shock in the intensive care unit, occupying a clinical gray zone: residual or recurrent shock may reflect expected postoperative inflammation, vasoplegia, hypovolemia and reperfusion, or it may signal undrained or progressive anatomical disease or microbiological failure. In this structured critical narrative review, reported following SANRA and not a systematic review, we propose that persistent shock after abdominal source control be interpreted as a time-dependent Bayesian signal. We provide operational definitions, distinguish the postoperative inflammatory–hemodynamic trajectory from ongoing disease, and map hemodynamic, metabolic, organ-dysfunction, abdominal, bedside, radiological, microbiological, surgical-context and cognitive triggers, organized into three phases—0–24 h (vigilant tolerance), 24–48 h (inflection point) and 48–72 h (presumed failure zone). Persistent shock is not, by itself, diagnostic of source-control failure, but it should raise the pre-test probability of ongoing anatomical or microbiological disease and trigger structured, interdisciplinary reassessment—re-imaging, antimicrobial review, drainage or reintervention, and formal exclusion of extra-abdominal causes—within 24–72 hours. The 48–72 h window is a safety threshold for mandatory reassessment, not a diagnostic threshold for failure. This hypothesis-generating algorithm is not a validated guideline and does not replace clinical judgment; it may reduce diagnostic inertia, and prospective, multicenter validation is required.

Highlights

- Persistent shock after abdominal source control is a time-dependent Bayesian signal.
- It is not diagnostic of failure but raises the pre-test probability of ongoing disease.
- A 24–48–72 h framework triggers structured, interdisciplinary reassessment.
- Anatomical and microbiological failure are reassessed on parallel tracks.
- The algorithm is hypothesis-generating and requires prospective, multicenter validation.

Key message

After abdominal source control, persistent or recurrent shock across the first 24–72 hours should shift the clinical posture from passive vasopressor titration to active, time-triggered reassessment for unresolved anatomical or microbiological disease.

Introduction

Intra-abdominal sepsis — most often arises from secondary peritonitis and complicated intra-abdominal infection — remains one of the most common surgical causes of septic shock and carries substantial ICU mortality [1,2]. Within the Sepsis-3 framework, septic shock identifies a subset with circulatory and cellular–metabolic abnormalities and a particularly high risk of death [3], and early, adequate source control is repeatedly identified as a central determinant of outcome [4,5]: the physical elimination or control of the infectious focus, whether by resection, repair, debridement, diversion, or percutaneous or endoscopic drainage, is what allows antimicrobial therapy and organ support to succeed.

Yet source control is not a binary, instantaneous event. After the index operation or drainage, a meaningful proportion of patients do not stabilize as expected; they remain vasopressor-dependent, hyperlactatemia or progressively organ-

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dysfunctional in the ICU. Series of patients managed with open abdomen or damage-control strategies for abdominal sepsis report persistently high mortality and identify several physiological and operative predictors of death [6], and broader cohorts of complicated intra-abdominal infection and generalized peritonitis confirm that a substantial subgroup deteriorates despite an apparently completed intervention [7,8]. This residual shock is the clinical problem that motivates the present review.

The difficulty is interpretive. Residual or recurrent shock after abdominal source control occupies a gray zone. On one hand, it may represent the expected postoperative state: systemic inflammatory response, vasoplegia, capillary leak, hypovolemia, reperfusion physiology, sedation effects and ongoing resuscitation, all of which are anticipated to improve over hours to days when the source has been controlled. On the other hand, the same picture may signal an undrained or residual collection, anastomotic dehiscence, ischemia or necrosis, missed or evolving perforation, ongoing peritonitis, or microbiological failure driven by resistant organisms, fungal infection or inadequate drug exposure [9,10]. Distinguishing these possibilities at the bedside, in real time, is genuinely difficult.

Current guidance addresses the bookends of this trajectory well. Recommendations on initial resuscitation, prompt empirical antimicrobial therapy and hemodynamic support are mature and widely disseminated through the Surviving Sepsis Campaign [11], and the principles of source control and of antibiotic choice and timing in intra-abdominal infection are similarly well described [4,9,10]. What is less well operationalized is the intermediate window — the first 24–72 hours after source control — during which clinicians must decide, often with incomplete information, whether the patient is following an expected recovery curve or whether the intervention has not worked. There is comparatively little structured guidance on when persistent shock should specifically trigger re-imaging, antimicrobial reassessment, drainage or reintervention, as opposed to continued titration of vasopressors and supportive care.

The central thesis of this review is therefore deliberately probabilistic. We argue that persistent shock after abdominal source control should be interpreted as a time-dependent Bayesian signal: it is not, in isolation, diagnostic of source-control failure, but it is sufficient to raise the pre-test probability of ongoing anatomical or microbiological disease and to mandate structured reassessment within a defined time window. We aim to (i) provide explicit operational definitions; (ii) separate expected postoperative physiology from ongoing disease; (iii) map the hemodynamic, metabolic, organ, abdominal, bedside, radiological, microbiological and cognitive triggers for reassessment; and (iv) propose a 24–48–72 h, time-triggered reassessment algorithm intended primarily to reduce diagnostic inertia.

To our knowledge, this specific operationalization of persistent shock after abdominal source control as a time-triggered reassessment signal has not previously been articulated as a structured critical narrative review; we advance this framing cautiously and present the resulting algorithm as hypothesis-generating rather than definitive.

Methods

This work is a structured critical narrative review, not a systematic review or meta-analysis. Its purpose is conceptual and clinical synthesis and the generation of a hypothesis-driven framework; it was designed and reported with reference to the SANRA (Scale for the Assessment of Narrative Review Articles) criteria [12]. No protocol was registered, PRISMA was not applied as the primary checklist, no meta-analysis was undertaken, and the review does not claim complete coverage of the literature.

Search strategy

We searched PubMed/MEDLINE, Embase (Ovid) and the Cochrane Library (CENTRAL and the Cochrane Database of Systematic Reviews) for literature published between January 2010 and 2026, supplemented by hand-searching of reference lists and relevant guideline repositories; earlier seminal references were retained where conceptually necessary (for example, consensus definitions). The core strategy combined the population (intra-abdominal sepsis, abdominal sepsis, secondary peritonitis, complicated intra-abdominal infection), the intervention/exposure (source control and its failure, reintervention, relaparotomy, reoperation, percutaneous drainage), and the physiological outcome of interest (shock, septic shock, vasopressor, lactate and lactate clearance, SOFA, ICU). Complementary searches addressed microbiological failure, open abdomen and damage-control surgery, intra-abdominal pressure and abdominal compartment syndrome, acute gastrointestinal injury, point-of-care ultrasound, and cognitive bias in critical-care decision-making. The full, reproducible search strings and their translation across databases are provided as Supplementary Material.

Study selection and mechanistic-relevance criteria

Because this was a structured critical narrative review rather than a systematic review, the search strategy was designed to identify clinically and mechanistically relevant bodies of evidence, not to provide exhaustive quantitative synthesis or meta-analysis. Beyond conventional clinical relevance, observational studies were prioritized when they satisfied predefined mechanistic-relevance criteria — that is, when they characterized a plausible causal pathway linking the index source-control event to subsequent shock (for example, residual or undrained infection, intra-abdominal hypertension, microbiological failure or drug-exposure failure), reported the temporal evolution of physiological variables, or illuminated a discrete failure mode amenable to reassessment. We included studies of adult critically ill or ICU patients with intra-abdominal sepsis undergoing source control; case reports were used only where they illustrate a specific failure mode or differential diagnosis. We did not draw primary inferences from pediatric-only, trauma-only (without sepsis) or elective uncomplicated abdominal-surgery literature. Throughout, we sought to keep direct evidence, indirect evidence, physiological plausibility and hypothesis clearly distinct, and to calibrate the strength of each statement to the strength of its support. To limit confirmation and selection bias, eligibility was assessed against the pre-specified mechanistic-relevance criteria by two reviewers independently, and we deliberately retained discordant and negative findings — for example, the contested results of extracorporeal blood-purification trials — rather than privileging confirmatory evidence.

Operational Definitions

Imprecise terminology is itself a source of diagnostic inertia. We therefore propose the following working definitions, intended to be pragmatic rather than authoritative.

Source control

Physical elimination or control of an infectious focus by surgery, percutaneous or endoscopic drainage, debridement, resection, repair, diversion or related intervention [4].

Adequate source control. An intervention that, on the best available intraoperative and post-procedural assessment, plausibly removes or controls the anatomical source of ongoing contamination or infection. Adequacy is a judgment made prospectively and is not guaranteed by the technical completion of an operation [4,5].

Source-control failure

Persistent or recurrent anatomical contamination or infection — for example a residual or undrained collection, anastomotic or stump dehiscence, ischemia or necrosis, uncontrolled perforation, or ongoing peritonitis — requiring re-imaging, repeat drainage, relaparotomy, reoperation or endoscopic intervention, or associated with death from uncontrolled abdominal sepsis [5,6].

Persistent shock

Continued need for vasopressors together with evidence of tissue hypoperfusion (for example persistent hyperlactatemia or absent lactate clearance) after initial source control and adequate resuscitation, consistent with the Sepsis-3 conception of septic shock [3].

Recurrent shock

Re-emergence or worsening of shock after an initial period of hemodynamic improvement — a trajectory that is often more informative than any single absolute value.

Anatomical failure

The subset of source-control failure attributable to an uncorrected or evolving structural problem amenable to drainage or operative correction [5,6].

Microbiological failure

Persistent infection driven by inadequate antimicrobial coverage, resistant organisms, fungal infection, inadequate drug exposure, delayed culture-guided adjustment, or failure to account for local resistance ecology [9,10,13].

Time-triggered reassessment

Structured reassessment activated by the temporal evolution (delta) of hemodynamics, lactate, organ dysfunction, abdominal findings, microbiology and imaging — rather than by any single threshold value at a single time point.

Time zero (t_0)

The completion of the index source-control procedure — operative or percutaneous — rather than the time of admission or diagnosis. In a patient initially managed non-operatively who subsequently requires rescue source control (for example after failed antibiotic-only therapy), the 0–24–48–72 h clock restarts at that rescue procedure.

Pathophysiology: Postoperative SIRS versus Ongoing Anatomical Disease

A degree of hemodynamic instability is expected after major source-control surgery for abdominal sepsis. Surgical trauma, the systemic inflammatory response, vasoplegia, capillary leak, reperfusion of previously hypoperfused tissue, residual hypovolemia, sedation and analgesia all contribute to a low-resistance, vasopressor-requiring state in the early postoperative period. When source control has been adequate, the expected trajectory is one of progressive improvement: lactate falls, vasopressor requirements decline, and organ dysfunction stabilizes and then resolves over the subsequent hours to days.

The interpretive principle we propose is dynamic, not static. It is the shape of the trajectory, rather than any single absolute value, that carries information. When the expected curve of improvement fails to materialize — when shock is sustained, plateaus, or recurs after transient improvement — the pre-test probability that an anatomical or microbiological problem remains unresolved rises. This reasoning is explicitly Bayesian and probabilistic, not deterministic: the same physiological picture has different implications at hour 6 and at hour 60. A particular trap deserves emphasis: pure, non-infectious distributive shock — for example severe pharmacological or sedation-related vasoplegia — may clinically mask ongoing anatomical failure, because a plausible alternative explanation for the vasopressor requirement can anchor the team and delay the search for an undrained or evolving abdominal source.

This framing is consistent with the broader literature on abdominal sepsis, in which delayed or failed source control, inappropriate initial antimicrobial therapy, and host factors such as immunosuppression are recurrently associated with worse outcomes [5,14,13], and in which biomarkers of inflammation and infection have been explored precisely because clinical signs alone are insufficiently discriminating early after intervention [15,16]. We summarize the central pathophysiological claim as follows:

A persistent or worsening inflammatory–hemodynamic trajectory after abdominal source control should not be interpreted solely as a pharmacological vasopressor problem; it should increase the pre-test probability of unresolved anatomical or microbiological disease. The progressive divergence between expected recovery and the principal failure trajectories is conceptually illustrated in Figure 1.

Hemodynamic and Metabolic Triggers

Triggers in this domain are best read as deltas and trends rather than as isolated thresholds. None is specific for abdominal source-control failure; collectively, however, an adverse trajectory should prompt structured reassessment.

Lactate kinetics and tissue perfusion

Failure of lactate to clear, a lactate rebound after an initial decrease, persistent metabolic acidosis and a worsening base deficit are all signals that tissue hypoperfusion is ongoing.

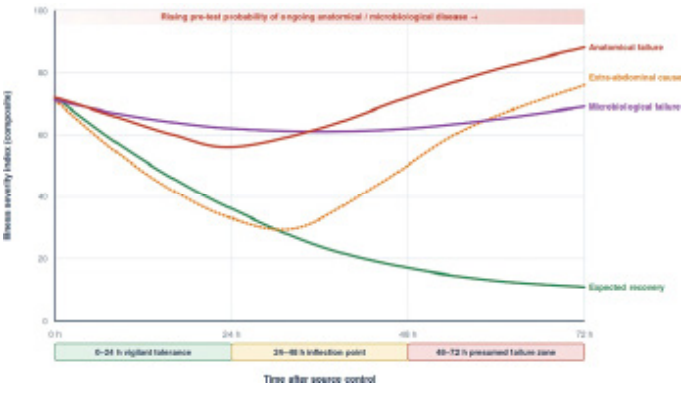

Figure 1. *The gray zone after abdominal source control. Source: Authors.*

Lactate is non-specific — it reflects global hypoperfusion, adrenergic load and impaired clearance as well as ongoing infection — but in the post-source-control context its failure to normalize should be read as a trigger to ask whether the source has truly been controlled, rather than as a problem to be managed by escalating vasopressors alone [17].

Vasopressor dynamics

Rising noradrenaline requirements beyond the first 24 hours, the need to add a second vasopressor, and recurrent shock after a period of weaning are clinically salient. The interpretiveb value again lies in the direction of travel: de-escalating vasopressor needs reassure, while escalation or recurrence should raise concern for unresolved disease and should not be normalized as expected postoperative behavior. As deliberately qualitative, non-validated illustrations of an adverse trajectory, a lactate that fails to fall by a clinically meaningful margin over the first hours (or rebounds after an initial decrease) and a norepinephrine requirement that escalates, or requires a second agent, rather than weaning are the kind of deltas that should prompt reassessment; the framework does not assign validated numerical thresholds, which remain to be derived.

Organ dysfunction

A SOFA score that is stable or worsening on day 2, new or progressive acute kidney injury and oliguria, worsening respiratory failure, thrombocytopenia and persistent acidemia each add weight to the probability that disease is ongoing. Severity scores and mortality-prediction tools (for example APACHE II, SAPS II and the Mannheim Peritonitis Index) are useful for risk stratification and prognostic communication in abdominal sepsis [18,17], but the methodological literature cautions that predictor and outcome definitions vary substantially across models, limiting their bedside transferability [19]. We therefore use trends in organ dysfunction as triggers for reassessment, not as confirmatory tests.

These markers are not specific for abdominal source-control failure. They are Bayesian triggers for structured reassessment.

Abdominal Triggers and Bedside Tools

Clinical abdominal signs

Increasing distension, ileus, enteral feeding intolerance, increasing pain where the abdomen is examinable, rising or changing drain output (particularly purulent, feculent or bilious effluent), recurrent fever and changes in the abdominal wall are all clinically meaningful. In the sedated, ventilated patient many of these signs are attenuated or absent, which is precisely why a structured, time-based prompt to reassess is valuable: the absence of localising signs does not exclude ongoing intra-abdominal disease [14,13].

Acute gastrointestinal injury

Gastrointestinal dysfunction can itself be conceptualised as an organ failure. The ESICM Working Group on Abdominal Problems defined acute gastrointestinal injury (AGI) and its grading, linking feeding intolerance, ileus and intra-abdominal hypertension with worse outcomes [20]; a deteriorating gastrointestinal picture after source control should therefore be incorporated into reassessment rather than treated as an isolated nutritional issue. Strategies such as chyme reinfusion and staged enteral nutrition in open-abdomen patients illustrate both the severity of gastrointestinal dysfunction in this population and the complexity of its management [21].

Intra-abdominal pressure

Intra-abdominal hypertension and abdominal compartment syndrome are reversible contributors to shock and organ dysfunction that are easy to miss without deliberate measurement, and the World Society of the Abdominal Compartment Syndrome consensus provides the definitions and measurement standards that make them actionable [22]. Raised intra-abdominal pressure impairs renal, respiratory and cardiovascular function and can arise from a wide variety of causes, including ileus, fluid overload, large collections and mass lesions, as well as after major abdominal and hernia surgery [23,24,25,26,27]. Intra-abdominal pressure also has pharmacological implications: it has been associated with altered pharmacokinetics of renally cleared antimicrobials such as aminoglycosides, which is relevant when microbiological failure is in the differential [28]. Bladder-pressure monitoring is inexpensive relative to the consequences of missed compartment syndrome [24], and emerging non-invasive detection methods are under investigation [29]. Tension pneumoperitoneum is a rare but immediately reversible cause of hemodynamic collapse that bedside decompression can correct [30]. Sedation strategy may itself modulate intra-abdominal pressure and the inflammatory response in abdominal sepsis [31]. For the closed abdomen, we anchor these triggers to the WSACS consensus: a sustained intra-abdominal pressure ≥12 mmHg defines intra-abdominal hypertension, and a sustained intra-abdominal pressure >20 mmHg accompanied by new organ dysfunction defines abdominal compartment syndrome [22]. The latter is a reversible cause that activates the "deteriorating at any point" fast-track — prompting immediate decompression — regardless of the 24–48–72 h clock.

Point-of-care ultrasound

Point-of-care ultrasound (POCUS) is a bedside triage tool, not a replacement for cross-sectional imaging. Its principal value in this setting is to reduce diagnostic inertia and to inform whether a critically ill patient should be escalated to computed tomography (CT), drainage or operative reassessment — particularly when transport to CT carries risk. While robust outcome data for POCUS altering source-control failure mortality remains limited, its diagnostic yield for detecting unexpected intra-abdominal fluid or pleural collections in the ICU makes it an essential non-invasive triage tool at the 48-hour inflection point [32,33,34]. Additional point-of-care ultrasound evidence specific to source-control failure should be incorporated as it accrues.

POCUS and intra-abdominal pressure measurement should be positioned as bedside triage tools that may support escalation to CT, drainage or operative reassessment, not as substitutes for definitive cross-sectional imaging or surgical judgment.

Open versus Closed Abdomen

The reassessment threshold is not the same for a patient whose fascia was closed primarily and one managed with an open abdomen or planned relaparotomy. These contexts differ in physiology, in the meaning of bedside measurements, and in the opportunity for re-inspection.

Open-abdomen and damage-control strategies — typically using temporary abdominal closure and negative-pressure wound therapy — are widely used for severe abdominal sepsis and are the subject of active investigation, including the COOL

trial program [35] and a series of comparative and registry studies of vacuum-assisted closure versus primary closure or on-demand relaparotomy [36-42]. In the open abdomen, a planned second look provides a scheduled opportunity to reassess the source directly, which partly substitutes for imaging-based decision-making; intra-abdominal pressure readings must be interpreted differently after decompression; and the physiological cost is higher, with greater fluid and protein losses, sustained inflammation, and risks including enteroatmospheric fistula and delayed closure [43-50]. Predictors of mortality specific to the open abdomen for abdominal sepsis have been described and can inform expectations during reassessment [6,51-54]. Importantly, serial CT in patients with an open abdomen has a low diagnostic yield when performed routinely rather than being prompted by an acute clinical or metabolic change; in this context the planned re-look guided by the trajectory, is usually more informative than calendar-driven imaging.

In the closed abdomen, the absence of a planned re-look places greater weight on cross-sectional imaging and on a low threshold for reintervention when the trajectory is adverse, because there is no scheduled mechanism to detect an evolving problem. Comparative studies of open versus primary closure in severe abdominal sepsis report differing complication and survival profiles and underline that neither strategy is uniformly superior [40,41]. The practical implication is that the same time-triggered prompts should be applied, but the response to a positive trigger — direct re-inspection versus imaging-led escalation — must be tailored to the abdominal-closure context. The algorithm should not be applied mechanically and identically to both. The principal differences in reassessment thresholds, diagnostic strategies, and responses between open- and closed-abdomen management are summarized in Table 1.

Feature	Open abdomen / damage control	Closed abdomen
Opportunity to re-inspect source	Scheduled second-look provides direct reassessment	No scheduled re-look; greater reliance on imaging
Interpretation of intra-abdominal pressure	Decompressed; intra-abdominal pressure less informative for compartment syndrome	Intra-abdominal pressure a key reversible trigger; measure deliberately
Physiological cost	Higher fluid/protein loss; sustained inflammation; fistula risk	Lower baseline loss; closure-related complications differ
Value of serial CT	Low yield if routine; reserve for acute clinical/metabolic change	Higher relative value when trajectory is adverse
Default reassessment response	Use planned re-look ± targeted imaging; titrate closure timing	Low threshold for CT and reintervention on adverse trajectory

Both contexts use the same time-triggered prompts (Section 8); the response to a positive trigger differs and should be tailored. The algorithm should not be applied identically to both.

The 24–48–72 h Time-Triggered Reassessment Framework

This is the central proposal of the review. We describe three temporal phases. The boundaries are deliberately approximate and are intended as prompts, not as validated cut-offs.

0–24 h — Phase of vigilant tolerance

In the first 24 hours, residual shock may legitimately reflect postoperative vasoplegia, systemic inflammation, hypovolemia, reperfusion, sedation or ongoing resuscitation. The goal is aggressive physiological optimization and close trend monitoring rather than immediate reintervention. Clinicians should track lactate, vasopressor dose, SOFA, urine output, temperature, drain output, the abdominal examination (where possible), intra-abdominal pressure and the adequacy of empirical antimicrobial therapy against the likely and cultured organisms [11,9].

24–48 h — Inflection point

By 24–48 hours, the absence of improvement, or any worsening, should trigger a formal interdisciplinary reassessment — ideally ICU, surgery, radiology and infectious diseases together where available. This reassessment should explicitly evaluate the lactate trajectory, vasopressor dynamics, SOFA, abdominal signs, drains, POCUS and intra-abdominal pressure, and the microbiological data and antimicrobial spectrum, and should reach an explicit decision about CT, drainage, relaparotomy, endoscopy or antimicrobial escalation. The act of convening this reassessment is itself the intervention the framework is designed to guarantee [55]. This reassessment does not require physical co-location: out of hours, it can be operationalized as a brief, time-stamped virtual or telephone huddle — the surgeon and intensivist on call as the minimum core, with remote radiology and infectious-diseases input and a low threshold to escalate to the attending — provided that the decision and its rationale are explicitly documented.

48–72 h — Presumed failure zone

Beyond 48–72 hours, persistent or recurrent shock should be treated as possible source-control failure until that has been formally excluded. This generally requires cross-sectional imaging, surgical reassessment and microbiological review, or explicit documentation of an alternative cause. Indefinite escalation of vasopressors without anatomical and microbiological reassessment should be actively avoided, because it substitutes pharmacological management for diagnosis.

The 48–72 h window should not be framed as a diagnostic threshold for source-control failure, but as a safety threshold for mandatory reassessment.

Differential Diagnoses and Microbiological Safeguards

Non-abdominal causes of persistent shock

Persistent shock after abdominal source control is not always abdominal. Pneumonia and ventilator-associated pneumonia, pulmonary embolism, myocardial infarction and stress cardiomyopathy, ongoing hemorrhage, unrecognized hypovolemia, adrenal insufficiency, sedation-related vasoplegia, catheter-related bloodstream infection, transfusion reactions and drug effects can all sustain shock and must be considered

in parallel rather than after abdominal causes are exhausted. Prolonged mechanical ventilation after abdominal surgery is itself associated with identifiable risk factors and complications and may both reflect and contribute to a complicated course [56]. The discipline the framework imposes is to make this differential explicit at each reassessment, so that an abdominal cause is neither anchored upon nor prematurely dismissed.

Microbiological failure

Microbiological failure is a distinct and treatable mechanism that can masquerade as, or coexist with, anatomical failure. It includes infection by multidrug-resistant Gram-negative organisms (including ESBL- and carbapenemase-producing Enterobacterales such as KPC), Pseudomonas, enterococci and anaerobes, and Candida; inadequate or inappropriately timed empirical therapy; delayed de-escalation or escalation; inadequate drug exposure at the site of infection; and failure to account for local resistance ecology and prior colonization [9,10,13]. Inappropriate initial antimicrobial therapy has been linked to worse prognosis in severe secondary peritonitis, with implications for both broad-spectrum coverage and carbapenem-sparing strategies [13]; immunocompromised patients show distinct microbial etiology and outcomes [14]; and fungal and unusual organisms (for example emphysematous infection or fungal gastric perforation) are reminders that the empirical regimen may be the problem [57,58]. The interaction between drug exposure and abdominal physiology — including the effect of intra-abdominal pressure on antimicrobial pharmacokinetics — further complicates dosing, and early review by infectious diseases or clinical microbiology is advisable when shock persists [28].

It is also important to recognize the limits of adjunctive therapies. Extracorporeal blood-purification and endotoxin-removal techniques (for example polymyxin B hemoperfusion, LPS-selective adsorbents and broad-spectrum hemoadsorption) have been studied in abdominal sepsis and septic shock with heterogeneous and still-contested results [59-64]; notably, endotoxin-removal therapy has been examined specifically in patients after successful infection-source control [59]. These modalities do not substitute for source control or for correct antimicrobial therapy, and their use should not delay anatomical and microbiological reassessment. A specific caveat applies while extracorporeal therapies are running: hemadsorption, endotoxin removal and concurrent renal replacement can lower vasopressor requirements, blunt fever, clear lactate by dialysis and remove circulating cytokines, thereby improving the very hemodynamic and metabolic markers on which the algorithm relies. An apparently favorable trajectory during such therapy should therefore be interpreted with caution, with greater weight given to anatomical and imaging reassessment so that unresolved source-control failure is not masked.

Persistent shock after abdominal source control should trigger not only anatomical reassessment, but also early antimicrobial reassessment based on cultures, source, prior colonization, previous antibiotic exposure, immune status, local resistance patterns, and risk factors for fungal infection.

The principal anatomical, microbiological, cardiovascular, respiratory, hemorrhagic, endocrine, iatrogenic, and device-related differential diagnoses are summarized in Table 2. These possibilities should be evaluated in parallel rather than sequentially.

***Table 2**. Differential diagnosis of persistent shock after abdominal source control*

Category	Examples	Clues	Immediate evaluation	Interaction with abdominal reassessment
Anatomical failure	Residual/undrained collection; dehiscence; ischemia/necrosis; missed perforation	Abdominal signs; rising drain output; adverse trajectory	CT; surgical review; drainage	Primary target of the algorithm
Microbiological failure	MDR/ESBL/KPC; Candida; inadequate/ inappropriate empirical therapy	Persistent fever/markers despite drainage; prior colonization	Cultures; ID review; spectrum/dose check	Parallel track; may coexist with anatomical failure
Cardiovascular	Myocardial infarction; stress cardiomyopathy	ECG/troponin; POCUS LV dysfunction	ECG, troponin, echocardiography	Extra-abdominal; do not anchor on abdomen
Respiratory	Pneumonia/VAP; pulmonary embolism	Hypoxaemia; imaging; risk factors	CXR/CT; D-dimer/CTPA as indicated	Common in prolonged ICU course
Hemorrhagic	Ongoing surgical or non-surgical bleeding	Falling hemoglobin; transfusion need	Hemoglobin trend; imaging; surgical review	May mimic septic shock; needs source control too
Endocrine / metabolic	Adrenal insufficiency	Vasopressor-refractory shock	Cortisol; empirical steroid trial	Extra-abdominal contributor
Iatrogenic / drug-related	Sedation-related vasoplegia; drug effects	Temporal link to agent/ dose	Review sedation and drug chart	Modifiable; reassess before escalating
Device / catheter-related	Catheter-related bloodstream infection	New fever; line in situ	Cultures; line review/ removal	Microbiological track overlap
Goals-of-care context	Futility after repeated failure	Cumulative organ failure; poor reserve	Honest prognostic appraisal; family discussion	Legitimate output when reassessment is exhausted

Abbreviations: CTPA, CT pulmonary angiography; CXR, chest radiograph; ECG, electrocardiogram; LV, left ventricular; VAP, AKI, acute kidney injury; CT, computed tomography; ESBL, extended-spectrum β-lactamase; ICU, intensive care unit; ID, infectious diseases; KPC, Klebsiella pneumoniae carbapenemase; MDR, multidrug-resistant; POCUS, point-of-care ultrasound; SOFA, Sequential Organ Failure Assessment.

Cognitive Bias, Diagnostic Inertia and Interdisciplinary Decision-Making

Much of the difficulty in this clinical space is cognitive rather than technical. After a technically successful operation, there is a natural optimism bias and therapeutic momentum: the team that performed adequate source control is psychologically invested in the expectation that it worked. Anchoring on the index diagnosis, diagnostic inertia, and the asymmetry between surgical reassurance ("the abdomen was clean") and intensive-care concern ("the patient is not improving") can delay reassessment at precisely the point where time matters most. The critical-care decision-making literature documents that cognitive biases and environmental, patient and personal factors systematically shape decisions in this setting [65,66], and that the interplay of technical devices, data and interprofessional dynamics influences ICU decision-making in ways that are not purely clinical [55].

Reframing persistent shock as a scheduled, shared checkpoint rather than as a contest between specialties may mitigate these biases. A structured, time-triggered reassessment functions as a cognitive forcing strategy: it converts an implicit, deferrable judgment into an explicit, calendared, multidisciplinary decision — a brief interdisciplinary huddle — with a documented rationale. The algorithm is therefore as much a communication and governance tool as a clinical one.

A time-triggered reassessment algorithm may function as a cognitive forcing strategy, reducing diagnostic inertia and reframing persistent shock as a shared interdisciplinary problem rather than a disagreement between surgical optimism and intensive-care concern.

The multidomain red flags that should activate structured reassessment—including hemodynamic, metabolic, organ-dysfunction, abdominal, bedside, radiological, microbiological, surgical-context, and cognitive-operational triggers—are summarized in Table 3.

Proposed Clinical Algorithm

The proposed algorithm (Figure 2) takes as its entry point an adult ICU patient who has undergone abdominal source control for intra-abdominal sepsis and who remains in, or re-enters, shock. It is organized around the three temporal zones and runs an antimicrobial-review track in parallel with the anatomical track.

Entry

Adult ICU patient after abdominal source control for intra-abdominal sepsis with persistent or recurrent shock.

Serial assessment (all phases)

Lactate and clearance, vasopressor dose and number, SOFA, urine output and acid–base status, abdominal examination and

Table 3. *Red flags for time-triggered reassessment after abdominal source control*

Domain	Trigger	Interpretation	Suggested action	Nature of supporting evidence
Hemodynamic	Rising vasopressor need >24 h; second vasopressor; recurrent shock	Adverse trajectory; increased probability of ongoing disease	Reassess source and antimicrobials; avoid blind escalation	Indirect / inferential
Metabolic	Absent lactate clearance; lactate rebound; worsening base deficit	Ongoing tissue hypoperfusion	Trigger reassessment; consider imaging	Indirect / mechanistic
Organ dysfunction	SOFA stable/worsening on day 2; new AKI; thrombocytopenia	Failure of the expected recovery curve	Interdisciplinary review	Indirect (prognostic scores)
Abdominal	Distension; feeding intolerance; ileus; purulent/feculent drain output; recurrent fever	Possible residual/recurrent intra-abdominal source	Imaging ± drainage ± reoperation	Direct + mechanistic
Bedside tools	POCUS free fluid/collection; raised intra-abdominal pressure	Triage signal toward CT/drainage/operation	Escalate to definitive imaging or operation	Consensus guideline + indirect
Radiological	New/enlarging collection; dehiscence; ischemia on CT	Anatomical source-control failure	Drainage / relaparotomy	Direct (when imaged)
Microbiological	MDR/ESBL/KPC, Candida; inadequate empirical cover; rising markers	Microbiological failure	Culture-guided escalation; ID input	Direct observational
Surgical context	Open abdomen vs closed; planned re-look due	Modifies threshold and response	Apply Table 3 modifier	Comparative observational
Cognitive / operational	Diagnostic inertia; surgical optimism vs ICU concern; no reassessment convened	Risk of delayed recognition	Convene scheduled interdisciplinary huddle	Indirect (decision science)

Abbreviations: AKI, acute kidney injury; CT, computed tomography; ESBL, extended-spectrum β-lactamase; ICU, intensive care unit; ID, infectious diseases; KPC, Klebsiella pneumoniae carbapenemase; MDR, multidrug-resistant; POCUS, point-of-care ultrasound; SOFA, Sequential Organ Failure Assessment. "Nature of supporting evidence" reflects the authors' qualitative appraisal in a narrative review and is not a formal evidence-grading (e.g. GRADE) assessment. Numerical thresholds are intentionally omitted and require derivation and validation.

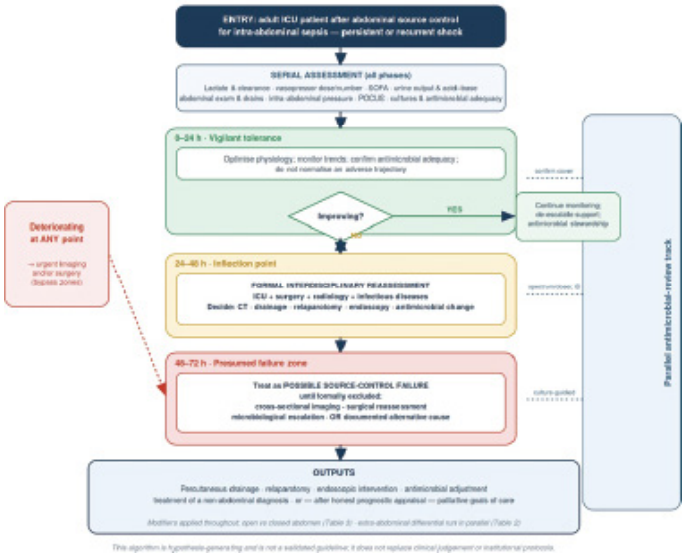

Figure 2. *Time-triggered reassessment algorithm.*
Source: Atuhors

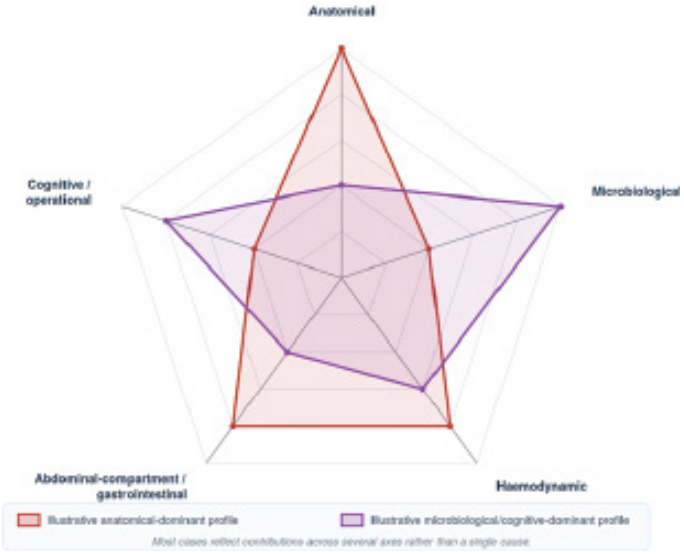

Figure 3. *Multidimensional model of persistent shock after source control.*
Source: Atuhors

drain output, intra-abdominal pressure, POCUS, cultures and antimicrobial adequacy.

0–24 h (vigilant tolerance)

Optimize physiology; monitor trends; confirm empirical antimicrobial adequacy; do not normalize an adverse trajectory.

Improving

continue monitoring, de-escalate support, pursue antimicrobial stewardship.

Not improving by 24–48 h

convene formal interdisciplinary reassessment (ICU + surgery + radiology + infectious diseases); decide explicitly on CT, drainage, relaparotomy, endoscopy or antimicrobial change.

Deteriorating at any point

urgent imaging and/or surgical review without waiting for the next time window.

Persistent/recurrent shock at 48–72 h

treat as possible source-control failure until formally excluded: cross-sectional imaging, surgical reassessment, microbiological escalation, or documented alternative (extra-abdominal) cause.

Modifiers

Apply an open-abdomen / damage-control versus closed-abdomen modifier (Table 3); run the extra-abdominal differential (Table 2) in parallel.

Outputs

Percutaneous drainage, relaparotomy, endoscopic intervention, antimicrobial adjustment, treatment of a non-abdominal diagnosis, or — where appropriate and after honest prognostic appraisal — a shift toward palliative goals of care.

This algorithm is hypothesis-generating and intended to reduce diagnostic inertia. It is not a validated guideline and should not replace clinical judgment, institutional protocols, local resources, or individualized surgical decision-making.

Although the algorithm is presented as a sequential, time-triggered pathway for operational clarity, persistent shock after source control is usually multidimensional. Anatomical, microbiological, hemodynamic, abdominal-compartment/gastrointestinal, and cognitive/operational factors may coexist and interact in the same patient. Structured reassessment should therefore interrogate all five domains rather than seek a single explanatory cause (Figure 3).

Limitations

Several limitations must temper the interpretation of this review. It is a narrative, not a systematic, review, and is therefore susceptible to selection bias in the synthesized evidence; no protocol was registered and no quantitative synthesis was performed. Intra-abdominal sepsis is markedly heterogeneous in source, organism, host and operative strategy, which limits generalizable thresholds. The physiological markers we use as triggers — lactate, vasopressor requirement and SOFA — have low specificity for abdominal source-control failure and can be driven by extra-abdominal disease. The 24–48–72 h boundaries are not validated cut-offs but pragmatic prompts.

A direct consequence of relying on triggers of low specificity is the risk of negative, or "white", relaparotomies — reoperations that reveal no correctable source. We regard a modest rate of negative findings as an acceptable price for reducing diagnostic inertia and the far more dangerous error of delayed recognition of a genuine, treatable source; nonetheless, this trade-off must be made explicit, audited locally, and minimized by combining triggers rather than acting on any single one, and by preferring image-guided or minimally invasive reassessment where it is safe and available. We deliberately avoid proposing a fixed acceptable rate of negative reoperation, as no validated threshold exists; the non-therapeutic-relaparotomy rate is better treated as a locally audited quality metric and as a pre-specified outcome for prospective validation, benchmarked against the on-demand versus planned-relaparotomy literature rather than against an arbitrary numerical ceiling. Applied too aggressively, the framework risks over-imaging and

unnecessary reintervention, with their attendant harm.

The thresholds and responses also differ between open and closed abdomens in ways that we have described qualitatively rather than quantitatively. Finally, the scope of this review is deliberately confined to patients who have already undergone operative or percutaneous source control. We therefore intentionally excluded conditions whose initial management is primarily conservative — for example uncomplicated appendicitis managed with antibiotics alone, as studied in the APPAC and CODA trials [67,68]. This focus sharpens the review's applicability to the post-operative and post-drainage ICU population, but it correspondingly limits external validity outside that setting; the framework is not intended to govern decisions in non-operative or watchful waiting pathways.

Future Directions

The framework is explicitly hypothesis-generating and invites prospective evaluation. Priorities include a prospective multicenter cohort or registry of patients with persistent shock after abdominal source control, capturing the temporal evolution of the proposed triggers, and the derivation and external validation of a time-triggered reassessment score. We anticipate that emerging inflammatory and bacterial biomarkers may add predictive value to purely physiological triggers, potentially helping to discriminate ongoing infection from resolving postoperative inflammation earlier than lactate or SOFA alone [15,16]; combining such biomarkers with trajectory data is a tractable next step. A natural translation is a digital reassessment score integrated into the electronic health record (EHR), which could ingest serial vital-sign, laboratory, drain and microbiology data, flag an adverse trajectory at the 24–48 h inflection point, and prompt — rather than replace — a documented interdisciplinary decision; such a tool would operationalize the cognitive-forcing function described above while remaining auditable. Studies should pre-specify analysis of the antimicrobial-reassessment track alongside the anatomical track, and should explicitly include open-abdomen and damage-control subgroups, which the literature suggests behaving differently [6,35,36]. Outcomes should be patient-centered and pragmatic — mortality, ICU and ventilator-free days, reintervention rate, time to recognition of source-control failure, and harms from over-imaging or unnecessary reoperation — and should be aligned with emerging core outcome sets for damage-control laparotomy and abdominal sepsis to enable comparison across studies [43,44]. Cost-effectiveness, including the relative costs of monitoring (for example intra-abdominal pressure measurement) versus missed failure, also warrants formal study [24].

Conclusion

Persistent shock after abdominal source control is not proof of failed source control. But it is not noise either. Interpreted as a time-dependent Bayesian signal, it should increase the pre-test probability of ongoing anatomical or microbiological disease and should drive structured, interdisciplinary reassessment. Time is the organizing principle: by 24–48 hours, the absence of improvement should trigger a formal multidisciplinary reassessment; by 48–72 hours, persistent or recurrent shock should be treated as possible source-control failure until it has been formally excluded. The algorithm we propose is a safety framework and a cognitive forcing strategy, not a validated rule, and it requires prospective, multicenter validation before it can be recommended for routine use.

Persistent shock after abdominal source control should shift the clinical posture from passive vasopressor titration to active, time-triggered reassessment for unresolved anatomical or microbiological disease.

Declarations

Ethics approval and consent to participate

Not applicable. This is a narrative review of previously published literature; it did not involve new studies on human participants, identifiable human data or animals, and no ethics approval or informed consent was required.

Consent for publication

Not applicable.

Availability of data and materials

Not applicable. No new datasets were generated or analyzed. All sources are cited in the reference list, and the complete search strategy is provided as Supplementary Material.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

I.A.F. conceived the work and the central thesis. J.R.A. and I.A.F. designed and conducted the structured literature search. J.R.A., A.C.M.R. and I.A.F. drafted the manuscript. A.C.M.R. and I.A.F. critically revised it for important intellectual content and supervised the work. All authors read and approved the final manuscript and agree to be accountable for all aspects of the work, in accordance with the ICMJE authorship criteria.

Use of artificial intelligence

Artificial intelligence-assisted tools were used to support language refinement, structural organization and editorial review, under full author supervision. The authors verified all scientific content, references, interpretations and conclusions, and remain fully responsible for the manuscript. The tool was not used to generate or fabricate data, results or citations, and is not listed as an author, consistent with ICMJE and COPE recommendations. [Specify the tool, version and date of use as required by the target journal's policy.]

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