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Non-occlusive mesenteric ischemia in critically ill patients: does bedside laparoscopy offer any real benefit?

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Abstract

Non-occlusive mesenteric ischemia in critically ill patients still has a poor prognosis. Despite several established risk factors, the interaction between clinical conditions and perfusion mismatch often leads to progressive organ failure. Workup is challenging because of the absence of typical abdominal signs and symptoms due to sedation, poor reactivity, need for ventilation, and confounding comorbidities. Moreover, imaging has poor specificity with findings often inconclusive. A bedside exploratory laparoscopy, as a complementary strategy, would allow for early and prompt diagnosis. Limits of a minimally invasive surgical rationale lie upon the effects of pneumoperitoneum induction, surgical stress, logistical resources, expertise, and costs.

Keywords: Acute abdomen, Intensive care unit, Laparoscopy, Non-occlusive mesenteric ischemia

Mesenteric ischemia in critically ill patients in intensive care unit (ICU) embraces subtle and rapidly progressive clinical and biochemical scenarios usually in a context of complex comorbidities, making diagnosis challenging. Notwithstanding undeniable improvements both in imaging and identification of pathognomonic and diagnostic criteria, splanchnic ischemias still have a poor prognosis in up to 69% of patients.[1] In the context of hemodynamic instability, of impaired visceral perfusion, and of prolonged hospitalization in ICU, the risks of Non-occlusive mesenteric ischemia (NOMI) increase, leading to a vicious circle of facing hypovolemia, hypotension, or organ failure.

NOMI occurs in presence of preserved vessels' patency and appears to be due to a mismatch between oxygen delivery and metabolic demands,[2] paradoxically presenting as a diagnosis of exclusion, where biochemical and radiological criteria often appear inexhaustible and thus resulting in a lack of prompt lifesaving diagnosis to contrast a dismal progression to sepsis and multiorgan failure.[3] Several risk factors could predispose to NOMI, such as age older than 50 years, low cardiac output states (congestive heart failure, ischemic heart disease, arrhythmias), end-stage renal failure and hemodialysis, hepatic failure, aortic insufficiency, multiple organ failure, sepsis, shock, intra-abdominal hypertension, drugs reducing splanchnic perfusion, chemotherapy, abuse substances, prolonged hospitalization in ICU, and severe acute respiratory syndrome coronavirus 2 infection.[4–6] However, the abovementioned factors appear to be poorly predictive, inconclusive, and often misleading. Subclinical and patchy metabolic decompensation mechanisms take over in about 20% of acute mesenteric ischemias.[7] Classical findings (necrosis, transmural ischemic damage, perforation, pneumatosis, peritonism) are rarely evident in early phases, contributing to an often fatal diagnostic delay, and even a prompt execution of a computed tomography (CT) angiography may be inconclusive. In fact, CT lacks specificity (33%–60%) and presents high rates of false negative findings with unspecific features in almost half of patients.[8]

Recently, Calame et al.[9] reported that the only findings are pneumatosis and mesenteric gas, but both are inconclusive common features both for occlusive and Non-occlusive forms. A differential approach would therefore be envisaged in such cases where no vascular occlusions is found. On the other hand, current guidelines still define CT-angiography as the cornerstone for the diagnosis of acute NOMI.[3] Imaging, therefore, should be reconsidered only for a differential diagnostic rationale struggling with negative radiological findings even if in case of high clinical suspicion.

Facing with diagnostic uncertainty and methodological difficulties, a rationale for returning to an exploratory approach has been recently argued. Although it can be considered obsolete and invasive, the transmutation of experiences in minimally invasive approaches in an intensive care environment has allowed us to hypothesize a role for bedside exploratory laparoscopic techniques. However, both safety profile and technical feasibility still raise questions and claim debate especially in a complex clinical unstable scenario. Theoretically, in such patients with acute or life-threatening intra-abdominal conditions, avoiding transfers to operative rooms could preserve them from adjunctive negative consequences and minimize procedural additional mortality risks. As reported in the Society of American Gastrointestinal and Endoscopic Surgeons Guidelines, a bedside laparoscopy could represent an irreplaceable and complementary tool to overcome face diagnostic uncertainty, such as the potential advantages of a lower invasiveness, rapidity, and direct visual evaluation avoiding futile exploratory laparotomies.[10–12]

Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

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However, it would appear erroneous to contextualize a theoretical role for bedside laparoscopy as an exclusive alternative to imaging. Probably, a proper dimension for a minimally invasive surgical rationale would be relegated to conditions of radiological uncertainty and high clinical suspicion in which a late diagnosis could jeopardize rescue maneuvers and interfere with the patients' prognosis.

In a retrospective study including 20 patients with suspected NOMI undergoing a dual-stage ICU bedside exploratory laparoscopy, Cocorullo et al.[13] reported that intense pallor of the visceral walls was the main laparoscopic evidence and that, in 70% of cases, the perfusion impairment was multisplanchnic. Surprisingly, in all cases, no pathognomonic tomographic signs of intestinal ischemia or necrosis were reported. At the first exploration, 30% underwent intestinal resection, whereas, at 48 hours, there was recurrence of intestinal infarction in 4 resected patients and necrosis in a further 5 patients. An early laparoscopic positivity correlated with significantly higher mortality rates compared with a deferred onset (50% vs. 40%), whereas a rapid supportive therapeutic strategy noticeably reduced mortality rates to 11%. No related procedural complications were reported, demonstrating safety and feasibility of bedside laparoscopy in critically ill ICU patients.

Bergamini et al.,[14] speculating about the role of bedside laparoscopy in ICU for suspected NOMI in patients with inconclusive CT scans, demonstrated a safer profile of a minimally invasive approach rather than laparotomy (7.6% vs. 31.8%) with shorter operative time (98 ± 31 vs. 138 ± 39, $P = 0.003$) and reduced blood loss (280 ± 52 vs. 315 ± 38, $P = 0.028$). However, laparoscopy presented higher false-positive rates (65% vs. 35%, $P = 0.004$) because of tactile feedback limits, reduced visceral exposure, need for sequential maneuvering angles, and pneumoperitoneum limits.

However, scarcity of literature and conflicting position statements do not account for the clinical staging role of an exploratory approach. Moreover, the diagnostic utility cannot ignore effects on patients' clinical conditions in such complex settings and, in particular, adjunctive surgical stress, which could prematurely precipitate unstable conditions. Carbon dioxide insufflation leads to increased intra-abdominal pressure and hypercapnia in response to mechanical and neuromodulatory responses, resulting in inferior caval compression, reduction of splanchnic flow, and diaphragmatic displacement. Moreover, altered compensation mechanisms could result in unpredictable clinical effects. Complex adaptations to caval compression involve a biphasic response, where a moderate increase in intra-abdominal pressure (5 mmHg) results in an increased cardiac output and right atrial pressure, whereas the plateau phase (15 mmHg) corresponds to a reduction in cardiac output, stroke volume, cardiac index, and venous return,[15] with splanchnic flow reversal on the mesenteric-portal system, aggravating the microvascular metabolic mismatch of NOMI. Moreover, a cardiodepressive response is also mediated by a transient increase in the mean arterial pressure and systemic vascular resistances. Among the neurohormonal response, a release of plasma catecholamines, cortisol, vasopressin, renin, and aldosterone has been demonstrated.[16] Concerning mechanical effects, diaphragmatic displacement would result into a reduction in lung compliance with an increase in the peak and plateau pressures with a reduction in functional residual capacity, increased ventilatory-perfusion mismatch, hypoxemia, and hypercapnia. Finally, peritoneal stretching would increase vagal tone, which could lead to bradyarrhythmias and asysolia (Fig. 1).

The systemic effects of pneumoperitoneum could however be mitigated by laparoscopic technical tricks. A low-pressure induction flow would allow for the creation of a satisfactory pneumoperitoneum with a good operating field. A reduced pneumoperitoneum, in a neutral supine position, would avoid autonomic compensation mechanisms and make suitable accesses for an intraperitoneal exploration. Finally, in the case of obese patients or patients with poor abdominal wall compliance, extranatomic fascial traction could be used as in the case of gasless laparoscopic surgery.

Faced with the absence of definite indications or contraindications on eligibility for exploratory bedside laparoscopy, the rationale should refer to well-known principles of emergency laparoscopy. Critically ill patients should be candidate only if hemodynamically stables.[17] Another murky aspect relies upon the timing for execution. As reported by the experience of Cocorullo et al.,[13] a 2-stage approach would

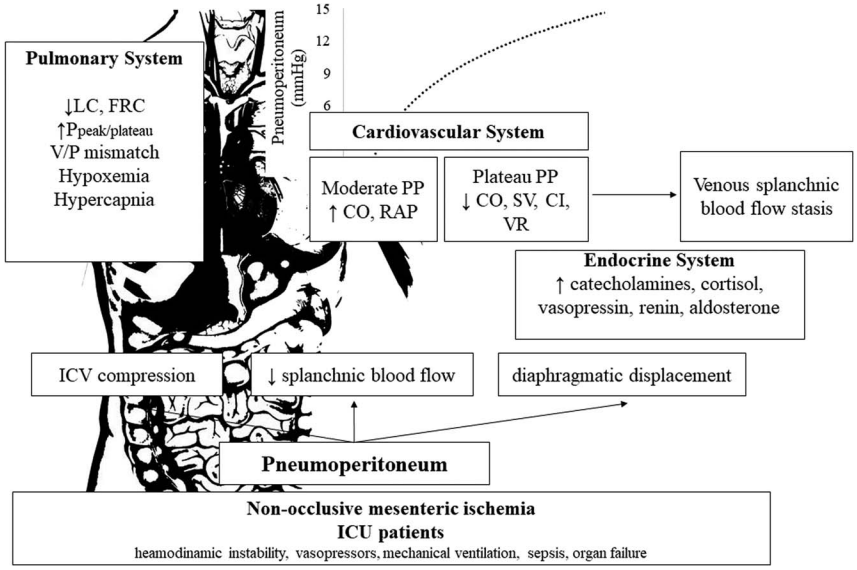

Figure 1. Systemic effects of pneumoperitoneum induction. CI, cardiac index; CO, cardiac output; FRC, functional residual capacity; ICU, intensive care unit; IVC, inferior vein cava; LC, lung compliance; PP, pneumoperitoneum; RAP, right atrial pressure; SV, stroke volume; V/P, ventilatory to perfusion; VR, venous return.

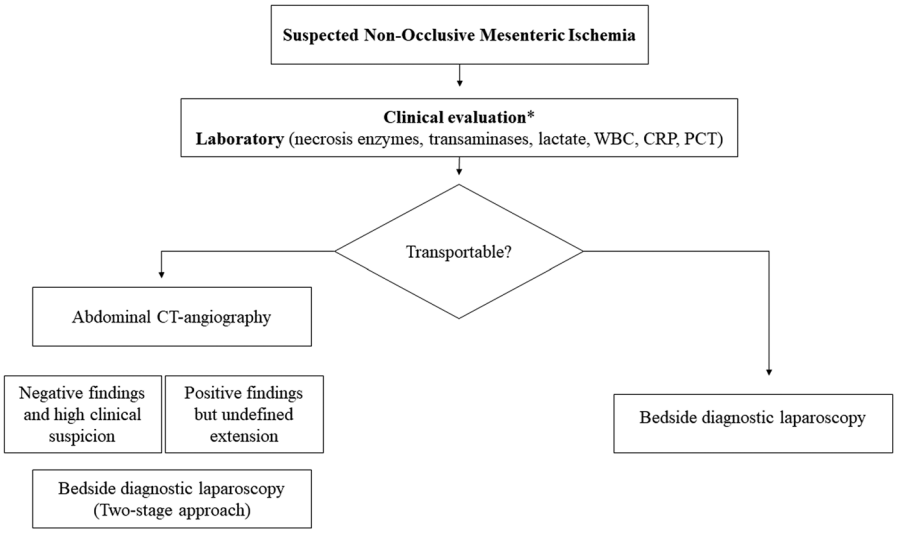

Figure 2. Diagnostic and workup flow chart for suspected NOMI. CRP, C-reactive protein; CT, computed tomography; NOMI, Non-occlusive mesenteric ischemia; PCT, procalcitonin; WBC, white blood cells count.

allow to diagnose both hyperacute and subtle onsets with the highest diagnostic performance rate but at the expense of an undefined incremental surgical risk.

In fact, it is a common clinical practice to face discrepancy between clinical-laboratory and radiological findings. In this scenario, the risks of slipping toward a diagnostic uncertainty, translating into a therapeutic delay and irreparably prejudicing the same indications for bedside laparoscopy due to the progressive multiorgan failure, significantly increase. Beyond medicolegal aspects between loss of chance of survival and overtreatment, a priority is the correct definition of the patient who is a candidate for a minimally invasive surgical approach in a resuscitation setting.

Probably, only the juxtaposition and dynamics between laboratory and radiological findings (eg, significant increase in tissue necrosis enzymes, lactic acidosis, increase in transaminases, changes in absolute visceral enhancement, appearance or increase in peritoneal effusion, peritoneal stranding, venous pneumatosis) should guide for a proper and strict indication. Finally, the rationale for intransportable patients still appears too poorly defined, as clinical conditions would similarly affect a transfer to operating rooms for subsequent resective surgery (Fig. 2).

In conclusion, bedside laparoscopy definitely provides some advantages in critically ill ICU patients with suspected NOMI, although the constraints of this strategy, such as techniques, limited surface window, and low sensitivity at the onset resulting in a false-negative exploration should be considered. This latter aspect could be mitigated by intraperitoneal navigation with vital dyes such as indocyanine green. Further aspects are timing and careful selection of the patients who could benefit from a direct endoscopic approach, as reference indicators or threshold for eligibility still lacks.

Conflict of interest statement

The authors declare no conflict of interest.

Author contributions

Barone M, Frontera R, and Liouras RV contributed to the concept. Barone M, Serano L, Iovino CG, Ippoliti M contributed to the design. Mucilli F, Vetrugno L, and Maggiore SM contributed to the supervision. Barone M, Frontera R, Liouras RV, Serano L contributed to materials. Barone M, Serano L, Ippoliti M collected and processed data. Barone M and Frontera R contributed to analysis and/or interpretation. Frontera R, Liouras RV, Iovino CG, and Ippoliti M contributed to literature search. All authors wrote and reviewed the manuscript.

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