Splenic abscess after splenic blunt injury angioembolization

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INTRODUCTION: Splenic Angioembolization (SAE), during Nonoperative Management (NOM) of Blunt Splenic Injury (BSI), is an effective therapy for hemodynamically stable patients with grade III, IV, and V OIS splenic injuries. We report a case of a patient with a blunt abdominal trauma due to an accidental fall, who presented splenic abscess a week after SAE and a review of the literature.

CASE REPORT: A 38-year-old male arrived at Emergency after an accidental fall with contusion of the left upper quadrant of the abdomen. Abdominal CT scan revealed the fracture of the lower splenic pole with intraparenchymal pseudoaneurysms (OIS spleen injury scale IV). Considering the hemodynamic stability, NOM was undertaken and SAE was performed. After a week, the patient developed a splenic abscess confirmed by Abdominal CT; therefore, splenectomy was performed. There was no evidence of bacterial growing in the perisplenic hematoma cultures but the histological examination showed multiple abscess and hemorrhagic areas in the spleen.

DISCUSSION: Splenic abscess after SAE during NOM of BSI is a rare major complication. The most frequently cultured organisms include Clostridium perfringens, Alpha-Hemoliticus Streptococcus, gram-positive Staphylococcus, gram-negative Salmonella, Candida, and Aspergillus. This case represents our first reported splenic abscess after SAE.

CONCLUSION: SAE is a very useful tool for BSI managing; splenic abscess can occur in a short time, even if it is a rare major complication, so it may be useful to monitor patients undergoing SAE, focusing not only on the hemodynamic parameters but also on the inflammatory and infectious aspects.

KEY WORDS: Abscess, Angioembolization, Spleen, Trauma

Introduction

In the last two decades, NOM of BSI has become the “gold standard” in the treatment of hemodynamically stable trauma patients. At present, SAE, which was first reported in traumatic splenic injuries in the early 1980s, is adopted as an effective procedure for hemodynamically stable patients with grade III, IV, and V splenic injuries (OIS), even in the absence of contrast blushes or pseudoaneurysms on the Abdominal CT scan. Recent studies demonstrated low rates of splenic abscess formation after SAE, respectively for distal and proximally SAE (0% to 1.9%). In this report, we present a case of a patient with a blunt abdominal trauma after an accidental fall, who developed a splenic abscess a week after SAE. A review of the indexed case-reports in literature was done searching on main databases (Pubmed-Medline). A study of the failure rates of SAE during NOM of BSI at our Unit from January 2007 to February 2014 was also carried out.
Presentation of the case

A 38-year-old male was observed at Emergency of our Hospital after an accidental fall at home with contusion of the left upper quadrant of the abdomen. The history showed no significant comorbidities, except for hypertension treated with calcium channel blockers, and a penicillin allergy. The laboratory exams revealed a mild leukocytosis with no significant modification of other parameters. A chest X-Ray showed a compound fracture of the back side of the VII, VIII, IX and X left ribs; Abdominal US demonstrated an effusion around the liver and in the pelvis.

An Abdominal CT scan showed a fracture of the lower splenic pole with evident areas probably related to the post-traumatic intraparenchymal pseudoaneurysms in the arterial phase (OIS Grade IV) (Fig. 1).

Due to hemodynamic stability, a NOM protocol was undertaken. The next day, the patient underwent a selective splenic artery angiography which confirmed the presence of multiple pseudoaneurysms of the splenic artery ramification in the equatorial-lower polar side of the spleen. A superselective embolization with spongostan gel and amagnetic metallic spirals was then performed (Fig. 2). Over the following days, the patient showed fever with laboratory findings of sepsis (WBC: 17.030 wbc/mm³; C-Reactive Protein: 15.05 mg/dl; Procalcitonin: 0.98 ng/ml), without a significant decrease in Hemoglobin values; therefore, an antibiotic-therapy with ciprofloxacin was undertaken. Abdominal US with contrast enhancement showed a small liquid effusion near the lower splenic pole, around the liver and in the mesogastric area. The spleen presented a dishomogeneous parenchyma especially in the equatorial area with normal vascularization of the lower and the upper poles. An abdominal CT scan showed a voluminous, dishomogeneous effusion inside the spleen (measures 11.5cm x 6.7cm x 16cm APxLLxCC) with hyperdense signal due to blood composition and air bubbles related to an ongoing septic-inflammatory process (Fig. 3). The effusion on the cranial side of the spleen reached the diaphragm with a satellite pleural effusion and a lower left lung lobe atelectasis.

The decision for a surgical approach was therefore taken. The exploration of the abdominal cavity showed peri-splenic, left-parietocholic and pelvic blood spillage that

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**Abbreviations**

SAE: Splenic Angioembolization  
NOM: Nonoperative Management  
BSI: Blunt Spleenic Injury  
OIS: Organ Injury Scale  
US: Ultrasound  
CT: Computed Tomography  
AP: Antero-posterior  
LL: Latero-lateral  
CC: Cranio-caudal

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Fig. 1: Abdomen CT: fracture of the lower splenic pole with arterial-phase evident areas probably related to post-traumatic intraparenchymal pseudoaneurysms (OIS spleen IV).

Fig. 2: Splenic artery angiography: presence of multiple pseudoaneurysms of the splenic artery ramifications for the spleen equatorial-lower polar side.
was removed and partially sampled for bacterial cultures; a massive hematoma was noted into the splenic area, surrounded by a fibrous encasement. The spleen seemed to be of normal size, with extensive laceration of the lower pole and with a diffuse necrotic area of the upper splenic pole; therefore a splenectomy was performed (Fig. 4).

Postoperatively, the patient was submitted to a large spectrum antibiotic therapy (Tigecycline and Teicoplanin) for a week. There was no evidence of bacterial growing in the perisplenic hematoma cultures. Due to the persistence of fever and the onset of hypoxia and hypocapnia, the patient was transferred to Intensive Care Unit, where he was treated with thoracentesis and Continuous Positive Airway Pressure (C-Pap). Due to the presence of thrombocytosis (1266x10^3/ul), the patient was submitted to two platelet-aphaeresis sessions. In the following days, clinical conditions gradually improved: the ultrasound scan showed only a residual small effusion (3 cm) around the spleen. The patient was discharged on the sixteenth post-operative day.

The histological examination showed multiple abscess and hemorrhagic areas with occasional small vessels thrombosis and initial collateral granulating tissue.

**Discussion**

Currently up to 80% of hemodynamically stable trauma patients are successfully treated adopting NOM protocols. The benefits of this approach include preservation of splenic immune function and reduction of rates of overwhelming postsplenectomy infection (OPSI). NOM has been accepted as the standard treatment for OIS grades I, II and III BSI but there is still no consensus in the management of the most severe splenic injuries. Banerjee et al. proposed incorporating the selective use of NOM and, if necessary, SAE, in hemodynamically stable patients with grade IV and V BSI in level I trauma centers, to reduce the rate of laparotomy recourse. According to the Eastern Association for the Surgery of Trauma practice management guideline, NOM of BSI is the first treatment of choice in hemodynamically stable patients, irrespective of the grade of injury, patient age, or the presence of associated injuries. SAE is performed during NOM of BSI from 1.4% to 10% respectively in low and high embolization centers.

To date, several retrospective studies have demonstrated that the use of angioembolization is a safe and effective adjunct to NOM of BSI, associated with a low incidence of complications and acceptable failure rate. According to a recent meta-analysis of Schnirger et al., the overall failure rate after SAE during NOM is 10.2%, ranging from 0% to 33.3%. Major complications requiring splenectomy range between 8.7% for proximal SAE and 10.8% for distal SAE: re-bleeding (4.7% - 6.3%), infarction (0%-1.6%) and infection (1.9% - 0%) . An age of over 65 years represents a risk factor for a...
higher rate of major complications. In a recent study of Ekeh et al., splenic abscess after SAE occurs in 6.8% of NOM for splenic trauma: 2 (33%) patients were treated with splenectomy, other 2 (33%) with laparoscopic drainage, 1 (17%) with CT-guided drainage and in 1 (17%), the abscess regressed with an antibiotic therapy. Splenic abscesses were more frequent in proximal splenic embolization, in contradiction with Schnürriger’s meta-analysis. Infections mainly regarded the spleen; to our knowledge, there is only one case of a perisplenic abscess. The most commonly cultured organisms include Clostridium perfringens, Alpha-Hemolyticus Streptococcus, gram-positive Staphylococcus, gram-negative Salmonella, Candida and Aspergillus. Our patient showed signs of infection approximately one week after SAE. Surgical perisplenic effusion cultures resulted negative despite clinical, laboratory and radiological signs indicating a splenic abscess; this last feature was subsequently confirmed by histological examination. Considering the period between January 2007 and February 2014, this case represents our first reported splenic abscess after SAE during NOM of BSI. During that period, we admitted 203 BSI, of which 38 were treated with NOM (18.7%). 12 SAE (31.5%) were performed and 3 procedures failed (25%): two because of pseudoaneurysm splenic artery formations and one due to the splenic abscess in the case which we reported.

Conclusion

SAE is a very useful tool in the treatment of BSI; splenic abscess can occur in a relatively short time, even if it is a rare major complication, so it may be useful to monitor patients undergoing SAE, focusing not only on the patient’s hemodynamic parameters but also on the inflammatory and infectious aspects.

References

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