Annals of Academy of Romanian Scientists
Online edition *Series: Medical Sciences*
ISSN 2285-4150 *Volume 2, Number 1-2/2011*

Review Article

NONOPERATIVE MANAGEMENT OF BLUNT INJURIES OF THE SPLEEN

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Abstract

Nonoperative management of blunt splenic injuries has become the standard of care for hemodynamically stable patients. Currently, nonoperative management is attempted in 60% to 80% of patients with spleen injuries and is successful in 85% to 94%. The addition of SAE for hemorrhage control and treatment of intraparenchymal vascular injuries (pseudoaneurysm, arteriovenous fistula) has raised the splenic salvage rates to 80-98%. We present the indications, contractions and dilemmas of this type of treatment and our results. NOM has revolutionized the care of blunt splenic trauma patients.

Keywords: *spleen, blunt trauma, nonoperative management*

Rezumat

Tratamentul nonoperator în traumatismele splenice contuzive reprezintă actual modalitatea optimă terapeutică la pacienții stabili hemodinamic. Actual, acest tip de tratament este aplicat la 60-80% din pacienți, rezultatele favorabile fiind cuprinse între 85-94% din cazuri. Utilizarea angioembolizării terapeutice pentru controlul hemoragiei active şi a leziunilor vasculare intraparenchimatoase (pseudoanevrismul intrasplenic, fistula arterio-venoasă intrasplenică) a crescut procentul salvării splenice la 80-98%. În articol sunt prezentate indicațiile, controversele şi dilemele tratamentului nonoperator în trauma splenică privite prin prisma experienței noastre. Tratamentul nonoperator a schimbat o dogmă chirurgicală în trauma contuzivă splenică, marcând o nouă etapă în traumatologie.

Cuvinte-cheie: *splina, contuzie abdominală, tratament nonoperator*

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"In Englysche it is named a man's Splene. A spongious substance lienge under the ribbes on the left side, and it doth make a man to bee mery and to laughe, although melancholy resteth in the splene if there be impedimentes in it. If any man be splenitike let him use mery company, be joconde, and not to study upon any supernatural thynges."

[**Andrew Boorde;** The Breviarie of Helthe, 1547- cit. by 1]

The spleen is the most injured organ in blunt abdominal trauma.

The spleen's journey from expendable to valuable was one of the longest afforded any human organ or organ system [2].

In summary the milestones in the therapy of injured spleen are presented below.

Billroth suggested over a 100 years ago that the injured spleen has the ability to heal itself [3]. He submited this theory following the post-mortem findings in a 43 year-old woman, who fell from height during work and died 5 days later from brain and abdominal injuries. The autopsy revealed a splenic injury without an obvious sign of recent bleeding. Therefore, Billroth wrote: "from the appearance of the rent, and the small quantity of blood effused, we concluded that this injury might have healed up completely" [cit. by 3].

But, this important observation received little attention probably because the surgical community was not ready for the nonoperative management of splenic injury [3] and the treatment in these cases was limited to splenectomy.

In 1919, Morris and Bullock explained the detrimental effects of asplenia, especially the increased susceptibility to infection. These authors expressed a precise and prophetic warning *"…that the human body deprived of its'spleen would show…increased susceptibility to infection and some of the fatalities…attributed to infection…may be due to splenectomy."* [cit. by 3]

O'Donnell was the first to report post-splenectomy infection in a child in 1929.

In 1940 Wanborough (Sick Children's Hospital of Toronto) initiated the nonoperative therapy for suspected splenic injury [4].

The dogma of splenectomy regardless of the extent of injury to the spleen persisted until the risk of overwhelming postsplenectomy infection (OPSI) was described by King and Schumacker in 1952. Although the risk of OPSI in the splenectomized trauma patient is very low (0.5% of all splenectomies in trauma patients and in over 20% of elective splenectomies for haematologic disorders) [5] the mortality remains high. OPSI is most frequent during the first 2 years of asplenia but there is a permanent risk of infection with a mortality of over 80%.

Till few decades ago the management of splenic injury was represented by total splenectomy, because most surgeons considered conservative therapy dangerous and even fatal.

More, the role of nonoperative management of blunt splenic splenic injuries in adults has been the focus of considerable controversy over the last twenty years because

this mode of therapy previously thought to be unacceptable. In 1984 Malangoni et al. [6] concluded that *"observation management in adults with splenic injury is hazardous… and may be counterproductive in efforts to salvage the spleen"*.

The management of splenic trauma has evolved with time, from splenectomy towards splenic preservation and NOM over the last decades.

The haematological and immunological changes after splenectomy have been the subject of intensive research in recent years. As a consequence there has been a clear trend towards splenic salvage.

Actually, NOM is considered to be the treatment of choice for haemodynamically stable patients with blunt splenic trauma, although surgery continues to be the standard for haemodynamically unstable patients.

Splenectomised patients showed in the postoperative follow-up a significantly increased infection rate (40%) when compared to patients with splenic preservation (10%) or nonoperative treatment (11%) even when they where matched in respect of multiple trauma using the Injury Severity Score [7].

Nonoperative management of select blunt injuries in children was first described by Upadhyaya and Simpson in 1968. They observed *"…frequently, severe blood loss in children is not evident after splenic injury… It is interesting to note that, in the majority of children in this series, the bleeding from the spleen had stopped by the time laparotomy was performed"* [cit. 8].

The majority of data supporting the safety and efficacy of nonoperative approach are derived from University Trauma Programs with a Pediatric Center.

The obvious attraction of this concept of therapy is that it achieves splenic salvage and avoids unnecessary surgery [9].

Critics of this approach stress the following points [9]:

- differences in splenic architecture between children and adults;
- postsplenectomy sepsis occurs less commonly in the adult;
- risks associated with blood transfusions.

So, the management of splenic injury changed gradually toward conservative treatment.

Knowing all these factors set the trend in splenectomy-conservative therapy debate (non-operatieve treatment, conservative surgery, spleen autotransplant); it is currently considered that traumatic splenic injury is no longer an absolute indication for splenic surgery, thus a proper reviewing of indications for emergency surgery in traumatic hemoperitoneum is needed.

When it comes to visceral injuries following abdominal trauma, there is nothing as radical as the non-operative treatment of hepatic and splenic injuries [10]. The treatment for blunt abdominal trauma has significantly changed thanks to new diagnostic methods and the accurate assessment of organ damage.

In order for non-operative treatment of splenic injuries to be the standard goal of therapy in hemodinamically stable patients, it is necessary to have an accurate knowledge of pacient selection criteria for non-operative treatment, as well as a precise assessment of the factors precluding conservative therapy. This becomes tangible due to diagnostic and therapeutic angiography addition.

However, employing non-operative treatment for splenic injuries in adults was iniatially a challenge for surgeons for several reasons: the post-splenectomy sepsis is less frequent and less severe compared to children; structural and vascular splenic changes according to age and possibly the type of force inducing the lesion make a spontaneous hemostatis unlikely; the risk of overlooked associated injuries; the ensuing possibility of delayed rupture of the spleen (DRS), splenosis or post-traumatic cyst [11].

Other explications, although not scientifically founded, include a much thinner and somewhat less elastic splenic capsule in adults [Morgenstern, Gross cit. 12], lesion disposition in relation to splenic vasculature (much more favorable when the lesion is parallel with the blood vessels), associated rib fractures. These discrepancies are explained by an increased severity of adult trauma which usually associates extra and intra-abdominal injuries requiring surgical intervention [1997, Powell cit. 13].

The traditionally criteria for NOM are [14, 15]:

- hemodynamic stability/ readily stabilizable;
- lack of rebound and guarding;
- blood transfusions ≤ 4 units;
- no altered level of consciousness;
- age younger than 55 years;
- documentation of splenic injury by imaging techniques.

The only absolute indication for emergency laparotomy is hemodynamic instability. [14, 15].

Complex/severe splenic injuries, age, pre-existent splenic diseases, number of units of trasfused blood, brain injuries are no longer considered absolute contraindications for NOM [16, 17, Gaunt, Avanoglou-cit. 14, 18, 19, 20].

"NOM for blunt splenic injuries replaces splenorrhaphy which was the usual method for preserving the spleen" [16]; Garber [21] is the author of a multicentric retrospective study, made in Ontario (Canada) which validates that NOM is the preferred therapeutic method (in 69% of patients), followed by splenectomy (28%) and splenorrhaphy (4%) in non-trauma centers and 65%, 33% and 2% respectively in trauma centers. The incidence of NOM has increased from 59% (1991) to 75% (1994) and that of splenectomy has decreased from 35% (1991) to 24% (1994). The incidence of splenorrhaphy has significantly dropped from 6% to 1%.

Even 2 units of transfused blood during the first 48 hrs (in order to maintain a HGB level above 8 g/dl) is compatible with a successful NOM [3, 5].

According to Longo, Uranüs and Sartorelli [5, 22, 23] predictive parameters for a successful NOM include:

- hemodinamycally stable/ readily stabilizable;
- blood transfusions < 4 units;
- age ≤ 55 years;
- early resolution of splenic abnormalities obvious on imagistic investigations;
- no lack of consciousness/ no brain injuries;
- no associated intra or retroperitoneal injuries (upon abdominal CT scan) that would require surgical intervention;
- no rebound or guarding;
- complete recovery of bowel movements.

Knudson [cit. 17] considers that the hemoperitoneum secondary to spleen/ liver injuries is absorbed after the 5th day from the initial insult. If free intraperitoneal blood is still present after day 5 upon CT scan there is the possibility of overlooked injuries or rebleeding.

Associated extra-abdominal injuries

Blunt aortic injuries accompany hepatic and splenic lesions in 15-20% of cases [Fabian, Hunt cit. 24]; Santaniello's study [24] states that 33% of the patients with blunt aortic injury have associated simultaneous hepatic/splenic lesions. Recent NOM protocols for splenic injuries debunk the "removal of spleen from the equation"myth. Santaniello's study shows that minor splenic injuries (grade I-II) associated with aortic lesions pose a minimum/no risk for anticogulation therapy. In this article's editorial Kenneth Mattox disagrees upon aknowledging these findings when dealing with aortic injury associated with major splenic lesions.

Sartorelli [23] considers that the outcome of NOM in multiple parenchymal trauma patients is not different from that of NOM in unique organ involvement. Furthermore, NOM in patients with associated brain injuries to hepatic/ splenic lesions is safe [Archer cit. 23, 25]. Garber [21] observed that chest injuries account for most of the associated lesions (77%), followed by head injuries (59%).

Age > 55 years

An age over 55 years was considered a criterion for an unsuccessfull NOM (Godley had a rate of success of 9% when employing NOM in elderly patients) [cit. 23]. Why? Elderly patients have diminishing biological reserves, structural alterations concordant with age make a spontaneous hemostasis unlikely, increased splenic frailty. In an attempt to decipher these statements, Barone [20] quotes 2 articles written by Morgenstern and published between 1983 and 1979. Morgenstern and Uyeda (1983) assert that *"there is an age factor in hemostasis of the spleen"* because young patients have *"functional smooth muscle and elastic within the capsule, septae and splenic vasculature"*. The older patients have anatomic changes that *"limit contraction and retraction of damaged vessels within the injured parenchyma"*.

In 1979 Morgenstern and Shapiro suggest that splenorrhaphy should be contraindicated in elderly patients. In 1964 Gross observes the structural distinction between the splenic capsule in young adults and elderly patients, stating that "after the age of 60 years the splenic capsule is thickening". Perhaps Gross's studies should be reviewed and set as a standard protocol for NOM in elderly patients. [Barone-20]. Sartorelli [23] reports favorable results for NOM in 83.3% of all patients > 55 years old, similar to those conveyed by Barone [83% - 20], Myers [26], Brasel [71% - 15] and Cocanour [18]. Furthermore, Clancy [27] declares that the percentage of conserved spleens in patients over 65 years of age is similar to that of younger patients (40 patients over 65 years of age have been treated successfully by NOM). It's not the age but the grade of splenic injury that increases the risk of failure for NOM [28]. The use of BOAST (***b****edside* ***o****rgan* ***a****ssessment with* ***s****onography after* ***t****rauma*) as well as permanent and careful monitoring of these patients ensures the success of favorable outcome with NOM [29].

Older adults had significantly higher mortality, but this was not a result of their splenic injury-therefore, age should not be a criteria for NOM of blunt splenic injury [30]. Careful selection of patients > 55 years old must be made to minimize the morbidity and mortality from failed attempts.

The level of consciousness

In the past patients with altered mental status were not treated conservatively because of overlooked intra-abdominal injuries that might require laparotomy. However, Archer's [31] and Keller's [25] juvenile studies did not warrant the existence of undiagnosed complications in children. Rozycki's study [29] corroborates Archer's findings, including for patients with a GCS ≤ 8, stating that: *"NOM is not only perfectly feasible in patients with severe brain damage, but efficient and safe"*. According to Pal [32] the CT scans respresent a very effective diagnostic method for hemodinamically stable patients with altered mental status and equivocal abdominal exam, having a sensitivity of 97.7%, a specificity of 98.5% and an overall accuracy of 99.4%. Authors consider that DPL is not necessary in this group of patients.

Archer's results (NOM in patients with altered mental status is safe in a strictly monitored environment) are confirmed by the rate of success of NOM in patients with GCS<13 (93%). Likewise, Cocanour [15] considers that brain injuries are not a contraindication for NOM.

Age > 55 years or abnormal neurologic status should not preclude NOM in hemodynamically normal patients.

The severity of splenic injury

The severity of splenic injury - it appears that NOM is effective in splenic injuries with an average lesional AAST score of 3 [33].

Failure of NOM increase significantly by grade of splenic injury [34]: grade I (4.8%), grade II (9.5%), grade III (19.6%), grade IV (33.3%) and grade V (75%). The grade of splenic injury correlated with the quantity of associated hemoperitoneum and both findings quantifying the magnitude of injury to the spleen [34]. Hiatt and Federico [cit. 17] considered the exact opposite to be true. There are a few studies (Nallathambi, Malangoni, Pickhardt, Brick, Mahon, Taylor, cit. 35, cit. 36] signaling the fact that splenic injuries have an impredictable progress and proving there is no obvious correlation between the anatomical lesional severity and clinical outcome. Velmahos debated these results based on his conclusions: AIS is a flawed system of staging intra-abdominal visceral injuries; a useful prediction model should be simple.

Pachter and Guth [16] did not find any correlation between the degree of hemoperitoneum and Injury Severity Scoring > 15 as predictors of failure of NOM.

Pathological spleen

Guth and Patcher [16] consider that pre-existent splenic diseases do not represent an absolute contraindication for NOM (HIV related splenomegaly). This approach is based on the theoretical presumption that these immunocompromised patients would be more prone to postsplenectomy infection than the general population. The splenomegaly

induced by tropical diseases (especially malaria) requires a conservative approach in the event of a trauma (NOM or splenorrhaphy).

92% of all the patients with cirrhosis had an unsuccessfull NOM with 55% of fatal cases after surgery (splenectomy as a consequence of failed NOM) [37]. NOM failure is explained by altered spontaneous hemostasis associating with pre-existent portal hypertension syndrome (which leads to increased hydrostatic pressure within the parenchyma); there is also a clotting factor deficit in decompensated hepatic cirrhosis with a subsequent coagulopathy. Therefore, the mortality rate is directly correlated with increased PT values (prothrombin time), high lesion score and low serum albumin levels. Coagulopathy is a risk factor for a trauma patient with cirrhosis (Wahlstrom 2000; Tinkoff 1990; Morris 1990 - cit. 37]. It is imperative to operate to stop the bleeding if the patient has a pre-existent coagulopathy worsened by the ongoing hemorrhage. When preexistent coagulopathy is the one responsible for the bleeding following trauma, then the bleeding disorder should be tackled first and then decide whether or not surgical intervention is still required. Fang considers that cirrhosis is a contraindication for NOM.

Patients with a prolonged PT should not be approached by NOM in case of splenic trauma even if cirrhosis is not present [38].

Religion

Religion represents an important factor when treating splenic injury. Zieg and co. [39] presented a case of a type A hemophiliac patient, a Jehova witness, with splenic trauma and favorable NOM outcome that was treated with recombinant factor VIII. There are 10 cases in english literature of hemophiliac patients and splenic trauma out of wich 3 had an excellent outcome for NOM.

The only absolute contraindication of NOM is represented by hemodinamic instability.

The benefits of NOM [26, 40] are:

- low morbidity and mortality;
- avoidance of a non-therapeutic laparotomy;
- no immediate/late complications that usually accompany a laparotomy;
- minimal blood transfusions
- decreased hospital stay (when other injuries prolonging the hospital stay coexist);
- maintened immunological function and prevention of OPSI.

Potential drawbacks of NOM:

- overlooked injuries;

  Allen and co. [cit. 41] observed that 2.3% of NOM patients have had other associated injuries that were initially overlooked and required surgery later on (delayed diagnosis for over 6 hrs in 20% on patients with blunt abdominal trauma), but with many intra-abdominal complications. In Sartorelli's study overlooked hollow viscus injuries totalized 0.8% of all cases [23].
- Impredictible time period for a second potential bleeding;
- Low splenic conservation rate following surgery for unsuccessfull NOM;

- A surgeon on call 24/7 and permanent clinical monitoring;
- Debates about the time period necessary for a complete recovery.

Delayed surgical exploration could be increase the risk of hemorrhagic shock, major blood disorders, excessive blood transfusions and potential death. In 90% of cases the failure of NOM is evident in the first 50 hrs from the initial insult. Velmahos [42] identified 4 independent risk factors for an unsuccessfull NOM: splenic injury severity score, hemoperitoneum of over 300 ml, positive FAST, necessary blood transfusions. Statistically speaking, when all 4 factors are present, NOM will fail in 96% of cases.

Complications following NOM [15, 22, 34, 43, 44] occurred in 40% of cases and consist of:

- persistent bleeding/ rebleeding;

  This is obvious when an altered status is present along with occurrence/re-occurrence of internal bleeding signs, an increased number of transfused blood in order to maintain a normal systolic blood pressure, a worsening CT/US image and a significant drop in hematocrit and hemoglobin. In most cases persistent bleeding is the culprit; delayed bleeding occurs in delayed rupture of the spleen (a real lesion- intrasplenic pseudoaneurysm) or in the case of a ruptured exapanding subcapsular hematoma (water is moving through osmosis leading to increasing size of the hematoma).

More than 90% of the NOM failures of are secondary to renewed bleeding

- post-traumatic splenic pseudocyst;
- splenic abscess-rare; blood-spread infection or vecinity contamination are the main causes; the treatment consists of percutaneous drainage and in case of failure, splenectomy;
- Splenosis

  means autotransplant of the splenic tissue in ectopic places, secondary to the trauma of the spleen's capsule. It is quite common. The most common location is the peritoneal cavity. It is thought that the incidence of the abdominal splenosis is around 50% of posttraumatic splenectomy [Schiff-cit. Aktekin - 42]. In 1978 Pearson suggests that the splenic autotransplant done after total splenectomy is a way of protection against sepsis and named this clinical entity "*the born-again spleen*" [46]. Posttraumatic ectopic splenic tissue could have a role in the persistence of immunologic spleen function and so it is not advised to remove it if there are no symptoms. Although the role of splenosis in immunological protection (especially OPSI) is controversed, taking into consideration the risk of this complication, the removal of these nodules is advised in two cases: bleeding and intestinal occlusion.
- Postembolization asplenia (functional splenic failure);
- Pulmonary complications;
- Deep venous thrombosis;
- Blood transfusion-induced pathology (HIV, hepatitis C).

Schreiber [cit. 47] reckons that HIV infection risk, that of human leukemic virus with T cells and of hepatitis B and C from 1 unit of transfused blood is 1 in 34000 cases, 88% of them being hepatitis B and C.

Failure of NOM

Occurs most frequently in the following circumstances:

- hemodinamic instability (systolic BP < 90 mmHg despite adequate resuscitation);
- age > 55 years;
- > 4 units of transfused blood to maintain a hemoglobin level over > 10 g/dl;
- Persistent leucocytosis;
- The onset or aggravating sings of peritoneal irritation (suggesting further bleeding/ other overlooked injuries);
- Worsening imagistical signs of splenic injury (repetead US exams)-post-traumatic splenic defect;
- Increasing volume of hemoperitoneum;
- Intra-abdominal compartment syndrome (intravesical presure > 20 cm H_2O).

According to Velmahos [14] the minimum time period necessary for a patient to be included in NOM protocol is 3 hrs.

The time interval between onset and reported NOM failure ranged between 6 and 94 hrs [22] with subsequent prolonged hospital stay (an average of 11.2 days). 67% of patients with unsuccessfull NOM had contrast blush (hyperdense, well delineated, intraparenchymal contrast collection) [48]. Therefore, he concluded that the risk for failing NOT when contrast blush is present is 24-fold increased.

NOM failure can be explained by complications and by the constant pressure physicians find themselves to discharge patients as soon as possible; some failures are evident after discharge which means it is very important to identify any problem before that. Velmahos [14] identified 2 independent risk factors for failing NOM: splenic injury ≥ 3 and more than 1 unit of transfused blood. When both factors are present NOM failing rate is as high as 97%; when none of these factors is present then NOM failing rate is 3%.

Unsuccessfull NOM rate ranges between 2% and 31% [16, 20, 34, 25, 33, 36, 37, 40, 48, 49, 50, 51, 52].

Gavant's and Federle's retrospetive studies [cit. 40] showed that contrast extravasation/ post-traumatic vascular injuries (contrast blush) visible on CT scans/ spiral CT scans with IV contrast are usually associated with an increased rate for unsuccessfull NOM (these lesions may also be present in low grade injuries I, II).

The presence of extravasation of contrast material ("*contrast blush*") on the initial or subsequent CT-scan represents a strong predictor (maybe the most significant factor) of failure of nonoperative management; Davis report failure of this approach in 13% of cases [19]. The vascular blush represents a well-circumscribed intraparenchymal contrast collection hyperdense with respect to the surrounding parenchyma *[Figures no.1, 2, 3, 4]*.

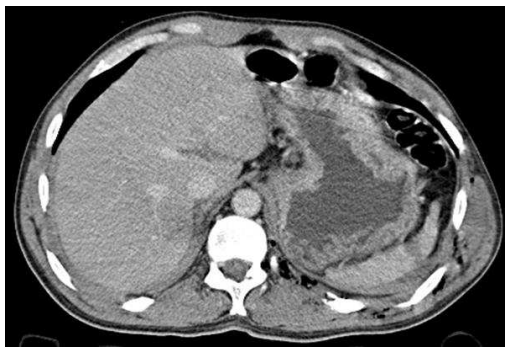

Figure no.1.

Grade II splenic injury - CT-scan image.

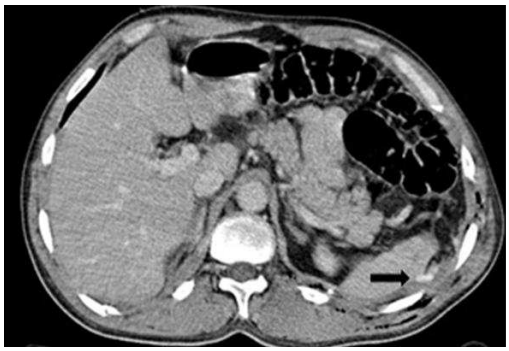

Figure no.2. *The same case - CT scan showing contrast extravasation (black arrow) (grade II splenic fracture); perisplenic hemoperitoneum.*

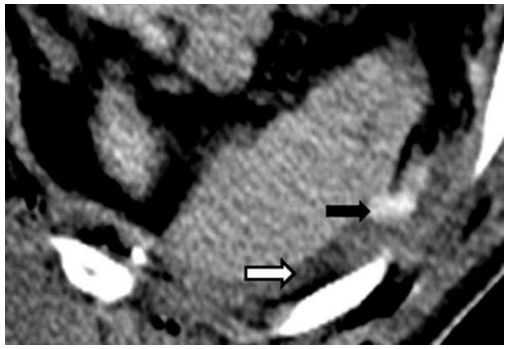

Figure no.3. *Enlarged image: CT scan showing contrast extravasation (black arrow) (grade II splenic fracture); perisplenic hemoperitoneum (white arow).*

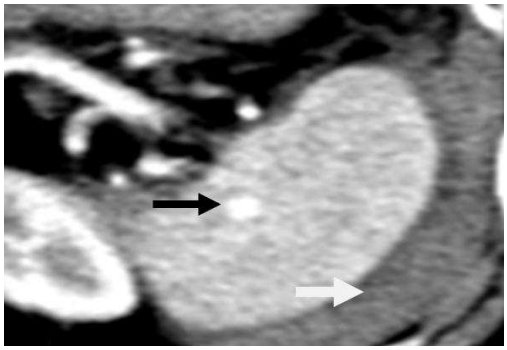

Figure no.4. *CT scan showing contrast blush in grade II splenic injury which was later confirmed by surgery (black arrow); perisplenic hemoperitoneum (white arrow).*

The natural history of the contrast blush is, in most cases the evolution to rupture and sometimes to selftamponade. The presence of contrast-blush/perisplenic contrast extravasation are signs of active hemorrhage and mandates an aggressive approach: angiography or exploratory laparotomy to ensure the hemostasis.

Successfull NOM

In adults it ranges between 61.5% and 97% [33, 34, 53, 54].

Pachter [53] reports their results: 53% in grade II injuries; 29% in grade III; 4% in grade IV; 1% in grade V lesions. The high percentage (97%) reported by Sclafani [54] is subsequent to the use of angiography and proximal angioembolization. NOT is successful in 97% of cases in children no matter the injury score [Velanovich cit. 55].

Hospital stay

It varies between 3 to 7 days when no other injuries are present to elicit a prolonged stay [11, 14, 15, 21, 22].

Splenic angiography (diagnostic and therapeutic)

Recent NOM protocols for splenic trauma include angiography (diagnostic and therapeutic) as an efficient alternative [56]. Angiography can have a diagnostic purpose as well as therapeutic (vascular embolization and hemostasis). The therapeutic interventional radiology techniques have now become essential in the management of splenic injuries in the modern trauma care.

The first angiographic embolization used Gelfoam (Katzen, 1976) and temporary ballon occlusion (Wholey, 1977) and were performed for hemostatic purposes prior splenectomy. Angiographic intervention as an adjunct to the management of splenic injury was initially described by Salvatore Sclafani in 1981 [57].

Vascular lesions visible on angiography are [56, 58, 59]:

- contrast extravasation inside or outside of spleen;
- a frank cutoff of a major vessel;
- intraparenchymal arterio-venous fistula;
- intrasplenic pseudoaneurysm;
- vascular compression by subcapsular hematoma;
- variable degree of devascularization and irregularities in contrast filling (that includes Seurat spleen: small, spot-like, delineated/diffuse contrast collections).

Indications for splenic angiography [58, 59, 60]:

- grade 3, 4, 5 splenic injuries;
- vascular lesions visible on initial CT scan (contrast extravasation, pseudoaneurysm, arteriovenous fistula, vessel truncation);
- active bleeding upon CT scan or contrast blush in a hemodinamically stable patient (upon repeated CT scans);
- large hemoperitoneum;
- inexplicable decrease of hemoglobin level when no other lesions are present.

When angiography is performed in all hemodynamically normal patients with splenic injury, only 30% require embolization [59].

Splenic angioembolization (SAE) can be:

- distal (supraselective) [see *Figures 5, 6*];
- proximal: splenic artery occlusion by coil embolization 2 cm beyond the origin of the dorsal pancreatic artery and proximal to the first pancreatica magna artery [54]. It produces hemostatis by decreasing the blood flow and intrasplenic pressure by occluding the main arterial conduit to the spleen. The viability of the remaining spleen is ensured by colateral blood flow (left gastric artery, short gastric arteries, omental arteries, pancreatic arteries, gastroepiploic collaterals). Sclafani [54] considers that the preservation of immunological functions is compatible with this procedure and even splenorrhaphy is facilited in case of surgical intervention.
- Combined: Diagnostic and therapeutic (embolization) angiography is performed after CT scans showed intrasplenic vascular damage. Embolization is carried out only if there is angiographic confirmation of the lesion [61].

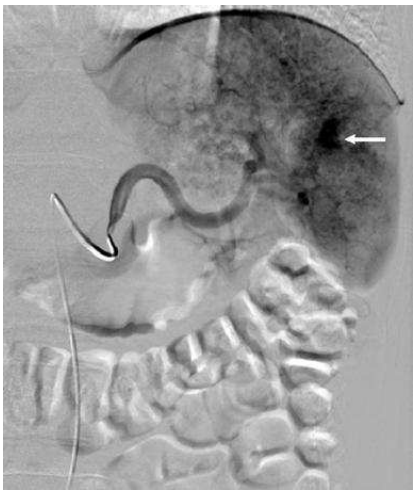

Figure no.5. *Splenic angiography-intraparenchymal contrast extravazation-active bleeding (white arrow).*

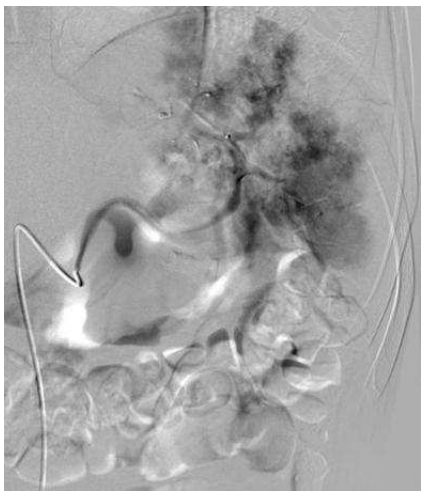

Figure no.6. *Final aspect after splenic angioembolization-bleeding stopped.*

Second-look angiography is useful in recurrent bleeding and after an initially negative angiography (10%) [62-Haan]. Haan employed prefentially distal SAE for small grade lesions and combined SAE for severe injuries (however with almost no statistical difference). Haan [62] also believes that "delayed vascular emergencies" (term first introduced by the Memphis group) are basically delayed diagnoses that become evident when performing angiography for severe splenic injuries (grade 3, 4, 5). The Memphis group (Davis, Fabian, Croce) proved that initial CT and angiographic scans can skip vascular injury due to arterial spasm at the moment of the examination but can later become clinically detectable; spiral CT scans identified 80% of all vascular lesions that were initially unnoticeable (spiral CT is used as a screening test for angiography). The

only statistically significant failure risk for NOM is the arterio-venous fistula which is treated not only by proximal SAE but by a more direct approach-distal SAE (combined technique) [63].

The conclusions inferred by Haan's study [64] are:

- Proximal SAE is a much more useful therapeutic method than distal embolization (because it decreases the splenic perfusion pressure); the exception is an arterio-venous fistula;
- The immunological consequences of proximal embolization are still unclear and require further investigation;
- The use of SAE decreases by 20% the failure rate of NOM in grade 4 and 5 injuries;
- SAE proved to be superior to surgical intervention when dealing with blunt splenic trauma in multiple trauma patients with brain injury.
- SAE is a useful and efficient method for NOM but it is necessary in only 7% of cases [61].

SAE indications [63, 64]:

- proximal SAE: it is indicated in hilar lesions;
  - > 3 distinct peripheral vascular lesions;
  - The injury affect more than 50% of the splenic parenchyma.
- Selective SAE: limited vascular injuries. It is proficient because it allows proper hemostasis and adequate perfusion to remaining organ.
- Combined SAE: for multiple vascular injuries (high injury scores).

It is recommended to perform multiple CT scans after SAE in order to monitor the vascular damage, pseudoaneurysm formation, size of infarctioned area and existence of localized infection (splenic abcess).

SAE represents an elegant alternative and is now part of all NOM protocols in trauma centers.

The benefits of SAE will need to be balanced against the potential for hemodynamic deterioration during angiography, delayed hemorrhage control, associated missed intraabdominal injuries and the failure rate of SAE [65].

Haan [66] has abandoned the use of selective embolization in favor of main coil splenic embolization because the last method is faster, less expensive and technically easier. He observed a new entity: persistent or new splenic pseudoaneurysm after main coil embolization and his conclusion was: these patients have similar splenic salvage rates to the overall cohort without additional therapies [66]. SAE added to NOM for severe splenic injuries (grades 3 to 5) and in all cases where signs of ongoing bleeding were present regardless of injury grade increase the NOM success rate and the splenic salvage rate [67]. Sabe [68] identified the patients at high risk for NOM failure if they had vascular blush or pseudoaneurysm on CT, grade 3 injury with large hemoperitoneum, or grade 4 injury; if we add the presence of intraparenchymal arteriovenous fistula [Haan's criteria-64] we have the indications for emergency splenic angioembolization. The importance of vascular splenic injuries was evidenced by Marmery which proposed a new CT classification of splenic trauma based on these findings (in this classification active bleeding, arteriovenous fistula, pseudoaneurysm and vascular injury are the main

parameters used to determine the grade of splenic injury) [69]. In a prospective clinical study the Japanese authors [70] have evaluated the use of splenic angioembolization in hemodynamically unstable patients in whom there is a transient response to initial fluid resuscitation. The results of their study support the routine use of SAE in this category of patients.

It is important to know if angiography and embolization improve salvage of an immunologically competent and normally functioning mass of splenic tissue or simply avoids an operation [Harbrecht-65]. Nakae [71] considers that splenic preservation treatment did not show discernible advantage over splenectomy in immunologic indices including IgM and 14 serotypes of anti-Streptococcus pneumonia antibodies but their studies did not delineate results specifically for SAE patients. Tominaga's studies [72] suggest that the immunologic profile of SAE patients is similar to controls and this supports the safe use of splenic angioembolization in managing the traumatically injured spleen. Their results are similar to those reported by Walusimbi [cit. 72] and demonstrates that the immunological profile of SAE patients is similar to blunt trauma patients without splenic injury [72]. But, Shih [73] says: SAE dysregulates the NF-kB (nuclear factor translocations) system and aggravates the cytokine hyporesponse upon endotoxin stimulation of peripheral blood mononuclear cells in patients with blunt spleen injury. This fact is very important because this procedure may induce alternations of immune response and cumulate in infectious vulnerability in injured patients [73].

The use of splenic angioembolization for traumatic injuries was initiated at our institution in 2009. The first successful splenic angioembolization in trauma in Romania was performed at Emergency Hospital Bucharest and published in "Chirurgia" in 2010 [74].

CT findings after SAE [75]

Areas of splenic infarction appear after SAE that have certain characteristics:

- Infarction appeared in 63% of cases after proximal SAE, but only in 20% of cases the area extended over more than 50% of splenic parenchyma. These areas are usually small in size, multiple, situated at the splenic border and heal completely.
- Infarction areas after distal SAE occur in 100% of cases with only 9% of cases affecting over 50% of the splenic parenchyma. They is usually a unique, large area immediately beneath the embolized blood vessel and heal completely in most cases.

Statistically speaking distal SAE triggered more splenic infarctions than proximal SAE.

- Combined SAE trigger splenic infarction in 71% of cases; in 20% of them more than 50% of splenic parenchyma was affected.

When air bubbles are visible within the splenic parenchyma it is necessary to rule out a splenic abcess. Likewise, the presence of air-fluid level in a subcapsular collection suggests the development of a splenic abcess (which can be drained percutaneously).

Discharge recommendations [23, 76]:

- **Grade I-II lesions:**
  - Avoidance of strenuous activities and sport (jogging, lifting > 20 pounds, 1 pound = 453.6 g);
  - Avoidance of construction work for 6-8 weeks;
  - Light activities (light work around the house, desk work, light aerobic activity) 2 weeks after the initial injury;
  - CT scan/US will be performed only if the clinical exam requires it.
- **Grade ≥ III lesions**:
  - Minimial activity for 1 week;
  - Light activity 4-8 weeks;
  - Avoidance of strenuous activities and sport for 10-12 weeks.
- **Grade IV, V lesions**:
  - Avoidance of strenuous activities and sport for > 3 months.
  - Mandatory CT scans or US.

Monitoring blunt splenic injuries patients for 3 to 5 days after injury should allow in-hospital identification of 95% to 97% of patients who will fail NOM [70].

The postdischarge evolution of NOM of blunt splenic injury has not adequately elucidated. Zarzaur [78] considers that 1.4% of persons discharged home after nonoperative management of blunt splenic injuries presents the risk at re-admisssion for splenectomy in a period of 180 days, but the majority of splenectomies occur within 8 days. According to Peitzmann [34], 0.76% of patients who were discharged after nonoperative management return to a trauma center for total splenectomy. In Crawford [79] and Savage [80] studies 0.16% respectively 0.34% of patients discharged alive with a NOM for blunt splenic injuries required splenectomy after discharge. The combination of increased use of NOM and decreasing hospital stays may increase the possibility of this evolution [78, 79].

The overall risk of in-hospital death in the patients re-admitted secondary to splenectomy was 3.7% [78].

Before discharge it is necessary an explicit patient education and close follow-up; in all cases of abdominal problems appeared after discharge the patients needs a complete medical examination in hospital.

The evolution of healing of the traumatic spleen

It was initially considered that patients undergoing NOM or splenorrhaphy require bed rest for 1 week and avoidance of physical activity for 6 months; the experimental studies performed on dogs and pigs by Dulchavsky and co. showed that splenic scarring consists of an extensive capsular fibrosis and fibrous reaction at splenorrhaphy site and paralleling intrasplenic septs [81]. Kluger [82] performed an experimental study on young rats and adult rats in order to clear up the cellular mechanism of splenic scarring after trauma and the influence of patient's age on the success of NOM.

He observed that the local bleeding resorbed in the first 48 hrs in youg rats and in 7 days in adult rats; he also noticed that splenic paranchyma regeneration occured in 14 days in young rats whilst in adult ones the process was incomplete by the 21st day. Peak

accumulation of myofibroblasts at the laceration site took place during day 2 in young rats and during day 4 in adult ones. Splenic lacerations heal through a regeneration process and not by colagen scarring.

Accelerated splenic healing that grants a successfull NOM in children and young adults is explained by this early accumulation of myofibroblasts at the lesion site. Benya [83] conducted a study that included children with grade I-II splenic injuries with complete resolution on CT scans after 4 months from the initial injury; for severe lesions the healing time is extended to over 6 months for grade III and over 11 months for grade IV injuries.

The author considers a complete resolution on CT scan when there are no abnormal areas in or around the spleen or when there is a mild residual deformation of the splenic outline (without the obvious presence of a hematoma/ perisplenic fluid collection).

Mean time-to-healing analysis revealed that patients with mild spleen injuries had more rapid healing (12.5 ± 19.0 days) compared with patients with severe spleen injuries (37.2 ± 27.5 days). The majority of those who will completely heal their injuries will do so at 2 to 2½ months, regardless of severity at presentation [80].

10% of patients discharged with a nonhealed spleen worsened over time and 2% required late intervention [80].

Our studies [84] have shown the evolutionary point of complete resorption of hemoperitoneum and healing of splenic laceration by capsular scarring with the development of chronic subcapsular hematoma disappears evolutionary time or turned it into a posttraumatic pseudocyst *[figures no. 7, 8]*.

Patients with AAST grade III or IV splenic injuries receive follow-up abdominal CT scans 4-6 weeks postinjury.

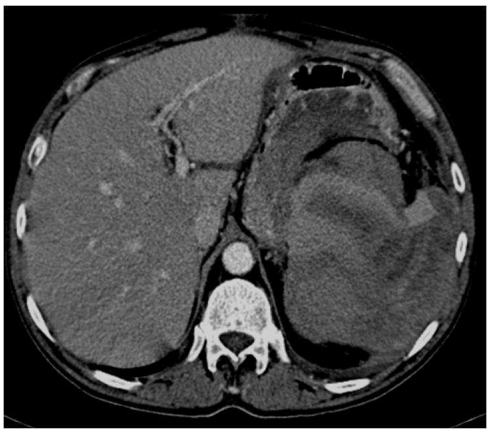

Figure no.7. *Grade IV splenic rupture – nonoperative management; CT image on admission.*

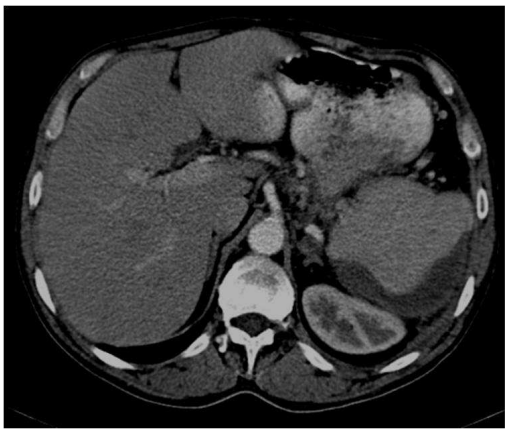

Figure no.8. *The same case- CT image after 1 month.*

Conclusions

Actually, NOM represents the "gold standard" in the treatment of blunt splenic injuries. The NOM has replaced the splenic salvage procedures in haemodynamically stable patients in most trauma centers.

NOM represents an effective and safe alternative for selected patients with splenic trauma. When dealing with splenic trauma NOM is the rule and not the exception [85] with its success relying upon adequate clinical assessment.

Criteria for immediate operation are:

- haemodynamic instability on presentation, despite fluid resuscitation with crystalloid solution or reccurence of instability after initial stabilization;
- peritoneal signs on physical examination and
- identification by CT scan of other concomitant intra-abdominal injuries that required surgical intervention.

Haemodynamic instability of a patient is defined like systolic arterial blood pressure lower than 90 mmHg on admission unresponsive to fluid resuscitation with fast infusion of 2 litres of crystalloid solution or systolic arterial blood pressure lower than 90 mmHg after initial stabilization**.**

TNO has demonstrated the efficacy and safety; "*nonoperative management is here to stay"* [86-Hoyt]; it is a flexible concept that can be modified depending on the clinical course of patients presenting potential alternatives including splenic angioembolization and conservative surgery.

"The spleen is just like a woman: mysterious, apparently capricious and hiding unsuspected resources. The life without it is possible. But, it's not the same life." [87].

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