European Journal of Trauma and Emergency Surgery (2020) 46:407–412
https://doi.org/10.1007/s00068-018-1032-6

ORIGINAL ARTICLE

The need for red blood cell transfusions in the emergency department as a risk factor for failure of non-operative management of splenic trauma: a multicenter prospective study

Paola Fugazzola[1] · Lucia Morganti[2] · Federico Coccolini[1] · Stefano Magnone[1] · Giulia Montori[1] · Marco Ceresoli[1] · Matteo Tomasoni[1] · Dario Piazzalunga[1] · Stefano Maccatrozzo[1] · Niccolò Allievi[1] · Savino Occhionorelli[2] · Luca Ansaloni[1]

Received: 3 April 2018 / Accepted: 8 October 2018 / Published online: 15 October 2018

Abstract

Introduction The majority of patients with splenic trauma undergo non-operative management (NOM); around 15% of these cases fail NOM and require surgery. The aim of the current study is to assess whether the hemodynamic status of the patient represents a risk factor for failure of NOM (fNOM) and if this may be considered a relevant factor in the decision-making process, especially in Centers where AE (angioembolization), intensive monitoring and 24-h-operating room are not available. Furthermore, the presence of additional risk factors for fNOM was investigated.

Materials and methods This is a multicentre prospective observational study, including patients presenting with blunt splenic trauma older than 17 years, managed between 2014 and 2016 in two Italian trauma centres (ASST Papa Giovanni XXIII in Bergamo and Sant'Anna University Hospital in Ferrara—Italy). The risk factors for fNOM were analyzed with univariate and multivariate analyses.

Results In total, 124 patients were included in the study. In univariate analysis, the risk factors for fNOM were AAST grade > 3 (fNOM 37.5% vs 9.1%, $p = 0.024$), and the need of red blood cell (RBC) transfusion in the emergency department (ED) (fNOM 42.9% vs 8.9%, $p = 0.011$). Multivariate analysis showed that the only significant risk factor for fNOM was the need for RBC transfusion in the ED ($p = 0.049$).

Conclusions The current study confirms the contraindication to NOM in case of hemodynamically instability in case of splenic trauma, as indicated by the most recent guidelines; attention should be paid to patients with transient hemodynamic stability, including patients who require transfusion of RBC in the ED. These patients could benefit from AE; in centers where AE, intensive monitoring and an 24-h-operating room are not available, this particular subgroup of patients should probably be treated with operative management.

Keywords Spleen trauma · Non-operative management · Trauma care · Non-operative management failure

Introduction

The non-operative management (NOM) is the gold-standard for the management of hemodynamically stable patients with splenic trauma without signs of peritonitis or associated injuries requiring a laparotomy [1]. NOM presents several advantages when compared to operative management (OM): a reduction in complications, mortality, costs, need of red blood cells (RBC) transfusions and, above all, the preservation of the immunologic function of the spleen [2–4]. As a matter of fact, the incidence of overwhelming post-splenectomy infections (OPSI) is 0.5–2% and the mortality rate ranges from 30 to 70%; the majority of lethal events occur within the first 24 h from the traumatic insult. Only prompt diagnosis and immediate treatment can reduce mortality [5, 6]. According to the current guidelines injury grade, haemoperitoneum entity, presence of contrast blush (CB) at CT scan, Glasgow Come Scale, age, presence of associated

✉ Paola Fugazzola
paola.fugazzola@gmail.com

[1] General and Emergency Surgery Department, ASST Papa Giovanni XXIII, Piazza OMS 1, Bergamo, Italy

[2] General Surgery Department, Sant'Anna University Hospital, Ferrara, Italy

lesions and need of RBC transfusions, should not be considered contraindications to NOM [3, 7–9]. Nevertheless, a proportion as high as 15% of NOM patients fails the initial conservative treatment [10–22]. Various predictors for failure of NOM (fNOM) have been proposed and analyzed; many retrospective and prospective observational studies, carried out on large samples [10–31], and a meta-analysis [21] described that risk factors for fNOM were not related to the hemodynamic status of the patients. The aim of the current study is to verify, through a prospective multicentre study, if the hemodynamic status of the patient can be considered as a singular decisive factor which may guide the management decision or whether other significant risk factors for fNOM should drive the therapeutic decision, especially in centers where angioembolization (AE), intensive monitoring and 24-h-operating room are not available.

Methods

This is a multicentre prospective observational study on patients aged older than 17 years with blunt splenic trauma, managed between 2014 and 2016 in two Italian Trauma Centers (TC) (ASST Papa Giovanni XXIII in Bergamo and Sant'Anna University Hospital in Ferrara).

Characteristics of patients and features of the traumatic events, as well as condition of the patients at arrival at the emergency department (ED) [systolic blood pressure (SBP), heart rate (HR), shock index (SI)] were registered. Blood gas test [pH, base excess (BE), lactates (Lac)], blood tests (CBC, platelet count, INR, fibrinogen) and E-FAST (extended-FAST) results were collected at arrival. The number of blood transfusions in ED was registered. We defined "hemodynamic instability" if a patient presented with a SBP lower than 90 mmHg or a SI higher than 1 or a BE lower than − 5 after resuscitation in the ED and without vasoactive drugs.

For patients who underwent a CT scan, the AAST classification for the splenic injury, the number of abdominal quadrants with haemoperitoneum and the presence of vascular lesions [contrast blush (CB), pseudoaneurysm (PSA), artero-venous fistula (AVF)] were reported. For patients who underwent urgent surgical intervention, intraoperative findings were registered. For each patient, the Injury Severity Score (ISS) was calculated and the presence of associated abdominal, pelvic or cerebral lesions was reported. Data regarding treatment and outcomes were analyzed: the initial management (observation, distal AE, proximal AE, splenectomy, intraperitoneal packing, hemostasis of the splenic injury, surgical intervention for other organ lesions), the time between the arrival and the first urgent intervention, and the need of further intervention during the index hospital stay (AE or splenectomy).

We defined OM when the patient underwent an urgent surgical intervention and if during surgery splenectomy or hemostatic splenic technique (e.g., splenic packing or splenorrhaphy) were performed. AE was performed in hemodynamically stable patients with evidence of splenic blush at CT scan and in patients with IV and V AAST injury grade. Patients with active bleeding at CT scan and I–II–III AAST injury grade were treated with distal AE, while patients with IV and V AAST injury grade were treated with proximal AE. These patients were included in the NOM group. Failure of NOM was defined by the necessity of performing splenectomy after primary NOM.

Statistical analysis

The risk factors for fNOM were analyzed with univariate and multivariate analyses. Statistical analyses were performed with Student's *t* test for continuous variables with normal distribution and with the Mann–Whitney test for non-normal distribution variables. Parametric variables were compared with Chi-square test. Significant *p* value was considered if lower than 0.05. The Software SPSS Version 20.0 (IBM Corp. Released 2011. IBM SPSS Statistics for Windows, Version 20.0. Armonk, NY: IBM Corp) was used for the statistical analysis.

Results

The study includes 124 patients aged older than 17 years with blunt splenic trauma: 66 of them were managed in a level I TC (ASST Papa Giovanni in Bergamo) and 58 in a level II TC (Sant'Anna University Hospital in Ferrara). The two groups were homogeneous in terms of epidemiologic features, trauma dynamics, ISS, splenic injury grade. Patients characteristics are reported in Table 1. 66 patients (53.2%) were treated with NOM and 58 (46.03%) with OM. Within the NOM group, 22 underwent AE (17.8% of total patients and 33.3% of NOM patients) at the arrival or during the hospital stay. Among them, 8 (36.4%) underwent proximal, 11 (50.0%) distal and 2 (9.1%) combined AE. Three patients died within the first week from trauma for causes not related to splenic trauma (the cause of death being cardiogenic shock for two patients and brain injuries for another patient); these patients were excluded from subsequent analysis. The rate of fNOM was 12.7%.

Considering the eight patients who failed NOM, two of them were operated on for hemodynamic instability (one patient with AAST splenic injury grade 4 and one patient with AAST grade 3 with CB—none of them previously treated with AE); three patients required surgical intervention for the persistence of CB or for the increase of haemoperitoneum at control CT scan, without hemodynamic

Table 1 Patient characteristics

Characteristics	*N* = 124 Mean ± DS Median (range)
Age (years)	50.23 ± 18.36 48.68 (17.00–91.00)
M/F	91/33 (73.4%/26.6%)
ISS	27.93 ± 13.02 27.00 (5.00–75.00)
Trauma mechanism of injury	
Invested pedestrian	11 (8.9%)
Car	38 (30.6%)
Motorbike	39 (31.5%)
Bike	5 (4.0%)
Precipitation	17 (13.7%)
Others	14 (11.3%)
Splenic injury grade	
AAST 1	3 (3.2%)
AAST 2	48 (38.7%)
AAST 3	34 (27.4%)
AAST 4	30 (24.2%)
AAST 5	5 (4.0%)
N.A.	3 (2.4%)

M/F male/female, *ISS* Injury Severity Score, *HR* heart rate, *NA* not available, *AAST* American Association for the Surgery of Trauma

compromission (two patients had undergone distal AE at the arrival for AAST grade 4 and 3 lesions, both with CB and a patient with AAST lesion grade 2); finally, three patients were operated for the development of perisplenic abscesses (one with AAST lesion grade 4, CB and PSA was managed with proximal AE, one had undergone AE for an AAST lesion grade 3, without CB, and one patient with AAST grade 2 lesion).

Failure of NOM occurred within 24 h in two cases (25.0%), between 24 and 48 h in one case (12.5%) and after 48 h in five cases (62.5%). The most tardive failure was observed after 21 days for development of an abscess.

At univariate analysis, the only risk factors for fNOM were the AAST splenic injury grade and, in particular, an AAST grade > 3 versus AAST ≤ 3 (fNOM 37.5% vs 9.1%, $p = 0.024$), and the need of RBC transfusion in the ED (fNOM 42.9% vs 8.9%, $p = 0.011$) (Table 2). Multivariate analysis revealed that the only significant risk factor for fNOM was the need of RBC transfusion in ED (Table 3) ($p = 0.049$).

Discussion

In the present study, the rate of fNOM was 12.7%, which is consistent with the rate ranging between 4 and 15% reported in the literature [10–22]. A vast heterogeneity in the reasons for failure of NOM is retrieved in previous studies [12, 17]. Among our patients, the causes of failure were the onset of hemodynamic instability (25%), the development of abdominal abscesses (37.5%), the persistence of CT CB after AE (25.0%) and the increase of haemoperitoneum (12.5%).

As a varying proportion of patients fail NOM, various predictors for NOM failure have been proposed and analyzed. Failure of NOM is increased significantly by AAST grade of splenic injury in the totality of previous studies, whether AE was included into [11, 12, 25, 26, 32] or excluded from [13, 17, 18, 24, 27, 32] the protocol for the management of splenic injuries. Within patients with AAST injury grade exceeding 3, the rate of fNOM reaches 54.6% [27]. In the present study, the fNOM rate for AAST splenic injury grade > 3 was 37.5% (vs 9% in AAST injury grades ≤ 3, $p = 0.024$), but this was not a risk factor at multivariate analysis. Taking into account that 62.5% of patients with AAST splenic injury grade > 3 was successfully treated with NOM, a high AAST grade should not univocally contraindicate NOM. Nevertheless, patients with AAST splenic injury 4 or 5 represent a group requiring prophylactic AE and intensive monitoring to prevent a rapid deterioration of the hemodynamic status.

The validity of age higher than 55 years as a risk factor of fNOM is still debated [11–15, 19, 22, 23, 27, 30, 32]. Advanced age seems to reduce elasticity and contraction capacity of the splenic parenchyma, decreasing significantly the rate of successful NOM from more than 90–80% [12]. According to the present data, age higher than 55 years did not represent a risk factor for fNOM, even if fNOM rate among patients younger than 55 years was 7.9% vs 17.4% in older patients (p = not significant). In other studies, age > 55 years was a risk factor for fNOM only in case of high AAST injury grade. In the current study, the rate of fNOM in patients older than 55 years with AAST splenic injury grade > 3 was 25.0%. Furthermore, failure of NOM in older patients is associated with a mortality rate 2.5 times higher, as compared with patients younger than 55 years, with an increased length of stay [22]. The higher mortality in older patients is confirmed by our results. Some authors [12] consider OM a prudent approach for these patients, since the failure rate is 30–40%. However, 75% of patients older than 55 years with high injury grade were successfully treated with NOM in the current study. These characteristics delineate a further category of patients who are likely to benefit from prophylactic AE and intensive monitoring.

Table 2 Univariate analysis for fNOM risk factors

Variables Mean ± DS Median (range)	Successful NOM	Failed NOM	*p* value
Age < 55 years	92.1%	7.9%	n.s.
Age > 55 years	82.6%	17.4%	
Age (years)	49.05 ± 18.01	57.49 ± 17.72	n.s.
	48.00 (18.00–87.00)	55.85 (36.00–91.00)	
No anticoagulant/antiplatelet therapy	84.2%	15.8%	n.s.
Anticoagulant/antiplatelet therapy	100.0%	0.0%	
HR (bpm)	86.48 ± 18.75	79.63 ± 18.03	n.s.
	85.00 (48.00–133.00)	73.50 (62.00–120.00)	
SBP (mmHg)	121.22 ± 20.91	107.88 ± 17.04	n.s.
	120.00 (70.00–170.00)	112.50 (80.00–130.00)	
AAST 1–2	95.0%	5.0%	n.s.
AAST 3	80.0%	20.0%	
AAST 4	62.5%	37.5%	
AAST ≤ 3	90.9%	9.1%	0.024
AAST > 3	62.5%	37.5%	
ISS	21.95 ± 10.12	36.00 ± 20.49	n.s.
	22.00 (5.00–48.00)	29.00 (11.00–75.00)	
Lac	2.93 ± 1.99	2.49 ± 1.73	n.s.
	2.23 (0.80–9.24)	2.49 (1.27–3.72)	
BE (mmol/L)	− 3.09 ± 3.99	− 5.80 ± 2.97	n.s.
	− 2.8 (− 14.50–2.10)	− 5.8 (− 7.9 to − 3.70)	
pH	7.32 ± 0.08	7.35 ± 0.02	n.s.
	7.34 (7.13–7.43)	7.34 (7.33–7.36)	
Hb (g/dL)	13.30 ± 2.37	13.40 ± 2.34	n.s.
	13.90 (6.00–16.80)	13.45 (10.10–16.50)	
Brain injuries	87.3%	12.7%	n.s.
No brain injuries	87.5%	12.5%	
AE	78.5%	21.4%	n.s.
No AE	89.8%	10.2%	
Associated abdominal lesions	87.0%	13.0%	n.s.
No associated abdominal lesions	87.5%	12.5%	
CB	78.6%	21.4%	n.s.
No CB	89.6%	10.4%	
PSA	50.0%	50.0%	n.s.
No PSA	88.1%	11.9%	
Number of quadrants with haemoperitoneum			
> 3	50.0%	50.0%	n.s.
< 3	89.5%	10.5%	
Fibrinogen (mg/dL)	217.61 ± 57.71	189.50 ± 3.53	n.s.
	210.50 (156.00–401.00)	189.50 (187.00–192.00)	
INR (s)	1.11 ± 0.15	1.10 ± 0.08	n.s.
	1.13 (0.66–1.38)	1.10 (1.04–1.16)	
PLT/mm^3	219.33 ± 46.57	218.00 ± 106.07	n.s.
	220.00 (137.00–315.00)	218.00 (143.00–293.00)	
Positive ECO fast	85.0%	15.0%	n.s.
Negative Eco fast	87.1%	12.9%	
Time between the arrival at ED and AE (min)	361.67 ± 566.01	193.12 ± 63.11	n.s.
	169.80 (55.00–1920.00)	169.00 (117.00–280.20)	
I level trauma center	91.4%	8.6%	n.s.
II level trauma center	82.1%	17.9%	
RBC transfusion in ED	57.1%	42.9%	0.011
No RBC transfusion in ED	91.1%	8.9%	

Table 2 (continued)

ISS Injury Severity Score, *HR* heart rate, *n.a.* not available, *SBP* systolic blood pressure, *ED* emergency department, *BE* base excess, *Lac* lactates, *Hb* hemoglobin, *RBC* red blood cells, *AAST* American Association for the Surgery of Trauma, *WSES* World Society of Emeregency Surgery, *PLT* platelet

Table 3 Multivariate analysis

Variables	*p* value	OR
RBC transfusion in the ED	0.049	6.44
AAST > 3	n.s.	–

RBC red blood cells, *AAST* American Association for the Surgery of Trauma, *ED* emergency department

Unlike other studies [3, 14, 15, 17–19, 24–27, 29, 31, 32], the present study does not confirm the presence of associated abdominal injuries, high ISS, low hemoglobin value at admission, entity of haemoperitoneum and the presence of CB at CT scan as risk factors for fNOM.

In the current study, the rate of fNOM for patients treated in level I TC was 8.6% vs 17.9% in level II TC, although this difference did not reach a significant difference; the literature reports that the level of the accepting TC is not associated with significant differences in the outcomes of NOM for splenic trauma [20, 27].

Finally, in concordance with previous studies [17, 26], the necessity of RBC transfusion in ED is the only risk factor for fNOM in multivariate analysis (42.9% vs 8.9%, $p = 0.011$). This is likely to represent the result of transient hemodynamic stability, in patients who sustain a partial or superficial response to fluid resuscitation. Although, as previously reported [12, 13, 17–19], the vital parameters at arrival did not influence the risk of fNOM, the need for RBC transfusion to reach hemodynamically stability could identify a group of patients with borderline hemodynamics, within the broader group of patients frequently labeled as 'responder' to fluid resuscitation.

The previous concept could play a role in the selection of patient amenable to NOM, AE or OM after splenic trauma; a great heterogeneity exists in the definition of 'hemodynamic instability', whereas the need for transfusion of blood products could represent a univocal definition in this.

A potential limitation of the current study resides in the fact that patients did not sustain isolated spleen injury; therefore, the associated lesions may have influenced the management and the outcomes. Furthermore, we reported heterogeneity in reasons for fNOM; only two patients failed for real hemodynamic instability.

Conclusions

The results of the present study confirm the current guidelines, which contraindicate NOM only in case of hemodynamic instability. Particular attention should be paid to patients with transient hemodynamic stability, including patients requiring RBC transfusion in ED. These patients could benefit from AE, and in Centers where AE, intensive monitoring and a 24/7 operating room are not available, they are probably best treated with OM.

Vice versa, high splenic injury grade, the presence of associated brain and abdominal lesions, the entity of haemoperitoneum, age, high ISS, the presence of CB, PSA and AVF, adverse blood test and blood gas tests results are not absolute contraindications to attempt NOM. However, an age older than 55 years identifies a category of patients who need intensive monitoring.

Acknowledgements The authors would like to thank and express gratitude to 'Associazione Marco Piazzalunga' (the friends of the Trauma Register of Papa Giovanni XXIII Hospital of Bergamo, Italy) for the support given to the trauma register and to the present analysis. This study has been possible mainly due to their financial funding to the register maintenance and to the intellectual support to this particular register inquiry.

Compliance with ethical standards

Conflict of interest There are no conflicts of interests.

Ethical approval Data were retrospectively extracted from ASST Papa Giovanni XXIII Trauma Register.

References

1. Marconi FT, Escocia AD, Montego BTP, Cruvinel NJ, Messias ZT, Pereira FG. Nonoperative management of splenic injury grade IV is safe using rigid protocol. Rev Col Bras Cir. 2013;40(4):323–8.
2. Hafiz S, Desale S, Sava J. The impact of solid organ injury management on the US health care system. J Trauma Acute Care Surg. 2014;77:2.
3. Stassen NA, Bhullar I, Cheng JD, et al. Eastern Association for the Surgery of Trauma. Selective nonoperative management of blunt splenic injury: an Eastern Association for the Surgery of Trauma practice management guideline. J Trauma Acute Care Surg. 2012;73(5 Suppl 4):S294–300.
4. Gaspar B, Negoi I, et al. Selective nonoperative management of abdominal injuries in polytrauma patients: a protocol

only for experienced trauma centers. Medic - J Clin Med. 2014;9(2):168–72.
5. Leone G, Pizzigallo E. Bacterial infections following splenectomy for malignant and nonmalignant hematologic diseases. Mediterr J Hematol Infect Dis. 2015;7(1):e2015057.
6. Shatz DV. MD, FACS. Vaccination practices among North American trauma surgeons in splenectomy for trauma. J Trauma. 2002;53:950–6.
7. Coccolini F, Montori G, Catena F, Kluger Y, Biffl W, Moore EE, Reva V, Bing C, Bala M, Fugazzola P, Bahouth H, Marzi I, Velmahos G, Ivatury R, Soreide K, Horer T, Ten Broek R, Pereira BM, Fraga GP, Inaba K, Kashuk J, Parry N, Masiakos PT, Mylonas KS, Kirkpatrick A, Abu-Zidan F, Gomes CA, Benatti SV, Naidoo N, Salvetti F, Maccatrozzo S, Agnoletti V, Gamberini E, Solaini L, Costanzo A, Celotti A, Tomasoni M, Khokha V, Arvieux C, Napolitano L, Handolin L, Pisano M, Magnone S, Spain DA, de Moya M, Davis KA, De Angelis N, Leppaniemi A, Ferrada P, Latifi R, Navarro DC, Otomo Y, Coimbra R, Maier RV, Moore F, Rizoli S, Sakakushev B, Galante JM, Chiara O, Cimbanassi S, Mefire AC, Weber D, Ceresoli M, Peitzman AB, Wehlie L, Sartelli M, Di Saverio S, Ansaloni L. Splenic trauma: WSES classification and guidelines for adult and pediatric patients. World J Emerg Surg. 2017;12:40. https://doi.org/10.1186/s13017-017-0151-4.
8. Moore FA, Davis JW, Moore EE Jr, Cocanour CS, West MA, McIntyre RC Jr. Western Trauma Association (WTA) critical decisions in trauma: management of adult blunt splenic trauma. J Trauma. 2008;65(5):1007–11.
9. Rowell SE, Biffl WL, Brasel K, Moore EE, Albrecht RA, DeMoya M, Namias N, Schreiber MA, Cohen MJ, Shatz DV, Karmy-Jones R, Moore FA. Western Trauma Association Critical Decisions in Trauma: management of adult blunt splenictrauma-2016 updates. J Trauma Acute Care Surg. 2017;82(4):787–93. https://doi.org/10.1097/TA.0000000000001323.
10. Peitzman AB, Harbrecht BG, Rivera L, Heil B, Workgroup. Eastern Association for the surgery of trauma multiinstitutional trials. Failure of observation of blunt splenic injury in adults: variability in practice and adverse consequences. J Am Coll Surg. 2005;201(2):179–87.
11. Renzulli P, Gross T, Schnüriger B, Schoepfer AM, Inderbitzin D, Exadaktylos AK, Hoppe H, Candinas D. Management of blunt injuries to the spleen. Br J Surg. 2010;97(11):1696–703.
12. Bee TK, Croce M, Miller PR, Pritchard FE, Fabian TC. Failures of splenic nonoperative management: is the glass half empty or half full? J Trauma. 2001;50:230–6.
13. Nix JA, Costanza M, Daley BJ, Powell MA, Enderson BL. Outcome of the current management of splenic injuries. J Trauma. 2001;50(5):835–42.
14. McIntyre LK, Schiff M, Jurkovich G. Failure of nonoperative management of splenic injuries: causes and consequences. J Arch Surg. 2005;140(6):563–8. **(discussion 568–9)**.
15. Ong AW, Eilertson KE, Reilly EF, Geng TA, Madbak F, McNicholas A, Fernandez FB. Nonoperative management of splenic injuries: significance of age.. J Surg Res. 2016;201(1):134–40. https://doi.org/10.1016/j.jss.2015.10.014.
16. Bhullar IS, Frykberg ER, Siragusa D, Chesire D, Paul J, Tepas JJ 3rd, Kerwin AJ. Age does not affect outcomes of nonoperative management of blunt splenic trauma. J Am Coll Surg. 2012;214(6):958–64.
17. Peitzman AB, Heil B, Rivera L, Federle MB, Harbrecht BG, Clancy KD, Croce M, Enderson BL, Morris JA, Shatz D, Meredith JW, Ochoa JB, Fakhry SM, Cushman JG, Minei JP, McCarthy M, Luchette FA, Townsend R, Tinkoff G, Block EF, Ross S, Frykberg ER, Bell RM. Blunt splenic injury in adults: multi-institutional study of the Eastern Association for the surgery of trauma. J Trauma. 2000;49(2):177–87.
18. Smith J, Armen S, Cook CH, Martin LC. Blunt splenic injuries: have we watched long enough? J Trauma. 2008;64:656–5.
19. Malhotra AK, Latifi R, Fabian TC, Ivatury RR, Dhage S, Bee TK, Miller PR, Croce MA, Yelon JA. Multiplicity of solid organ injury: influence on management and outcomes after blunt abdominal trauma. J Trauma. 2003;54(5):925–9.
20. Harbrecht BG, Zenati MS, Alarcon LH, Ochoa JB, Puyana JC, Schuchert VD, Peitzman AB. Is outcome after blunt splenic injury in adults better in high-volume trauma centers? Am Surg. 2005;71(11):942–8. **(discussion 948–9)**.
21. Cocanour CS1, Moore FA, Ware DN, Marvin RG, Duke JH. Age should not be a consideration for nonoperative management of blunt splenic injury. See J Trauma. 2000;48(4):606–10. **(discussion 610–2)**.
22. Harbrecht BG, Peitzman AB, Rivera L, et al. Contribution of age and gender to outcome of blunt splenic injury in adults: multicenter study of the eastern association for the surgery of trauma. J Trauma. 2001;51(5):887–95.
23. Siriratsivawong K, Zenati M, Watson GA, Harbrecht BG. Nonoperative management of blunt splenic trauma in the elderly: does age play a role? Am Surg. 2007;73(6):585–9. **(discussion 590)**.
24. Velmahos GC, Zacharias N, Emhoff TA, et al. Management of the most severely injured spleen: a multicenter study of the Research Consortium of New England Centers for Trauma (ReCONECT). Arch Surg. 2010;145(5):456–60.
25. Velmahos GC, Chan LS, Kamel E, Murray JA, Yassa N, Kahaku D, Berne TV, Demetriades D. Nonoperative management of splenic injuries: have we gone too far? Arch Surg. 2000;135(6):674–9. **(discussion 679–81)**.
26. Jeremitsky E, Kao A, Carlton C, Rodriguez A, Ong A. Does splenic embolization and grade of splenic injury impact nonoperative management in patients sustaining blunt splenic trauma? Am Surg. 2011;77(2):215–20.
27. Watson GA, Rosengart MR, Zenati MS, Tsung A, Forsythe RM, Peitzman AB, Harbrecht B. Nonoperative management of severe blunt splenic injury: are we getting better? G. J Trauma. 2006;61(5):1113–8. **(discussion 1118–9)**.
28. Krause KR, Howells GA, Bair HA, Glover JL, Madrazo BL, Wasvary HJ, Bendick PJ. Nonoperative management of blunt splenic injury in adults 55 years and older: a 20-year experience. Am Surg. 2000;66(7):636–40.
29. Sartorelli KH, Frumiento C, Rogers FB, Osler TM. Nonoperative management of hepatic, splenic, and renal injuries in adults with multiple injuries. J Trauma. 2000;49(1):56–61. **(discussion 61–2)**.
30. Albrecht RM, Schermer CR, Morris A. Nonoperative management of blunt splenic injuries: factors influencing success in age> 55 years. Am Surg. 2002;68(3):227–30. **(discussion 230–1)**.
31. Schurr MJ1, Fabian TC, Gavant M, Croce MA, Kudsk KA, Minard G, Woodman G, Pritchard FE. Management of blunt splenic trauma: computed tomographic contrast blush predicts failure of nonoperative management. J Trauma. 1995;39(3):507–12. **(discussion 512–3)**.
32. 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;43(9):1337–46.