

Prognostic Factors in Non-Occlusive Mesenteric Ischemia: A Pragmatic Pre-Operative Score for the Prediction of 28-day Mortality

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Abstract

Background

The prognosis of critical ill patients with non-occlusive mesenteric ischemia (NOMI) is poor and not fully understood. Preoperative prognostic factors are needed. The aim of this study was to determine preoperative factors associated with 28-day mortality in a cohort of ICU patients requiring laparotomy for NOMI. The secondary objective was to determine general prognostic factors associated with NOMI.

Methods

This retrospective observational study was performed in a University Hospital among critically ill patients 18 years old or older who underwent a laparotomy for NOMI from January 1, 2009 to December 31, 2019, and who had an available contrast enhanced CT with at least one portal venous phase. Variables were collected at the time of the CT. All variables associated with 28-day mortality were entered into a multivariate cox regression model and were used to compute a NOMI mortality score.

Results

During the study period, 154 patients underwent laparotomy for NOMI after having benefited from an abdominal enhanced CT. The 28-day mortality rate was 56%. Variable at the time of ICU admission and at the time of the CT were collected. Surgical and histopathologic findings were recorded. Multivariable analyses on 28-day mortality including variables at the time of the CT identified three independent variables (i.e. lactates > 7mmol/l, prothrombin rate < 60% and kidney infarction), included in a simple mortality score. For each variable associated with 28 days mortality, 1 point was attributed. Among the study population, the probability of 28-day mortality was 26% (11/42), 54% (26/48), 77% (23/30) and 100% (21/21) for a survival score of 0, 1, 2 and 3, respectively. A second explorative multivariate cox regression model including the variables at the time of ICU admission showed that jejunal transmural necrosis was the only operative finding associated with death (HR = 2.26 CI95%[1.14–4.71]).

Conclusion

We identified three preoperative factors independently associated with short-

Introduction

Acute mesenteric ischemia (AMI) is a life-threatening condition associated with high mortality, ranging from 20 to 77%¹⁻⁷. However, the different mechanisms leading to AMI do not share the same prognosis. Prognosis in arterial occlusive mesenteric ischemia (OMI) is well established and depends mainly on the

time to revascularization of the superior mesenteric artery (SMA) and the extent of bowel necrosis^{8,9}. The ultimate stage of bowel necrosis is transmural bowel necrosis, which is irreversible, and requires surgical resection⁸.

Prognosis in non-occlusive mesenteric ischemia (NOMI)¹⁰ is poorer than in OMI. NOMI occurs essentially in critically ill patients, and can be considered as the ultimate stage of gastro-intestinal failure^{11,12}. Mortality is very high, ranging from 52 to 77%^{4,6,13}. The prognosis of NOMI is related not only to bowel ischemia, but also to kidney and liver ischemia¹⁴. In this context, bowel ischemia can be considered more as symptom of multi-organ failure^{1,13}.

Although the two diseases are very different from a pathophysiological point of view, most of the previously published studies examining the prognosis of AMI have included both OMI and NOMI¹⁻³. Furthermore, among the studies focused on NOMI^{4,6}, a substantial proportion of patients did not undergo laparotomy, resulting in a potential bias in predicting prognosis, since the definition of NOMI is neither standardized nor consensual.

While predictors of transmural bowel necrosis might help determine which patients need emergency laparotomy, other parameters are required to identify patients who are unlikely to benefit from surgery. Indeed, some very sick patients with NOMI have a poor prognosis, independently of the presence or extent of transmural necrosis. Identifying patients with no chance of survival may improve care by avoiding invasive treatment such as emergency laparotomy.

Thus, the aim of this study was to determine preoperative factors associated with 28-day mortality in a retrospective cohort of intensive care unit (ICU) patients undergoing laparotomy for NOMI. The secondary objective was to determine overall prognostic factors in NOMI.

Materials And Methods

From January 1, 2009 to December 31, 2019, all consecutive patients admitted to the surgical and medical ICUs of the university hospital of Besancon, France, and undergoing exploratory laparotomy were assessed for inclusion. The inclusion criteria were: 1) laparotomy for NOMI; and 2) an available contrast enhanced CT with at least one portal phase. NOMI was defined as evidence of bowel suffering without mesenteric vessels occlusion on preoperative abdominal computed tomography (CT) that required surgical exploration. During the inclusion period, 227 patients underwent laparotomy for NOMI. Patients with a history of occlusive AMI or with superior mesenteric artery occlusion (n=10), patients without available CT scan (n=26) and patients without enhanced abdominal CT (n=25) and without available pathological reports or medical charts (n= 12) were excluded (**Figure 1**). Data were retrospectively extracted from the medical charts. Clinical and biological values at ICU admission and at the time of the CT were extracted. Surgical findings were collected. Macroscopic necrosis at surgical exploration and pathological diagnosis when bowel resection was performed, were collected according to bowel segment (i.e. stomach, jejunum, ileum, right colon, left colon). In case of open-close surgery, surgical necrosis was

considered as transmural necrosis. Survival was calculated as the time from the abdominal CT until death, or the study cut-off date (31 December 2019). Patients still alive at the study cut-off date were censored.

Imaging analysis

All abdominal CT scans were retrospectively reviewed by two radiologists (P.V. and P.C. with 5 and 9 years of experience respectively in the field of abdominal imaging) on a PACS workstation (Carestream Health, Rochester, NY), followed by a consensus read in case of disagreement. Radiologists were blinded to the surgical and pathological results, and to the final outcome. Ischemic features of abdominal organ were analyzed, i.e.: Absence of small bowel enhancement, hyperenhancement of the adrenal gland, kidney, liver and spleen infarction (**Figure 2**). Portal venous gas and the maximal diameter of SMA and celiac trunk was also recorded.

Statistical Analysis

Categorical data are expressed as number and percentage and were compared using the chi square or Fisher's exact test. Continuous variables are expressed as mean±standard deviation and were compared with the Student t test when the distribution was normal, or expressed as median and interquartiles (IQR) and compared by the Wilcoxon test, when the distribution was not normal.

To establish a preoperative prognostic score for 28-day mortality, a first multivariate Cox regression model was built. In this first model, all variables recorded at the time of the CT and associated with 28-day mortality were selected. For laboratory tests, cut-off levels for continuous variables were chosen according to the best area under the curve (AUC). For liver function variables (i.e. prothrombin rate, AST, and bilirubin), to avoid collinearity, only the variable most strongly associated with 28-day mortality was selected, and the cut-off level was also determined as the best AUC for the prediction of 28-day mortality. For the abdominal CT variables, the following variables were entered into the model: absence of small bowel enhancement, dichotomized axial diameter of celiac axis and SMA. Among variables relating to other organ involvement, only those most strongly associated with 28-day mortality were entered, due to collinearity (i.e. kidney infarction). Selection of the best model was made using a backward stepwise procedure. The three variables found to be independently associated with 28-day mortality were used to compute a NOMI mortality score. Lactate level and prothrombin rate were entered in another multivariate Cox regression model as continuous variables, and a backward stepwise process was used to select the final variables to construct a nomogram. Performance of the mortality score and of the nomogram for the prediction of 28-day mortality in patients suffering from NOMI was evaluated by means of the area under the receiver operating characteristics curve (AUROC) and compared by the DeLong test.

A final multivariate model was constructed for exploratory purposes, and included age, sex, and all variables associated with mortality at 28 days with a p-value <0.05 by univariate analysis. This regression model included variables at ICU admission, variables at the time of CT, CT features, and operative findings.

Kaplan Meier curves were built for the three mortality score variables, and for the score itself. Kaplan Meier curves were also built according to the three main operative situations (i.e. open-close surgery, bowel resection, no surgical necrosis). All tests were two-sided. A p-value <0.05 was considered statistically significant. All analyses were performed with R version 3.4.4 (R Core Team 2017) and results were reported according to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines¹⁵.

Ethical considerations

The study was approved by the institutional review board, and the need for informed consent was waived. General information was provided to the families in the ICU via posters in the patients' rooms and in waiting areas explaining that data concerning their next of kin might be used later for scientific analyses and could be published using anonymized data, unless they explicitly expressed their opposition. No opposition was expressed for this study.

Results

During the study period, 154 patients (mean age was 69 ± 10 years) underwent laparotomy after enhanced abdominal CT scan. Laparotomy was performed within 24 hours of the CT scan in 139 (92%) patients, one day later in 11 (7%), and two days later in 4 (3%). Overall, 110 patients (71%) underwent digestive resection. Among the remaining 44 patients, there was no appearance of transmural necrosis in 29 (19%), and extensive necrosis with open-close surgery in 15 (10%) (**Figure 1**). NOMI were secondary to surgery in 87/154 patients (56%) and to medical conditions in the remaining 67/154 patients (44%). Cardiac surgery was the main surgical etiology of NOMI (n=36, 23%) followed by abdominal surgery (n=27, 18%) and by aortic surgery (n=14, 9%). Sepsis was the main medical etiology of NOMI (n=23, 15%), followed by cardiorespiratory arrest (n=15, 10%), acute respiratory failure (n=14, 6%) and Hemorrhage (n=10, 6%). The mean time between ICU admission and abdominal CT was 2.7 days (range 0 – 33 days). Among the 154 patients, 87 (56%) died within 28 days after the abdominal CT.

Variables associated with 28-day mortality

Variables associated with 28-day mortality are summarized in **Table 1**. Regarding the variables at ICU admission, mean arterial pressure was lower in patients who died than in survivors ($p < 0.001$). Concerning the variable at the time of abdominal CT scan, patients who died within 28 days were more likely to have catecholamine infusion ($p < 0.001$). Platelet count and prothrombin rate were lower in patients who died ($p = 0.003$ and $p < 0.001$ respectively). Lactate levels, bilirubin and ASAT were higher in those who died ($p < 0.001$, $p = 0.008$ and $p < 0.001$ respectively).

The CT features associated with 28-day mortality are shown in **Table 1**. All the CT features suggestive of organ infarction were associated with death. Absence of small bowel enhancement, kidney infarction, liver and spleen infarction were more frequent in those who died ($p = 0.008$, $p < 0.001$, $p = 0.002$, $p = 0.003$ respectively). Axial diameter of the mesentery arteries was significantly lower in patients who died

($p = 0.007$ and $p < 0.001$ respectively). Portal venous gas was not associated with 28-days mortality (16 [18%] in survivors vs 7 [10%] in non survivors, $P=0.253$).

SMA diameter was not correlated with catecholamine infusion or noradrenaline flow (Pearson correlation coefficient, $r = -0.09$, 95%CI [0.25 - 0.06] and -0.09 , 95%CI [-0.25 - 0.07] respectively), whereas celiac trunk diameter was ($r = -0.27$; 95%CI [-0.41 - -0.12] and -0.22 , 95%CI [-0.36 - -0.06] for catecholamine infusion and noradrenaline flow respectively). Kidney infarction was not correlated with creatinine level ($r = -0.01$, 95%CI [-0.17 - 0.15]).

Pre-operative prognostic score for 28-day mortality

After multivariate analysis for the prediction of 28-day mortality including variables at the time of CT, a simple mortality score was established (**Table 2**). For each variable associated with 28-day mortality (i.e. lactates > 7 mmol/l, prothrombin rate < 60 % and kidney infarction), 1 point was attributed. Among the study population, probability of 28-day mortality was 26% (31/42), 54% (26/48), 77% (23/30) and 100% (21/21) for a mortality score of 0, 1, 2 and 3, respectively (**Figure 2**). The AUC for 28-day mortality prediction was 0.79, 95%CI [0.72-0.86]. The results of the Cox regression model including continuous variables (i.e. Lactates and prothrombin rate) with the nomogram built according to the four significant variables (i.e. Lactates, prothrombin rate, catecholamine infusion and kidney infarction), together are provided in **Supplementary Table 1** and **Supplementary Figure 1** respectively. The comparison of the AUC between the nomogram and the NOMI mortality score for 28 days mortality is available in the **Supplementary Figure 2**.

Influence of operative findings on 28-day mortality

The 15 patients with extensive necrosis and without bowel resection (i.e. open-close surgery) died within 24 hours. 6 of them (40%) had a mortality score of 3. Median survival in the 29 patients without surgical necrosis was 8 days, 95%CI [3 – 21 days] and 64 days, 95%CI [15 – 241 days] in the patients with bowel resection (**Supplementary Figure 3**). Both stomach necrosis visible at surgery, and transmural necrosis diagnosed by pathology were associated with 28-day mortality ($p = 0.01$, **Table 1**). Macroscopic small bowel necrosis appearance was not associated with 28-day mortality (40 [46%] in non survivors vs 26 [39%] in survivors, $P=0.2$, whereas small bowel transmural necrosis was (38 [44%] in non survivors vs 17 [25%] in survivors, $P=0.002$). Macroscopic jejunal necrosis appearance was associated with 28-day mortality (26 [30%] in non survivors vs 8 [12%] in survivors, $P<0.001$, as jejunal transmural necrosis was (25 [29%] in non survivors vs 7 [10%] in survivors, $P<0.001$). Macroscopic ileal necrosis appearance was not associated with 28-day mortality (32 [37%] in non survivors vs 22 [33%] in survivors, $P=0.2$, whereas small bowel transmural necrosis was (31 [36%] in non survivors vs 13 [19%] in survivors, $P=0.002$). Patients with small bowel transmural necrosis had a median survival of 2 days, 95%CI [1 - 19 days] vs 31 days, 95%CI [13 – 80 days] in patients without.

In the 110 patients with bowel resection, only small bowel transmural necrosis was associated with 28-day mortality (median survival 10 days, 95%CI [2 – 234 days] vs 80 days 95%CI [36 vs 1256 days]).

Overall multivariate analysis of prognostic factors in NOMI

The prognostic factors for 28-day mortality identified by multivariate analysis among the variables at ICU admission, at the time of the CT, and operative findings are provided in **Table 3**. Age was associated with an increased risk of death (HR = 1.03 CI95%[1.00 – 1.06], P = 0.025). Lactates >7 mmol/L (HR = 2.99 CI95%[1.63 – 5.47], P < 0.001), kidney infarction (HR = 1.88 CI95%[1.10 – 3.22], P = 0.021), and SMA diameter \leq 5 mm (HR = 1.77 CI95%[1.04 – 3.01], P = 0.034) were independent risk factors for death within 28 days. Among all the operative findings associated with death by univariate analysis, only jejunal transmural necrosis was an independent risk factor for 28-day mortality (HR = 2.26 CI95%[1.14 – 4.71], P = 0.019).

Discussion

In this analysis of a large single-center cohort of ICU patients with NOMI who underwent laparotomy, we observed a 28-day mortality rate of 56%. We also found that a simple score combining lactates > 7 mmol/L, prothrombin rate < 60% and kidney infarction on CT could accurately predict 28-day mortality with a good AUC (0.79, 95%CI [0.72-0.86]). Importantly, the 21 patients with a mortality score of 3 died within 10 days of their abdominal CT scan. This finding is of major relevance, since in patients with a mortality score of 3, laparotomy appears to be futile. Interestingly, the three factors in the score represent the three features of NOMI, since lactate, prothrombin rate and kidney infarction are reflected by gut, liver and kidney injury, respectively. Alternatively, in such a critical situation, one could suggest bedside abdominal surgical exploration. This approach has been proven feasible and safe and would allow rapid abdominal exploration before therapeutic decision making.

Lactate level is a useful laboratory test to estimate overall ischemia, especially gut ischemia¹. In OMI, it can be considered specific since the *primum movens* is gut ischemia. However, the situation is different in NOMI, as there is concomitant gut, kidney and liver failure. Lactates therefore lose their specificity for the prediction of intestinal necrosis and must therefore be used as a prognostic marker. Liver insufficiency seem to play a significant role in the prognosis of NOMI. All bloods tests of liver parameters were significantly associated with 28-day mortality by univariate analysis, and the mortality score includes a liver-associated variable (i.e. prothrombin rate). The liver and the gut are very close in terms of vascularization and function. As a consequence of this proximity, it can be hypothesized that ischemia of the liver and the gut occur at the same time, and that each impacts on prognosis independently.

Among all the organs involved in the low-flow state (i.e. bowel, liver, spleen, adrenal gland), kidney infarction was the most influential CT feature on 28-day mortality. In NOMI, on top of bowel infarction, kidney infarction can be considered as the breakdown of the last line of defense against the low flow state and ischemia. Integrating CT features when determining prognosis in NOMI is an interesting option, since their prognostic value has often been neglected in favor of their diagnostic value^{16,17}. The diameter of the mesenteric vessels (SMA or celiac trunk) provides essential data about the low-flow state, and above all, about how the organism is adapting to the low flow state. Interestingly, the axial diameter of

the SMA was not correlated with catecholamine infusion or noradrenaline flow, whereas celiac trunk diameter was. The SMA axial diameter can thus be considered as an independent risk factor for death, independently of catecholamine infusion. This is similar to what we observed with kidney infarction, which was not correlated with creatinine levels. In summary, abdominal CT reveals prognostic features that appear to be independent of clinical or blood test data, and which must therefore be considered when assessing the prognosis of a patient with NOMI.

Bowel irreversible transmural necrosis (ITN) has been extensively studied in OMI, and transitioning from reversible to non reversible (mural – transmural) necrosis is a turning point in patient prognosis in OMI⁸. To the best of our knowledge, our study is the first to assess the impact of transmural necrosis in NOMI. First, we showed that even in NOMI, small bowel ITN remains the only relevant operative finding. Indeed, surgical necrosis including mural and transmural necrosis was not associated with 28-day mortality, whereas small bowel ITN was. Interestingly, more than the extent of ITN, jejunal ITN was the only independent prognostic factor for 28-day mortality. We found that colon necrosis had no impact on survival in these patients. All these findings show that even in NOMI, small bowel ITN remains the determinant operative finding in the prognosis and must therefore be the main focus of interest in the diagnosis of NOMI.

Strengths

Our study has several strengths. NOMI was histologically proven in 71%, and surgically proven in 81%. Second, the NOMI mortality score makes multimodal assessment possible, integrating biological and radiological features. Finally, we describe a pragmatic approach for predicting prognosis using simple parameters available at the time of CT scan. NOMI usually occurs as a second hit after major surgery or sepsis, resulting in a remarkably high mortality rate. Therefore, in some case, clinicians may consider care limitation when a very sick patient develops a second shock related to NOMI. In such situations, the NOMI mortality score might be a simple tool to identify patients with a very high probability of death despite emergency laparotomy, avoiding futile and invasive procedures.

Limitations

Our study also has some limitations. First, because it was a single-centre study, a center effect cannot be ruled out. Second, we did not report data related to medical management of NOMI, but all patients were treated in a similar manner, with vascular filling, vasopressors, and antibiotics. Third, the proportion of death resulting from withdrawal or withholding of care was not available. However, only 6/15 (40%) patients with a mortality score of 3 underwent an open-close surgery, suggesting that the mortality score can overcome the confusion bias associated with subsequent withdrawal of therapy, following laparotomy showing extensive necrosis without possibility of resection. Finally, we were unable to assess whether the delay in diagnosis of bowel ischemia had a real influence on the prognosis. Indeed, the diagnosis of NOMI is usually made at the time of a CT scan showing evidence of bowel ischemia, whereas the exact time of onset of ischemia is impossible to identify.

Conclusion

We identified three preoperative factors independently associated with short-term mortality in NOMI, namely lactates > 7 mmol/L, prothrombin rate < 60% and kidney infarction on CT. These findings were combined into a simple score that was able to accurately predict 28-day mortality. These findings may help to avoid unnecessary laparotomies in patients with a high likelihood of death. In addition, we found that jejunal transmural necrosis was the only independent operative finding associated with 28-day mortality.

Abbreviations

AMI: Acute Mesenteric Ischemia

AUROC: Area Under the Receiver Operating Characteristics Curve

AST: Aspartate aminotransferase

CT: Computed Tomography

CI: Confidence interval

ICU: Intensive Care Unit

IQR: Interquartile Range

HR: Hazard ratio

NOMI: Non-Occlusive Mesenteric Ischemia

OMI: Occlusive Mesenteric Ischemia

SMA: Superior mesenteric artery

Declarations

- **Specific author contributions:**
 - Planning and/or conducting the study: P.C., H.W., A.D.;
 - Collecting data P.C., P.V., H.W.,
 - Interpreting data: S.P.F., B.H., Z.L., E.D., G.P., P.C.,
 - Drafting the manuscript: P.C., H.W., A.D., G.P., E.D.
 - All authors avec read and approved the final manuscript
- **Ethics approval and consent to participate:** The study was approved by the institutional review board, and the need for informed consent was waived. General information was provided to the families in

the ICU via posters in the patients' rooms and in waiting areas explaining that data concerning their next of kin might be used later for scientific analyses and could be published using anonymized data, unless they explicitly expressed their opposition. No opposition was expressed for this study.

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- **Acknowledgments:** None
- **Availability of data and material:** The datasets used and analysed during the current study are available from the corresponding author on reasonable request.

References

1. Leone M, Bechis C, Baumstarck K, et al. Outcome of acute mesenteric ischemia in the intensive care unit: a retrospective, multicenter study of 780 cases. *Intensive Care Med* 2015;41(4):667–76.
2. Caluwaerts M, Castanares-Zapatero D, Laterre P-F, Hantson P. Prognostic factors of acute mesenteric ischemia in ICU patients. *BMC Gastroenterol* 2019;19(1):80.
3. Acosta-Merida MA, Marchena-Gomez J, Hemmersbach-Miller M, Roque-Castellano C, Hernandez-Romero JM. Identification of risk factors for perioperative mortality in acute mesenteric ischemia. *World J Surg* 2006;30(8):1579–85.
4. Suzuki S, Kondo H, Furukawa A, et al. Prognostic Factors of Preoperative Examinations for Non-occlusive Mesenteric Ischemia: A Multicenter Retrospective Project Study Conducted by the Japanese Society for Abdominal Emergency Medicine. *World J Surg* 2020;44(11):3687–94.
5. Stahl K, Busch M, Maschke SK, et al. A Retrospective Analysis of Nonocclusive Mesenteric Ischemia in Medical and Surgical ICU Patients: Clinical Data on Demography, Clinical Signs, and Survival. *J Intensive Care Med* 2020;35(11):1162–72.
6. Sakamoto T, Fujiogi M, Matsui H, Fushimi K, Yasunaga H. Clinical features and outcomes of nonocclusive mesenteric ischemia after cardiac surgery: a retrospective cohort study. *Heart Vessels* 2020;35(5):630–6.
7. S A, A A, P S, O E. Epidemiology, risk and prognostic factors in mesenteric venous thrombosis [Internet]. *Br. J. Surg.* 2008 [cited 2020 Oct 14];95(10). Available from: <https://pubmed.ncbi.nlm.nih.gov/18720461/>
8. on behalf of the SURVI group, Nuzzo A, Maggiori L, et al. Predictive Factors of Intestinal Necrosis in Acute Mesenteric Ischemia: Prospective Study from an Intestinal Stroke Center. *Am J Gastroenterol* 2017;112(4):597–605.
9. Kärkkäinen JM, Acosta S. Acute mesenteric ischemia (part I) - Incidence, etiologies, and how to improve early diagnosis. *Best Pract Res Clin Gastroenterol* 2017;31(1):15–25.
10. Schoots IG, Koffeman GI, Legemate DA, Levi M, van Gulik TM. Systematic review of survival after acute mesenteric ischaemia according to disease aetiology. *Br J Surg* 2004;91(1):17–27.

11. Alukal JJ, Thuluvath PJ. Gastrointestinal Failure in Critically Ill Patients With Cirrhosis. *Am J Gastroenterol* 2019;114(8):1231–7.
12. Padar M, Starkopf J, Uusvel G, Reintam Blaser A. Gastrointestinal failure affects outcome of intensive care. *J Crit Care* 2019;52:103–8.
13. Bourcier S, Oudjit A, Goudard G, et al. Diagnosis of non-occlusive acute mesenteric ischemia in the intensive care unit. *Ann Intensive Care* 2016;6(1):112.
14. Guillaume A, Pili-Floury S, Chocron S, et al. Acute Mesenteric Ischemia Among Postcardiac Surgery Patients Presenting with Multiple Organ Failure. *Shock Augusta Ga* 2017;47(3):296–302.
15. E von E, Dg A, M E, Sj P, Pc G, Jp V. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) Statement: guidelines for reporting observational studies [Internet]. *Int. J. Surg. Lond. Engl.* 2014 [cited 2020 Dec 2];12(12). Available from: <https://pubmed.ncbi.nlm.nih.gov/25046131/>
16. Mazzei MA, Gentili F, Mazzei FG, Grassi R, Volterrani L. Non-occlusive mesenteric ischaemia: CT findings, clinical outcomes and assessment of the diameter of the superior mesenteric artery: Don't forget the reperfusion process! *Br J Radiol* 2018;20180736.
17. Nakamura Y, Urashima M, Toyota N, et al. Non-occlusive mesenteric ischemia (NOMI): utility of measuring the diameters of the superior mesenteric artery and superior mesenteric vein at multidetector CT. *Jpn J Radiol* 2013;

Tables

Table 1. Characteristics of the study population associated with 28-day survival (univariate analysis).

Variables	n = 154	No = 87	Yes = 67	P value
Age, years	68.6 ± 9.9	69.5 ± 10.3	67.5 ± 9.2	0.209
Sex (male)	100 (65)	57	43	0.998
Variables at ICU admission				
Mean arterial pressure, mmHg	76 (61 - 88)	71 (59 - 88)	78 (67 - 89)	0.001
Catecholamine infusion	121 (79)	65 (75)	56 (84)	0.189
Mechanical ventilation	98 (64)	69 (79)	29 (43)	0.001
Variables at the time of the CT				
Mechanical ventilation	110 (71)	58 (67)	52 (77)	0.142
Respiratory rate (breaths/minute)	22 (22 - 26)	22 (18 - 26)	23 (20 - 28)	0.282
Catecholamine infusion	100 (65)	68 (78)	32 (48)	0.001
Noradrenalin, mg/h,	0.6 (0 - 1.4)	1 (0.3 - 1.7)	0 (0 - 0.8)	0.001
Hemoglobin (g/dL)	10.3 ± 2.4	9.8 (8.6 - 11.2)	10.1 (8.9 - 12.1)	0.106
Platelets (/mm ³)	173 (120 - 265)	154 (113 - 214)	205 (145 - 301)	0.003
Lactates, mmol/L	4.2 (2.1 - 7)	5.3 (3.3 - 6.4)	3.3 (1.9 - 6.4)	0.001
Creatinine, mmol/L	152 (111 - 208)	156 (107 - 218)	148 (118 - 190)	0.814
Bilirubin, mmol/L	16 (12 - 41)	22 (13 - 62)	14 (11 - 25)	0.008
ASAT, IU/L; median (IQR)	80 (36 - 477)	190 (49 - 860)	48 (32 - 111)	< 0.001
Prothrombin rate, %	55 (38 - 71)	47 (34 - 59)	69 (53 - 75)	< 0.001
pH	7.32 (7.22 - 7.41)	7.27 (7.14 - 7.38)	7.39 (7.28 - 7.45)	< 0.001
PaO ₂ (mmHg)	10.5 (8.6 - 14)	10.9 (8.6 - 14.9)	10 (8.7 - 12.4)	0.379
PaCO ₂ (mmHg)	4.6 (3.8 - 5.8)	4.6 (3.8 - 5.63)	4.7 (4.2 - 5.8)	0.328
Bicarbonates (mmol/l)	18 (14 - 22)	16 (12 - 19)	21 (18 - 24)	< 0.001
CT variables				
Absence of small bowel enhancement	61 (40)	43 (49)	18 (27)	0.008

Hyperenhancement of adrenal gland	70 (45)	51 (59)	19 (28)	<.001
Kidney infarction	51 (33)	42 (48)	9 (13)	<.001
Liver infarction	39 (25)	31 (36)	8 (12)	0.002
Spleen infarction	75 (49)	52 (60)	23 (34)	0.003
SMA axial diameter (mm)	6 (5 - 7)	5 (5 - 6)	6 (5 - 7)	0.007
Coeliac trunk axial diameter (mm)	5 (4 - 6)	5 (4 - 6)	6 (5 - 8)	< 0.001
Bowel transmural necrosis				
Stomach	6 (4)	5 (6)	1 (1)	0.010
Small bowel	55 (36)	38 (44)	17 (25)	0.002
Jejunum	32 (21)	25 (29)	7 (10)	< 0.001
Ileum	44 (29)	31 (36)	13 (19)	0.001
Colon	75 (49)	37 (42)	38 (57)	0.3
Right colon	44 (29)	25 (29)	19 (28)	0.5
Left colon	55 (36)	31 (36)	24 (36)	0.6
Number of bowel segments involved	1 (0 - 2)	1 (0 - 2)	1 (0 - 1)	< 0.001

AST= Aspartate aminotransferase; CT= computed tomography; ICU=Intensive care unit;
NAD=noradrenalin; PaO2= Arterial blood oxygen tension; PaCO2= Arterial blood carbon dioxide tension;
SMA=Superior mesentery artery;

Table 2. Preoperative prediction of 28-day mortality. Multivariate Cox regression model.

Variables	HR	IC 95%	P-value	Day 28 survival score
Clinical data				
Catecholamine infusion	1.72	0.95 – 3.10	0.071	-
Bloods tests				
Lactates > 7 mmol/l	2.84	1.71 – 4.71	<0.001	1 point
Prothrombin rate < 60%	1.84	1.03 – 3.27	0.038	1 point
CT features				
Kidney infarction	2.21	1.38 – 3.58	<0.001	1 point
Superior mesentery artery diameter ≤ 5 mm	1.50	0.92 – 2.43	0.097	-

**18 observations deleted due to missingness (final model included 136 patients)*

CT= Computed Tomography; CI= Confidence Intervals; HR= Hazard Ratio.

Table 3. Overall multivariate analysis including variables at ICU admission, at the time of the CT, and operative findings.

	HR	CI95%	P-value
Variables at ICU admission			
Age	1.03	1.00 – 1.06	0.025
Male sex	-	-	0.061
Average blood pressure < 70 mmHg	-	-	0.070
Mechanical ventilation	-	-	0.204
Variables at the time of the CT			
catecholamine infusion	-	-	0.371
Lactates > 7 mmol/l	2.99	1.63 – 5.47	<0.001
Prothrombin rate < 60%	-	-	0.140
pH < 7.33	-	-	0.777
Kidney infarction	1.88	1.10 – 3.22	0.021
Superior mesentery artery diameter ≤ 5 mm	1.77	1.04 – 3.01	0.034
Operative findings			
Stomach transmural necrosis	-	-	0.374
jejunal transmural necrosis	2.26	1.14 – 4.71	0.019
Ileal transmural necrosis	-	-	0.416
Extent of transmural necrosis (number of segment)	-	-	0.871

**18 observations deleted due to missing data (final model included 136 patients)*

CT= Computed Tomography; CI= Confidence Intervals; ICU= Intensive Care Unit; HR= Hazard Ratio.

Figures

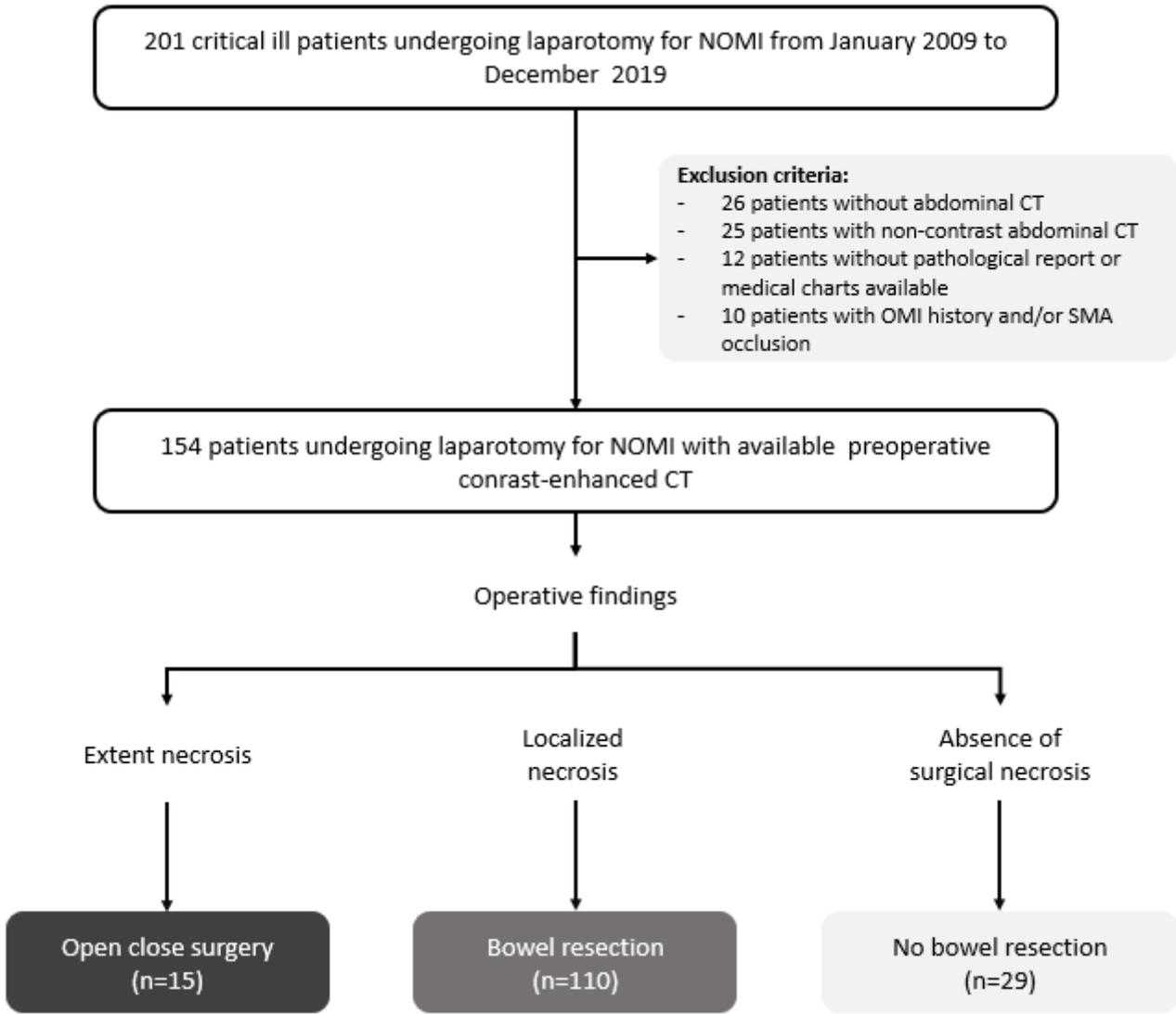


Figure 1

Flow chart of the study population. CT: computed tomography; OMI: Occlusive Mesenteric Ischemia; NOMI: Non-Occlusive mesenteric ischemia; SMA: Superior Mesentery Artery

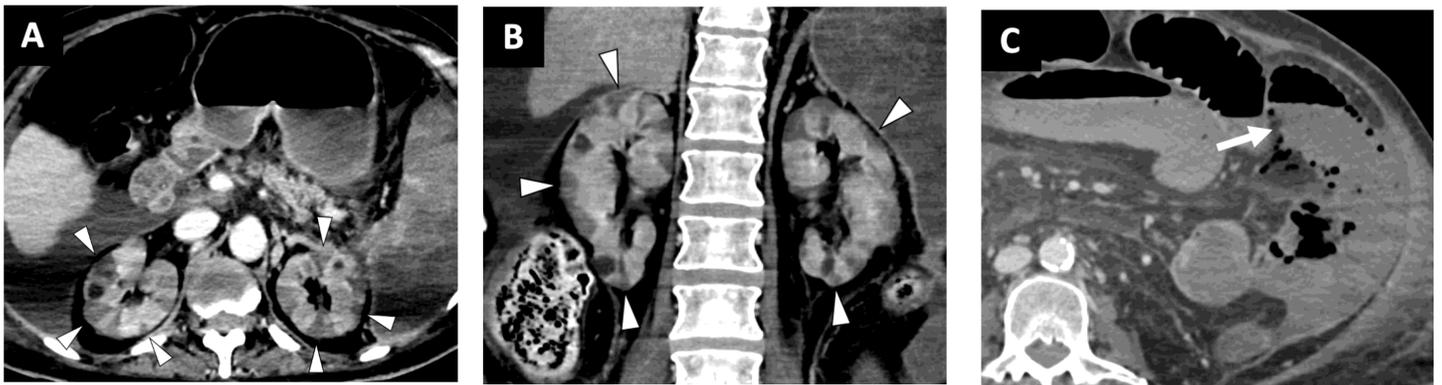


Figure 2

68-year-old man with non-occlusive mesenteric ischemia following sepsis with lactate level of 9.55 mmol/L, and a prothrombin rate of 43%. A. Abdominal CT in the portal venous phase shows kidney infarctions (arrowheads). B. Coronal reconstruction. Kidney infarctions (arrows). C. Same examination, abdominal slices. Absence of jejunal wall enhancement (arrow), pneumatosis and mesenteric gas. The patient underwent laparotomy associated with jejunal resection. Pathological analysis confirmed transmural necrosis. The patient died 2 days after abdominal CT.

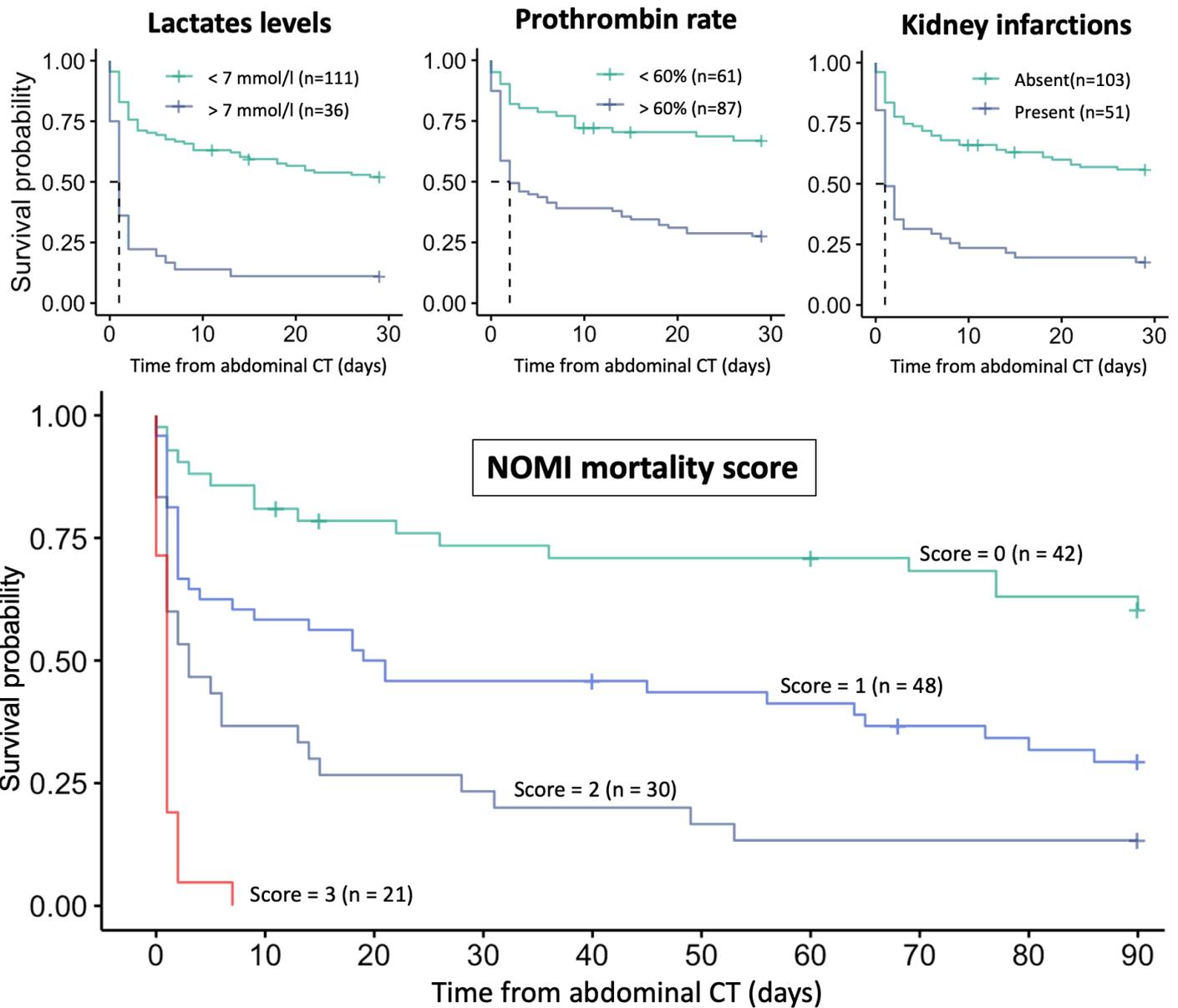


Figure 3

Survival probability curves (Kaplan–Meier method) according to the three factors of the mortality score and to the NOMI mortality score.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- [SUPPLEMENTARYFIGURE1.docx](#)
- [SUPPLEMENTARYFIGURE2.docx](#)
- [SUPPLEMENTARYFIGURE3.docx](#)
- [SUPPLEMENTARYTABLE1.docx](#)