Failure Risk Factor For Non-Operative Treatment of Splenic Trauma

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Research article

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Abstract

**Background:** Splenic trauma is a common pattern for admission in blunt abdominal trauma. The objective of this study is to identify risk factors for failure of non-operative management (NOM) in splenic trauma.

**Methods:** This is a retrospective monocentric analysis of a prospectively collected database. All patients admitted in the university hospital of Nice [Centre Hospitalier Universitaire (CHU) de Nice, France] for a splenic trauma from January 1\(^{st}\) 2006 to January 6\(^{th}\) 2018 were included. Primary outcome was the need for delayed splenectomy as an indicator of NOM failure.

**Results:** Two-hundred-eighty patients were included in this study. Most splenic lesions were severe grades (grade 3 or higher). In total, 83 splenectomies were performed urgently, i.e. 29% of patients; 88 angio-embolizations, i.e., 31% of patients with a success rate greater than 80%; 14.7% of 136 patients who had no previous angio-embolization required secondary splenectomy; 19.7% of the 61 patients who had anterior angio-embolization required secondary splenectomy. Age was not found associated with a higher failure rate (44 years in successful embolization vs 37.5 years in NOM-failure group, p = 0.15). Higher drop in hemoglobin levels between admission and 6 hours after admission was detected in the embolization failure group (-1.44 g/dl) as compared with the successful group (-0.68 g/dl), which approached statistical significance (p = 0.064).

**Conclusions:** Hemoglobin monitoring in the hours following the admission of a patient with splenic trauma might be an important factor during the medical supervision of hemodynamically stable patients. Early identification of patients at high risk of NOM failure by hemoglobin monitoring may prevent late splenectomy.

**Level of evidence:** IV (retrospective study)

**Background**

Abdominal trauma is present in 7 to 10% of severely injured patients. In the case of blunt abdominal trauma, 45% have a splenic lesion, making it the most frequently injured organ in blunt abdominal trauma (1).

CT-scan is the reference imaging exam for trauma patients whose hemodynamic status is considered stable or responds to vascular filling (2, 3). CT-scan can identify splenic lesions, provide a morphological description according to the AAST classification (American Association for the Surgery of Trauma) (4), but the scanner is not the only parameter to consider when choosing the best treatment for trauma patients.

The gold-standard treatment for patients with splenic injury in blunt abdominal trauma with stable hemodynamic status is the non-operative management (NOM), which is performed in approximately 80%
of patients (5, 6). This medical management is effective in 62 to 92% of cases, likely because of the increasingly frequent use of splenic angioembolization (5, 7–10). It is difficult to identify patients who will have NOM failure and require secondary splenectomy (5, 6, 11–14). Many risk factors for NOM failure have been proposed, but none can accurately predict the failure of NOM.

This study aims to find parameters to predict NOM failure in case of blunt abdominal trauma with a splenic lesion, which will help in selecting patients in whom splenectomy first intention would be indicated.

**Methods**

This a retrospective monocentric study of a prospectively collected database. All patients admitted in the university hospital of Nice (Centre Hospitalier Universitaire (CHU) de Nice, France) for a splenic trauma from January 1st 2006 to January 6th 2018 were included. Primary outcome was the need for delayed splenectomy in patients treated with NOM as an indicator of NOM failure.

The following data were collected: epidemiological data, gender, circumstances of the accident, hemodynamic status at admission, injury associated with the calculation of ISS score (Injury Severity Score), patient management, length of stay, and possible morbidity and mortality [according to Clavien-Dindo classification (15)].

Abbreviated Injury Scale (AIS) classification was used to classify trauma severity from CT scans of hemodynamically stable patients or according to operative findings.

Biological data were also analyzed, such as hemoglobin (g/dl), arterial pH, and arterial lactates at admission and every 6 hours during the first 36 hours of management.

Hemodynamic stability was defined by heart rate between 50 and 90 beats per minute and systolic blood pressure above 90 mmHg. Permissive hypotension was not a goal at the time of the study.

Patients with extra-abdominal trauma requiring therapeutic procedure other than splenic were included in the splenic NOM group.

Selective distal splenic embolization was proposed in hemodynamically stable patients for whom the CT scan revealed active contrast leakage or splenic pseudoaneurysm. Proximal prophylactic angioembolization was not proposed in this study.

In the case of NOM, abdominopelvic CT-scan with contrast injection at arterial and portal time was routinely performed on the 5th day after the trauma. In the event of discomfort, deglobulization, appearance of abdominal pain, or other clinical signs considered worrying, an emergency CT scan could be performed earlier.

The primary endpoint was NOM failure with secondary splenectomy.
The failure of NOM was considered in the following cases:

- Multiple intrasplenic pseudoaneurysms (>3) at the control CT scan performed at day 5 after the trauma;
- Illness with hypotension, hemorrhagic shock or massive deglobulization suggestive of a secondary rupture of the spleen and imposing an emergent splenectomy;
- Infection of the previously embolized splenic parenchyma areas: abdominal pain in the left hypochondrium, fever, biological inflammatory syndrome, and signs of infection of the embolized territory with CT injected (intra-parenchymal air bubbles).

This latter situation represents the late failures of NOM, referred to as secondary splenectomy in our study.

The database is approved by the French committee for privacy statements (Commission Informatique et Liberté) and authorized by the University Hospital of Nice. The data is stored on a secure server at Nice University Hospital.

A statistical analysis was performed. Data extraction and descriptive statistics were performed using Stata→15.0 software (Stata Corp., College Station, USA).

Univariate analysis was performed looking for NOM failure factors and angioembolization using a t-test.

**Results**

A total of 280 patients were included in our study. Descriptive data are listed in Table 1. More than 70% of the patients were men, and most of the splenic lesions were severe (grades >3).
| Variable                                | N (%)          |
|-----------------------------------------|----------------|
| Gender                                  |                |
| -Female                                 | 77 (27.5)      |
| -Male                                   | 203 (72.5)     |
| Missing data                            | 0              |
| Road accident:                          |                |
| -Yes                                    | 197 (70.9)     |
| -No                                     | 81 (29.1)      |
| Missing data                            | 2              |
| Hemodynamic instability at admission:   |                |
| -Yes                                    | 96 (34.3)      |
| -No                                     | 184 (65.7)     |
| Missing data                            | 0              |
| Spleen lesion stage:                    |                |
| -None                                   | 3 (1.1)        |
| -1                                      | 46 (16.4)      |
| -2                                      | 52 (18.6)      |
| -3                                      | 71 (25.4)      |
| -4                                      | 85 (30.4)      |
| -5                                      | 23 (8.2)       |
| Missing data                            | 0              |
| Hepatic lesion stage:                   |                |
| -None                                   | 228 (81.4)     |
| -1                                      | 13 (4.6)       |
| -2                                      | 14 (5.0)       |
| -3                                      | 12 (4.3)       |
| -4                                      | 13 (4.6)       |
| -5                                      | 52 (18.6)      |
| Total                                   | 0              |
| Missing data                            |                |
| Lesion Type                        | Count   | Percentage |
|-----------------------------------|---------|------------|
| Thoracic lesion stage:            |         |            |
| - None                            | 104     | 37.1       |
| - Present                         | 176     | 62.9       |
| Missing data                      | 0       |            |
| Renal lesion:                     |         |            |
| - Yes                             | 61      | 22.7       |
| - No                              | 208     | 77.3       |
| Missing data                      | 11      |            |
| Pelvic lesion:                    |         |            |
| - Yes                             | 49      | 17.6       |
| - No                              | 230     | 82.4       |
| Missing data                      | 1       |            |
| Cerebral lesion:                  |         |            |
| - Yes                             | 50      | 18.8       |
| - No                              | 216     | 81.2       |
| Missing data                      | 14      |            |
| Active bleeding on CT scan:       |         |            |
| - Yes                             | 54      | 20.4       |
| - No                              | 211     | 79.6       |
| Missing data                      | 15      |            |
| Emergency splenectomy:            |         |            |
| - Yes                             | 83      | 29.6       |
| - No                              | 197     | 70.4       |
| Missing data                      | 0       |            |
| Delayed splenectomy:              |         |            |
| - Yes                             | 32      | 11.4       |
| - No                              | 248     | 88.6       |
| Missing data                      | 0       |            |
| Splenic embolization:             |         |            |
| - Yes                             | 88      | 31.4       |
| - No                              | 192     | 68.6       |
During the study, 83 splenectomies were performed urgently, i.e. 29% of patients and 88 angioembolizations i.e., 31% of all patients, with a success rate greater than 80%.

The mortality rate after emergency splenectomy was high, reaching 10%.

Table 2 shows the continuous variables. The median age was 39 years, and the Glasgow Coma Scale was 13 at admission. The ISS score was high, with a median of 28.9. There was no statistically significant difference with ISS score between successful NOM and failure of NOM.
Table 2

Major parameters of various groups of the study. Values are median values. Hb: hemoglobin in g/dl; lactates in mmol/l; H0 and H6: hemoglobin level at admission (H0) and 6 hours after admission (H6); AIS: Abbreviated Injury Scale; ISS: Injury Severity Score; LOS: length of stay (days); NOM: Non-operative management.

| Nb of patients | Age | Abdominal AIS | ISS | Hb H0 | Hb H6 | lactates H0 | lactates H6 | LOS |
|----------------|-----|---------------|-----|-------|-------|-------------|-------------|-----|
| Total          | 280 | 35            | 3   | 25    | 12.3  | 11.9        | 2.47        | 2.36| 10  |
| Emergent splenectomy | 83  | 34.5          | 4   | 38    | 10.9  | 11.9        | 3.155       | 2.84| 12  |
| NOM            | 197 | 35            | 3   | 20    | 13.1  | 12          | 1.72        | 1.75| 9   |
| NOM with angi-embolisation | 88  | 34            | 3   | 20    | 13.1  | 11.9        | 1.81        | 1.76| 9   |
| Success of angi-embolisation | 66  | 36            | 3   | 20    | 13.2  | 11.7        | 2.48        | 1.83| 12  |
| Delayed splenectomy | 32  | 40.5          | 4   | 27    | 13.6  | 11.25       | 3.115       | 0.92| 15  |

Hemoglobin levels were also monitored during the first 36 hours, ranging from an average of 12 g/dl at entry to 10.8 g/dl at 36 hours.

Some data were missing and could not be included in our study. This mainly concerns biological data, such as pH changes, lactate levels, and changes in hemoglobin. This can be explained by the variability of care depending on the patient’s condition and the blood test performed, which is considered repetitive for some.

Figure 1 shows the time when angioembolization was performed in the case of NOM. In 57% of cases, angioembolization was performed on the day of admission, which corresponds to patients presenting with active bleeding or aneurysm and in a stable hemodynamic state. This rate then decreases until we observe a second peak on the 5th day (11% of patients), corresponding to the completion of a control CT-scan.

It can be observed that 28% of splenectomies were performed on the day of the admission, followed by a decrease, with a subsequent increase at D5 and D8 (12.8% of splenectomies). Again, these peaks correspond to the control imaging with the presence of a significant lesion not accessible to angioembolization or deglobulization (Fig. 2).

Embolization was not found as a protective factor for performing secondary splenectomy. A total of 14.7% of the 136 patients (i.e. 20 patients) who did not have primary angioembolization required
secondary splenectomy, and 19.7% of the 61 patients (i.e. 12 patients) who had primary angioembolization required secondary splenectomy (p = 0.382).

There is no statistically significant difference between age and failure of nonoperative management; the median age of the successful embolization group was 44 years vs 37.5 years in the angioembolization failure group (p = 0.15).

The last analysis compared the decrease in hemoglobin between admission (H0) and 6 hours after admission (H6) from management between the angioembolization group and the angioembolization failure group. The hemoglobin differential between H0 and H6 appears to increase in patients with failed angioembolization (Table 3).

| Hemoglobin variation between admission and 6 hours after admission for patients with non-operative management and angio-embolization. |
|-----------------|-----------------|-----------------|
| Patients        | Hemoglobin variation | Standard deviation | CI 95% (p) |
| Successful NOM due to angio-embolization | 66 | 0.68 | 1.84 | 0.06–1.3 |
| NOM Failure despite angio-embolization | 12 | 1.44 | 1.17 | 0.59–2.28 (p = 0.064) |

The group of patients who had an angioembolization failure had a decrease in hemoglobin level of -1.44 g/dl between H0 and H6 vs. -0.68 g/dl for those who had a successful angioembolization (p = 0.064).

**Discussion**

NOM is the gold-standard treatment for hemodynamically stable trauma patients and is eligible for 80% of patients (5, 6). Performing a CT scan with different injection times and especially arterial allows a precise morphological assessment and guides the therapeutic strategy. CT enables searches for active bleeding or pseudoaneurysm resulting in angioembolization (5, 7–10). However, NOM can fail in multiple ways, leading to secondary splenectomy (5, 6, 11–14).

Many risk factors for NOM failure have been proposed, but none can accurately predict the failure of NOM.

Grade 3 or higher lesions are more at risk of NOM failure (according to AAST classification) (5, 6, 14, 16, 17). In AAST 4 and 5 splenic lesions, the failure rate of NOM is 54.6% (19).
Arterial blush on CT in the absence of angio-embolization is described as a risk factor of conservative treatment failure (5, 8, 18), as well as diffuse hemoperitoneum (14, 16).

A severe injury associated with a high ISS would be predictive of a higher rate of NOM failure. Different ISS values are reported in the literature as limiting to a NOM [e.g., 15 (9), 25, and others (14, 16, 17)]. Combining a splenic trauma with a brain injury can complicate the surveillance (19).

The importance of age in NOM has been debated. Age greater than 55 years would increase the risk of NOM failure, especially for high-grade lesions (5, 14, 20); age greater than 40 years could be a NOM failure factor, according to other authors (16). In our study, the median age was 39 years. We detected no statistically significant difference for age; nor did we detect a statistically significant difference according to AAST classification, ISS, or mechanism of injury.

Biological abnormalities, such as a decrease in hemoglobin, pH, or an increase in arterial lactates, might be factors for NOM failure. Alcohol consumption is common during trauma and might result in a falsely high value of lactate or prolong lactate clearance; it must, therefore, be taken into account when evaluating patients with a high blood alcohol level (21). The value of lactate and bicarbonates was identified as an important independent predictor of the polytrauma patient with acute alcohol and drug use (22, 23).

In our study, there is a difference in hemoglobin values at H0 and H6 between the groups having failed angioembolization and the successful group of angioembolization. This result, however, remains nuanced since it is, by little, not statistically significant (p = 0.064). This result can probably be explained because of the small number of patients. This result is nevertheless interesting since it is understood that biological deglobulization may be the first sign of true bleeding requiring secondary splenectomy.

In the literature, a decreased value of hemoglobin in the hours following the admission of trauma patients has never been described as NOM failure factors. On the other hand, the need for transfusion of red blood cells is a risk factor for NOM failure (16, 18).

In our study, the lack of data can represent a bias. Thus, the study of lactate levels could not be performed; this is explained by the disparity of care and evolution of patients. Indeed, lactate often dosed at H0 was not necessarily dosed in the aftercare. A study addressing the variation of lactatemia as a failure factor of NOM in splenic trauma might be interesting.

In this study, we could not demonstrate that angioembolization prevented secondary splenectomy. However, this is not comparable to the lack of benefit and is likely due to the lack of power of the study.

A multivariate analysis of the different predictors of NOM failure would also be useful. Unfortunately, we could not achieve this because of the amount of missing data. A multicenter prospective study would address these limitations and should now be conducted to confirm the role of deglobulization in the management of patients with splenic trauma.
Conclusion

Surgical emergencies are an important part of the activity of our hospitals. The trauma patient is a key issue because of the acute care required and the choice of the most appropriate treatment. NOM has become the standard for a hemodynamically stable patient, even though it has a significant failure rate. Deglobulization 6 hours after admission could represent a new risk factor for NOM failure. The need to highlight predictive factors of NOM failure in multicenter prospective trials is essential to achieve optimal patient care.

List Of Abbreviations

NOM: non-operative management
CHU: Centre Hospitalier Universitaire (French word for Teaching Hospital)
AAST: American Association for the Surgery of Trauma
AIS: Abbreviated Injury Scale
ISS: Injury Severity Score

Declarations

Consent for publication
Not applicable

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Ethics approval and consent to participate: this study was approved by the ethical committee of the hospital. The database is declared to French national database services (Commission Nationale Informatique et Liberté)

Availability of data and materials: the datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

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All authors have been actively involved in the conception of this manuscript and approved this version
Author Contribution statement:

- literature search has been done by Dr Tokoto, Dr Maubert and Dr Massalou
- study design and data collection have been done by Dr Massalou,
- data analysis has been done by Dr Douissard
- Data interpretation, writing and critical revision have been done by all the authors,

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References

1. Costa G, Tierno SM, Tomassini F, Venturini L, Frezza B, Cancrini G, et al. The epidemiology and clinical evaluation of abdominal trauma. An analysis of a multidisciplinary trauma registry. Ann Ital Chir. 2010 Apr;81(2):95–102.
2. Becker CD, Mentha G, Terrier F. Blunt abdominal trauma in adults: role of CT in the diagnosis and management of visceral injuries. Part 1: liver and spleen. Eur Radiol. 1998;8(4):553–62.
3. Watson GA, Hoffman MK, Peitzman AB. Nonoperative management of blunt splenic injury: what is new? Eur J Trauma Emerg Surg Off Publ Eur Trauma Soc. 2015 Jun;41(3):219–28.
4. Kozar RA, Crandall M, Shanmuganathan K, Zarzaur BL, Coburn M, Cribari C, et al. Organ injury scaling 2018 update: Spleen, liver, and kidney. J Trauma Acute Care Surg. 2018 Dec;85(6):1119–22.
5. Coccolini F, Montori G, Catena F, Kluger Y, Biffl W, Moore EE, et al. Splenic trauma: WSES classification and guidelines for adult and pediatric patients. World J Emerg Surg WJES. 2017;12:40.
6. Smith J, Armen S, Cook CH, Martin LC. Blunt splenic injuries: have we watched long enough? J Trauma. 2008 Mar;64(3):656–63; discussion 663–665.
7. Zarzaur BL, Dunn JA, Leiningter B, Lauereman M, Shanmuganathan K, Kaups K, et al. Natural history of splenic vascular abnormalities after blunt injury: A Western Trauma Association multicenter trial. J Trauma Acute Care Surg. 2017;83(6):999–1005.
8. Capecci LM, Jeremitsky E, Smith RS, Philp F. Trauma centers with higher rates of angiography have a lesser incidence of splenectomy in the management of blunt splenic injury. Surgery. 2015 Oct;158(4):1020–4. discussion 1024–1026.
9. Peitzman AB, Heil B, Rivera L, Federle MB, Harbrecht BG, Clancy KD, et al. Blunt splenic injury in adults: Multi-institutional Study of the Eastern Association for the Surgery of Trauma. J Trauma. 2000 Aug;49(2):177–87. discussion 187–189.
10. Dolejs SC, Savage SA, Hartwell JL, Zarzaur BL. Overall Splenectomy Rates Stable Despite Increasing Usage of Angiography in the Management of High-grade Blunt Splenic Injury. Ann Surg.
11. Bee TK, Croce MA, Miller PR, Pritchard FE, Fabian TC. Failures of splenic nonoperative management: is the glass half empty or half full? J Trauma. 2001 Feb;50(2):230–6.
12. Roy P, Mukherjee R, Parik M. Splenic trauma in the twenty-first century: changing trends in management. Ann R Coll Surg Engl. 2018 Aug;16:1–7.
13. Zarzaur BL, Rozycki GS. An update on nonoperative management of the spleen in adults. Trauma Surg Acute Care Open. 2017;2(1):e000075.
14. Bhangu A, Nepogodiev D, Lal N, Bowley DM. Meta-analysis of predictive factors and outcomes for failure of non-operative management of blunt splenic trauma. Injury. 2012 Sep;43(9):1337–46.
15. Dindo D, Demartines N, Clavien P-A. Classification of surgical complications: a new proposal with evaluation in a cohort of 6336 patients and results of a survey. Ann Surg. 2004 Aug;240(2):205–13.
16. Olthof DC, Joosse P, van der Vlies CH, de Haan RJ, Goslings JC. Prognostic factors for failure of nonoperative management in adults with blunt splenic injury: a systematic review. J Trauma Acute Care Surg. 2013 Feb;74(2):546–57.
17. McIntyre LK, Schiff M, Jurkovich GJ. Failure of nonoperative management of splenic injuries: causes and consequences. Arch Surg Chic Ill 1960. 2005 Jun;140(6):563–8. discussion 568–569.
18. Scarborough JE, Ingraham AM, Liepert AE, Jung HS, O’Rourke AP, Agarwal SK. Nonoperative Management Is as Effective as Immediate Splenectomy for Adult Patients with High-Grade Blunt Splenic Injury. J Am Coll Surg. 2016;223(2):249–58.
19. Alabbasi T, Nathens AB, Tien H. Blunt splenic injury and severe brain injury: a decision analysis and implications for care. Can J Surg J Can Chir. 2015 Jun;58(3 Suppl 3):108–17.
20. Ong AW, Eilertson KE, Reilly EF, Geng TA, Madbak F, McNicholas A, et al. Nonoperative management of splenic injuries: significance of age. J Surg Res. 2016 Mar;201(1):134–40.
21. Dezman ZDW, Comer AC, Narayan M, Scalea TM, Hirshon JM, Smith GS. Alcohol consumption decreases lactate clearance in acutely injured patients. Injury. 2016 Sep;47(9):1908–12.
22. Dunne JR, Tracy JK, Scalea TM, Napolitano LM. Lactate and base deficit in trauma: does alcohol or drug use impair their predictive accuracy? J Trauma. 2005 May;58(5):959–66.
23. Javali RH, Ravindra P, Patil A, Srinivasarangan M, Mundada H, Adarsh SB, et al. A Clinical Study on the Initial Assessment of Arterial Lactate and Base Deficit as Predictors of Outcome in Trauma Patients. Indian J Crit Care Med Peer-Rev Off Publ Indian Soc Crit Care Med. 2017 Nov;21(11):719–25.

Figures
Figure 1

day of angio-embolization during hospital length of stay
**Figure 1**

day of angio-embolization during hospital length of stay
Figure 2

day of splenectomy during hospital length of stay
Figure 2

day of splenectomy during hospital length of stay