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Case Report

Surgical management of atraumatic splenic rupture

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ABSTRACT

Atraumatic splenic rupture (ASR) is a rare, spontaneous, and potentially life-threatening condition that occurs in the absence of trauma; yet the management of ASR has largely defaulted to the treatment algorithm related to blunt splenic trauma. Our aim is to determine if it is appropriate and safe to use the treatment algorithm for blunt splenic trauma in the management of both pathological and non-pathological ASR. We present a case of non-pathological ASR that was successfully managed without splenectomy. A comprehensive literature review on spontaneous ASR was also performed to include publications from January 1975 to February 2015. 914 total cases of ASR were identified: 70 non-pathological and 844 pathological. Overall, 86.5% of these patients received splenectomy based on the presence or absence of traditional signs of clinical instability or deterioration, as utilized in cases of traumatic splenic rupture. There were only 11 non-pathological cases detailed. Unlike our present case, all of these cases resulted in splenectomy. The present case is that of a 37 year-old man with grade III splenic rupture, successfully managed without surgery or splenectomy. Details regarding ASR patients are limited in current literature. The majority of patients with ASR will receive splenectomy, regardless of etiology. Our review would suggest that it is appropriate and safe to pursue conservative non-surgical therapy in select patients who present with ASR of any given etiology (even non-pathological), while utilizing the same indications for splenectomy used in cases of blunt abdominal trauma.

Keywords: Atraumatic, Idiopathic, Non-pathological, Plenic rupture, Spontaneous

INTRODUCTION

Atraumatic splenic rupture (ASR) is a potentially life-threatening condition that occurs spontaneously, in the absence of trauma. Fortunately, it is also rare, with an estimated incidence of around 1.2 cases per year.[1] The presentation of ASR can be non-specific and subtle, presenting most often with a sudden onset of abdominal pain, abdominal tenderness, nausea, and/or vomiting.

More severe cases may present with peritoneal findings, Kehr's sign, or signs of shock.[2] Due to its low incidence, non-specific presentation, and lack of trauma history, ASR is easily misdiagnosed for other more common causes of LUQ abdominal pain such as diverticulitis, pancreatitis, and nephrolithiasis.[3]

This delay in diagnosis can result in persistent and significant internal hemorrhage, hemodynamic instability, and eventually death. The mortality rate for ASR is relatively high, reported in one systematic review to be 12.2%.[4]

ASR is separated into two distinct categories: pathological and non-pathological. The majority of cases are pathological, signifying that they are at least in part due to one or more identifiable underlying etiologies, including infection, bleeding disorders, and malignancy, which have the potential to cause splenic enlargement or fragility, leading to spontaneous rupture. Non-pathological ASR on the other hand is frequently defined

in the literature by the Orloff and Peskin criteria, which include: thorough history reveals no antecedent trauma; no evidence of disease in organs other than the spleen that can cause rupture; no perisplenic adhesions or scarring consistent with trauma or past rupture; and normal spleen on gross and histological examination.[5] In a systematic review of ASR, Renzulli et al. noted that 93.0% of patients with ASR are cases of pathological splenic rupture, whereas 7.0% have no identifiable etiologic cause and are thus non-pathological (idiopathic) cases.[4]

Because the etiologies of ASR (Table 1) encompass a wide breadth of causes from infectious to neoplastic to non-pathological, the initial work-up of a patient newly diagnosed with ASR is unclear. The management of ASR has instead largely defaulted to the treatment algorithm related to blunt splenic trauma, an entity more commonly seen in practice.[6,7] However, it is unclear as to how the management of ASR should differ from that of blunt splenic trauma, particularly regarding to the timing or indications for surgical intervention. As it stands, the management algorithm designed for blunt splenic trauma largely focuses on the hemodynamic stability of the patient, recommending observation and conservative management if the patient is stable and splenectomy if the patient is unstable.

We present a case of non-pathological ASR in an otherwise healthy 37-year-old patient. In doing so, we highlight factors contributing to the medical management of ASR. The goal of this review is to identify any differences in management and outcomes of pathological and non-pathological ASR in current literature, and to determine if it is appropriate and safe to use the treatment algorithm for blunt splenic trauma in the management of ASR.

CASE REPORT

A 37 year-old man was admitted with a three-hour history of left lower quadrant abdominal pain. He was abruptly awoken by what he described as a "constant eight out of ten cramping" abdominal pain. He endorsed chills, myalgias and pain that worsened with deep breathing. He denied any change in bowel movements, blood or mucus in his stool, nausea/vomiting, or bruising to his abdomen. Patient denied history of trauma or similar symptoms in the days and weeks leading up to this pain. He denied recent travel, fevers, malaise, or rashes. The patient's past medical history was remarkable only for morbid obesity, smoking (0.5 packs/day x 18 years), and occasional symptoms of acid reflux, although he was not taking any medications regularly at the time of presentation. Upon admission, his vital signs showed mild hemodynamic instability: BP 130/64 mmHg, HR 111 bpm, temp 37.1 °C, RR 28, SpO_2 96% on room air. On physical exam, diffuse abdominal pain and tenderness greatest in the LUQ was noted; otherwise the abdomen was soft without hepatosplenomegaly, rebound or guarding. Within hours, the patient's pain continued to worsen and he began to feel pain radiating to his left shoulder.

Initial labs were remarkable only for increased WBC counts. Hemoglobin and hematocrit values were within normal range, 14.5 g/dL and 42% respectively. D-dimer, troponin, EKG, and chest x-ray were unremarkable. An intra-abdominal process was suspected given the normal EKG, low clinical risk for pulmonary embolism with negative d-dimer, and normal CXR. CT abdomen/pelvis with contrast was subsequently performed and found to demonstrate a grade III splenic laceration with subcapsular hematoma and surrounding hemorrhage (Figure 1). The spleen showed multiple areas of hypoattenuation, and a large amount of high attenuation fluid was seen both surrounding the spleen and liver and also tracking along the paracolic gutters into the pelvis. There was no evidence of active extravasation, although the report noted relative flattening of the IVC suggestive of intravascular volume depletion from hemorrhage.

The patient remained hemodynamically stable despite downtrending hemoglobin found on repeat labs, thus the patient received only NS fluid bolus without blood transfusion. Surgery was consulted and made the recommendation for the patient to be admitted for initial close monitoring. Serial hemoglobin and hematocrit were obtained every four hours and the patient was made aware that he may require emergent splenectomy should he not remain stable with non-operative management. To further evaluate infectious etiology with EBV, Monospot test was obtained and confirmed negative. On hospital day two his hemoglobin fell to 9.4 from 11.2 g/dL the day prior, without any change in clinical symptoms. Repeat abdominal CT was performed but demonstrated stable splenic laceration unchanged from initial imaging. Throughout his four-day stay in the hospital, the patient's pain improved with successful non-operative management. Vital signs progressed back into normal ranges.

Mildly tender in the LUQ but hemodynamically stable, the patient was discharged on hospital day four. Recovery was overall uncomplicated. Three clinic follow-up visits over the next two months documented a gradual improvement in abdominal pain and tenderness, resolving hematoma on repeat CT imaging, and a return to his baseline health.

A comprehensive literature review on spontaneous ASR was performed. All publications from January 1975 to February 2015 were examined. The literature search (PubMed, Cochrane Library, and UpToDate) was limited to patients over 18 years old, and restricted to papers written in English. Search terms were as follows: atraumatic (all fields) and "splenic rupture" MeSH terms or splenic (all fields) and rupture (all fields) or splenic rupture (all fields); spontaneous (all fields) and atraumatic (all fields) and splenic rupture (mesh terms) or

splenic (all fields) and rupture (all fields) or splenic rupture (all fields). Papers without full text availability, editorials, or opinion pieces were excluded. Splenic ruptures of predominantly traumatic origin or considered delayed splenic ruptures were additionally excluded.

All publications were reviewed and the following information gathered: patient sex, age, clinical symptoms, vitals on admission, physical exam findings, labs, hemoglobin level, imaging, splenectomy (including surgical indications) or conservative management, mortality, splenic pathology, and underlying etiology.

Table 1: Causes of a traumatic splenic rupture.

Infectious	Malignancy	Inflammatory	Primary splenic	Iatrogenic	Non-pathological, i.e. Idiopathi	Mechanical disorder	Toxin or chemical	Hematologic
N = 262	N = 200	N = 194	N = 107	N = 76	N = 70	N = 40	N = 19	N = 19
Viral	Leukemia	Pancreatitis	Ceroid histiocytosis	Anticoagulant use	Triggering factor (ex. Coughing, sneezing, emesis)	Pregnancy	Snake bite	Idiopathic thrombocytopenic purpura
Hepatitis	Lymphoma	Amyloidosis	Splenic peliosis	G-CSF	No triggering factor	Portal hypertension	Iv drug use	Hemophilia
Hiv	Metastasis	Lupus erythematosus	Splenic cyst	Thrombolytic agents		Congestive splenomegaly	Cocaine	Factor xiii deficiency
Infectious mono	Malignant spindle cell tumor	Rheumatoid arthritis	Splenic hemangioma	Dialysis				Protein s deficiency
CMV	Non-small cell lung cancer	Polyarteritis nodosa	Splenic malignancy (ex. Angiosarcoma)	Lithotripsy				Hemolytic anemia
Rubella	Pancreatic carcinoma	Wegener's granulomatosis	Splenic infarction, artery dissection, or venous thrombosis					
Varicella	Melanoma		Congenital malposition (i.e. Short splenic pedicle)					
Bacterial	Choriocarcinoma							
TB	Myleodysplastic syndromes							
Typhoid	Multiple myeloma							
Endocarditis	Polycythemia vera							
Pneumonia								
Legionella								
Bartonella								
Fungal								
Aspergillosis								
Protozoal								
Babesia								
Malaria								
Toxoplasma								
Other								
Syphilis								

Table 2: Data of the published literatures on non-pathological a traumatic splenic rupture.

Age (years) /Sex	Trigger	Vitals on admission (mmHg, bpm, breaths/min, °C)	Physical exam findings	Lab work-up a diagnostic approach	Hgb g/dL	Diagnostic imaging	Extent of splenic injury	Management	Indication for surgery	Reference
37/M	No	BP 130/64 HR 111 RR 28 Temp 37.1 O2 96% on RA	LUQ tenderr no HSM, Ke sign	CBC, BMP, blood smear, lipase, UA, D-dimer, troponins, EKG, Monospot INR	9.4	CXR, CT	Grade III	Conservative management*	n/a	Present Case Report
44/M	cough 2° to PNA	BP 90/45 HR 128 Temp 39.8 O2 96% on RA	LUQ tenderness, guarding, Kehr's sign	CBC, EKG, UA, urine culture, LFTs,BMP; serology for EBV, CMV, and hepatitis	11.4	CT	Grade V	Urgent splenecton discharged POD6	CT findings	Kocael et al[33]
56/M	No	BP 110/78 HR 110 RR 20 O2 91% on 5L	Left-sided abdominal tenderness, Kehr's sign	CBC	7.5	CT	Grade IV	Splenectomy (afte failed conservative approach)	Continued hemodyna-mic instability	Weaver et al[57]
27/M	trivial injury	BP 80/60 HR 60 RR 26 Afebrile	Guarding an rebound tenderness	CBC, LFTs, electorlytes, blo glucose, BUN, amylase, EKG	11.5	CXR, plain abdomina l films, CT	macerat-ed spleen	Splenectomy, discharged POD7	CT findings	Sowers and Aubrey-Bassler[54]
30/M	NA	NA	NA	NA	12.2	no CT	-	Splenectomy	peritonitis	Elvy et al[22]
40/M	NA	sys BP 88	NA	NA	10.2	no CT	-	Splenectomy	peritonitis	Elvy et al[22]
51/M	NA	sys BP 70	NA	NA	6.7	CT	Grade V	Splenectomy	Hypovole-mic shock	Elvy et al[22]
52/M	NA	sys BP 110	NA	NA	8.9	CT	Grade V	Splenectomy	Hypovole-mic shock	Elvy et al[22]
69/F	COPD exacer-bation	BP 99/49 HR normal O2 99% on 28% O2	Lethargic, lower abdominal tenderness on palpation	CBC	10.4	CT	large intraspl-enic hematom a	Splenectomy	Clinical deterioratio n, acute anemia, CT findings	Amonkar and Kumar[10]
61/F	No	VSS, WNL	LUQ guarding	CBC	Normal	CXR, CT	large splenic hemato-ma	Splenectomy	Clinical deteriorati-on, CT findings	Amonkar and Kumar[10]
20/M	No	BP 120/71 HR 87 RR 16 Temp 37	Epigastric and LUQ tenderness with guarding, occult blood on rectal exam,Kehr's sign	CBC,BUN,Cr, lipase, total bilirubin, LDH, ALP, LFTs, UA	15.1	Abdomin al plain films, CT	Abdomi-nal free fluid	Splenectomy (after failed conservative approach), discharged POD7	Clinical worsening, CT findings	Laseter and McReynolds[37]
50/M	No	BP 137/73 HR 124 RR 24	Intermitte nt pain with palpation, Kehr's sign	EKG, blood glucose, repeat EKG, ABGs,CBC,c oagulation studies,amyla se,cardiac enzymes, echo	NA	CXR, CT	large perisple-nic hemato-ma	Splenectomy, discharge POD3	Worsening pain, sudden drop in BP and in Hct, CT findings	McDonal[41]

NA = not available; VSS = vital signs stable; WNL = within normal limits; ABGs = arterial blood gases; HD = hospital day *included IVF resuscitation, surgery consult, close monitoring and admission, serial CBC.

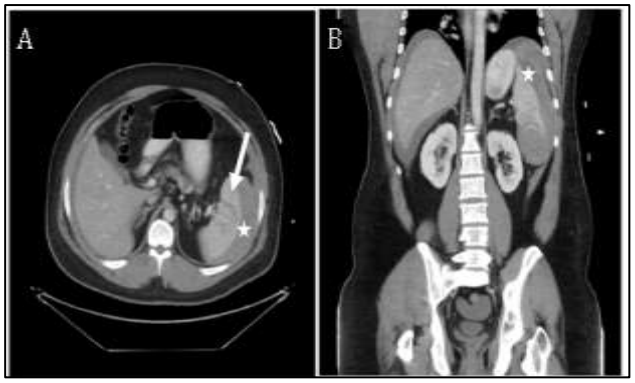

Figure 1: CT abdomen/pelvis obtained on day of admission. (A, B) Axial and coronal views of our patient's grade III splenic laceration (white arrow) with subcapsular hematoma and surrounding hemorrhage (stars) tracking around the spleen and liver and into the pelvis; no evidence of active extravasation.

Our search identified 51 peer-reviewed journal articles regarding ASR, together presenting 914 total cases.[4,11-59] 70 cases were non-pathological (representing 8% of all ASR cases reviewed) and 844 (or 92%) were pathological. Many etiologies were identified in our review of the literature and can be organized into eight broad categories (Table 1). We found that although the etiologies are numerous, infectious causes made up the majority of the cases (31%), followed by malignancies (24%) and inflammatory causes (23%). Although we attempted to identify patient information regarding characteristics, diagnostics, clinical management, and outcomes in each case, this information was provided for only 36 patients (less than 5%) out of all reported cases of ASR that were either associated with one of these three most common etiologies or considered to be non-pathological. Overall, 86.5% of these patients received splenectomy.

Underlying infectious etiology

Four cases were detailed, of which splenectomy was performed in two. The indication for surgery was hemodynamic instability paired with CT findings in one case, and new onset of fevers in the other. The two cases that were managed conservatively involved patients that were hemodynamically stable without peritoneal signs. Despite each patient having a different underlying infection (infectious mononucleosis, *Bartonella henselae*, *Babesiosis*, and acute HIV), all four patients survived.

Underlying neoplastic etiology

13 cases were detailed, 12 of which were considered candidates for splenectomy. Two of these 12 died before undergoing planned splenectomy, resulting in only ten patients having undergone splenectomy. Indications for surgery included: worsening pain coupled with CT findings, hemodynamic instability, hypovolemic shock, acute or severe drop in Hgb, signs of peritonitis, and clinical deterioration. There were no deaths within one month post-splenectomy, although two of the ten died within two months presumably due to their underlying malignancy. One patient was managed conservatively and survived the acute splenic rupture. However, this patient died three months later due to complications from the underlying malignancy.

Underlying inflammatory etiology

Eight cases were detailed. Seven cases resulted in splenectomy. Indications for surgery included: hemodynamic instability, severe or acute anemia, cardiac arrest, EKG ischemic abnormalities paired with anemia, CT findings alone, and worsening pain. Of these patients, all survived the first month post-splenectomy, while one "eventually" died due to infection. The one patient that was managed conservatively, a 49 yong female presenting with stable vital signs and no peritoneal signs, survived.

Non-pathological etiology

11 cases were detailed (Table 2). All cases resulted in splenectomy mean age of 45 years (range: 20-69), two females, one patient with fever, 36% hemodynamically unstable, 55% with peritoneal signs, and one patient with Hgb <7 g/dL. Two patients did not receive imaging but underwent immediate laparotomy for signs of peritonitis. Other indications for surgery included: hemodynamic instability, clinical worsening, hypovolemic shock, and sudden drop in Hgb. While no cause was definitively identified, some cases were noted to have possible triggers for the acute rupture including: severe cough, trivial injury, and COPD exacerbation. When grade of splenic injury was provided, it was consistently either grade IV or V. All patients survived, though it is noted that three months post-splenectomy one patient died due to stroke thought to be unrelated to ASR.

DISCUSSION

The amount of specific data available in literature regarding the details of ASR cases and the management courses is very limited. Currently the indications for splenectomy in ASR, regardless of known or unknown etiology, have generally included those identified in association with splenic rupture owing to blunt abdominal trauma: hemodynamic instability, high grade splenic injury, and signs of peritonitis.[8]

Renzulli et al performed a comprehensive review of ASR in the literature and similarly found the paucity of patient data as well as the simple rarity of ASR limiting to their analyses.[4] They were able however to find an overall ASR-related mortality rate of 12.2%, as well as to make several conclusions relating to risk factors and management of ASR as a general entity. While neither infectious nor inflammatory etiology were stand-alone

risk factors for increased ASR-related mortality, the underlying etiology of neoplastic disorders was found to be particularly associated with poorer outcomes regardless of whether they were managed with or without splenectomy, while non-pathological etiology was associated with a decreased ASR-related mortality rate. They concluded that patients with non-malignant pathological etiology could be treated with organ-preserving surgery, a non-surgical approach, and/or transcatheter arterial embolization. Yet it was their recommendation that all patients with ASR of malignant etiologies undergo splenectomy or transcatheter arterial embolization as a temporary stabilizing measure. In their review, over 84% of ASR patients underwent splenectomy (either as initial treatment or after failed non-conservative treatment).

Corresponding to the results of Renzulli et al, our literature review demonstrated that 86.5% of ASR patients underwent splenectomy. In comparison, only 40-50% of patients with traumatic splenic injury have historically required operative management, which most typically is splenectomy.[9,10] Thus it appears that if a patient presents with splenic rupture in the absence of trauma, they will be significantly more likely to undergo splenectomy than if their rupture had been of traumatic etiology. Despite the much higher likelihood of splenectomy, we found that most management decisions in cases of ASR were in due course made based on a combination of objective and subjective factors not unlike the traditional indications for splenectomy in traumatic rupture. Commonly implicated in the decision to perform splenectomy in ASR were factors such as clinical deterioration, development of more severe symptoms or peritoneal signs, and an abrupt decline in hemoglobin.

Furthermore, the decision for and success of surgical versus non-surgical management of the acute splenic rupture does not appear to be significantly affected by the underlying etiology, contrary to the findings of Renzulli et al. We have found that in each etiological category of ASR patients infectious, inflammatory, malignant, and non-pathological although there was at least one case successfully managed without surgery, the significant majority of patients underwent surgery. Given this observation and the low numbers of patients with detailed clinical courses reported, we do not believe the existing literature supports the use of underlying etiology as a stand-alone variable affecting the indications for splenectomy. Despite recognizing the limited and retrospective nature of the literature on this subject, we conclude that it appears reasonable to rely on the traditional signs of clinical instability or deterioration to influence the decision to proceed with splenectomy in patients with ASR, independent of underlying etiology.

Nevertheless, we still recommend that a thorough etiological work-up be carried out in any patient presenting with ASR since the identification of a specific underlying diagnosis could influence the necessity of other specific treatments and affect the long term clinical course of the patient. Specific work-up should initially include CBC and abdominal imaging (generally CT scan) to evaluate for evidence of malignancy or underlying inflammatory etiologies. It should be noted that while suspicion for underlying inflammatory etiology may have been raised by CBC abnormalities, most diagnoses were confirmed post-splenectomy through splenic histopathology. Additionally, if history or physical exam suggests infection, a full infectious disease work-up may be of value and should be guided by the patient's history, specific signs, extra-abdominal symptoms, and likely infectious exposures.

Finally, with regards to patients specifically described as having non-pathological ASR, all individually described cases to date have been managed with splenectomy. The successful non-operative management of our present case would be the first detailed in the literature. We do note our patient's grade III splenic injury to be unique, with other documented cases all grade IV or higher. This raises the concern that our patient is different from the typical patient with non-pathological ASR, presenting with less advanced pathology than is to be expected in most patients when no underlying etiology is found. Thus the success of our patient may not be representative of the true likelihood for success with non-operative management of these patients in general. Nonetheless, the success of our present case demonstrates that it is safe for select patients presenting with ASR of unclear etiology to be managed conservatively, as long as the traditional indications for splenectomy in trauma patients hemodynamic instability, high-grade splenic injury, or clinical deterioration are absent. This recommendation differs with that of Renzulli et al, who concluded that despite a lower ASR-related mortality rate of 2% in patients with non-pathological ASR, these patients should receive total splenectomy even if hemodynamically stable.[4]

By using the success of our patient to open the possibility for conservative management in ASR of unknown etiology, future patients who present similarly may enjoy the benefits of non-operative treatment in terms of decreased length of stay, avoidance of operative morbidity, and preservation of immune function. However, it should be discussed with these patients at the outset of their initial hospitalization that should they show any signs or symptoms of deterioration, splenectomy is likely to be performed. Lastly, we would recommend in cases determined to be suitable for non-operative management, patients should be closely followed in the immediate time period after splenic rupture as there still may be an underlying disease process that was not identified in the initial hospitalization. The current literature, unfortunately, does not provide any data or long-term outcomes to help define the specifics of such an optimal surveillance plan.

CONCLUSION

As presentation can be non-specific and mortality rates of ASR can be relatively high, it is important that providers have a low threshold to suspect ASR. Although data and literature regarding ASR are limited, review of cases with ASR in the context of identified malignant, infectious, inflammatory, or non-pathological etiologies show that the vast majority of patients will undergo splenectomy. While we recommend that all attempts be made to identify any underlying etiologies in order to direct future treatment and improve the patient's overall health in the long-term, our review suggests that the etiology of ASR does not independently influence management or survival. Patients of any etiology, including those patients with no underlying identifiable pathology, can be successfully managed with or without splenectomy while experiencing comparable survival outcomes. Despite the overall higher likelihood of splenectomy in ASR patients, the indications for splenectomy appear to be analogous to those traditionally used in traumatic splenic rupture, with consideration of the patient's entire clinical picture in addition to hemodynamic stability, grade of splenic injury, and presence or absence of peritonitis.

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REFERENCES

1. Elvy A, Harbach L, Bhangu A. Atraumatic splenic rupture: a 6-year case series. Eur J Emerg Med. 2011;18:124-6.
2. Instructions for sports medicine patients, 2012. Available at https:// www.elsevier.com /books/ instructions-for-sports-medicine-patients /safran/ 978-1-4160-5650-8. Accessed on 2 June 2016.
3. Cartwright SL, Knudson MP. Evaluation of acute abdominal pain in adults. Am Fam Physician. 2008;77:971-8.
4. Renzulli P, Hostettler A, Schoepfer AM, Gloor B, Candinas D. Systematic review of atraumatic splenic rupture. Br J Surg. 2009;96:1114-21.
5. Orloff MJ, Peskin GW. Spontaneous rupture of the normal spleen; a surgical enigma. Int Abstr Surg. 1958;106:1-11.
6. Moore FA, Davis JW, Moore EE, Cocanour CS, West MA, Mcintyre RC. Western trauma association (WTA) critical decisions in trauma: management of adult blunt splenic trauma. J Trauma. 2008;65:1007-11.
7. Testerman GM, Easparam S, Jacome F. Western trauma association blunt splenic injury algorithm is useful in spontaneous rupture of a normal spleen. Am Surg. 2011;77:85-6.
8. Kokabi N, Shuaib W, Xing M, Harmouche E, Wilson K, Johnson JO, et al. Intra-abdominal solid organ injuries: an enhanced management algorithm. Can Assoc Radiol. 2014;65:301-9.
9. Matbouly EM, Jabbour G, Menyar EA, Peralta R, Abdelrahman H, Zarour A, et al. Blunt splenic trauma: Assessment, management and outcomes. Surgeon. 2016;14(1):52-8.
10. Hildebrand DR, Sassi A, Ross NP, Macvicar R, Frizelle FA, Atson AJ. Modern management of splenic trauma. British Med J. 2014;348:1864.
11. Amonkar SJ, Kumar EN. Spontaneous rupture of the spleen: three case reports and causative processes for the radiologist to consider. Br J Radiol. 2009;82:111-113.
12. Anyfantakis D, Kastanakis M, Karona P, Fragiadakis G, Kokkinos I, Bobolakis E. Spontaneous rupture of the spleen masquerading as a pulmonary infection. Case Rep Surg. 2014;2014:196525.
13. Azar F, Brownson E, Dechert T. Cocaine-associated hemoperitoneum following atraumatic splenic rupture: a case report and literature review. World J Emerg Surg. 2013;8(1):33.
14. Bandiera G, Poulin E. Unusual case of acute onset abdominal pain: uses and limitations of medical imaging. Canadian J Emergency Med. 2003;5:263-7.
15. Bouliaris K, Karangelis D, Daskalopoulos M, Spanos K, Fanariotis M, Giaglaras A. Hemorrhagic shock as a sequela of splenic rupture in a patient with infectious mononucleosis: focus on the potential role of salicylates. Case Rep Med. 2012; 2012:497820.
16. Carlin F, Walker AB, Pappachan JM. Spontaneous splenic rupture in an intravenous drug abuser. Am J Med. 2014;127:7-8.
17. Carta G, Dalfonso A, Nallbani A, Palermo P, Franchi V, Patacchiola F. Spontaneous rupture of splenic hemangioma in puerperium. Clin Exp Obstet Gynecol. 2012;39:407-8.
18. Chan RS, Abdul Azi YF, Chandran P, Ng EK. Splenic amyloidosis: a rare cause of spontaneous splenic rupture. Singapore Med J. 2011;52:232-5.
19. Chau EMC, Chan ACW, Chan WK. Spontaneous atraumatic rupture of a normal-sized spleen due to AL amyloid angiopathy. Amyloid. 2008;15:213-5.
20. Deprez FC, Colin GC, Hainaut P. Spontaneous (atraumatic) splenic rupture complicating anticoagulant treatment for splenic infarction. Acta Cardiol. 2013;68:421-4.
21. Santis GC, Oliveira LC, Ramos AF, Silva ND, Falcão RP. Pathologic rupture of the spleen in a patient with acute myelogenous leukemia and leukostasis. Rev Bras Hematol Hemoter. 2014;36:290-2.
22. Duan YF, Jiang Y, Wu CX, Zhu F. Spontaneous rupture of primary splenic angiosarcoma: a case report and literature review. World J Surg Oncol. 2013;11:53.
23. Etzion Y, Benharroch D, Saidel M, Riesenberg K, Gilad J, Schlaeffer F. Atraumatic rupture of the spleen associated with hemophagocytic syndrome and isolated splenic peliosis. Case report. Acta

Patho Microbiol Immunol Scandinavica. 2005;113:555-7.
24. Fallingborg J, Laustsen J, Jakobsen J, Winther P, Svanholm H. Atraumatic rupture of the spleen in periarteritis nodosa. Acta Chir Scand. 1985;151:85-7.
25. Farley DR, Zietlow SP, Bannon MP, Farnell MB. Spontaneous rupture of the spleen due to infectious mononucleosis. Mayo Clin Proc. 1992;67:846-53.
26. Forghieri F, Morselli M, Leonardi G, Potenza L, Bonacorsi G, Coluccio V, et al. Atraumatic splenic rupture in patients with myelodysplastic syndromes: report of a case occurred during treatment with 5-azacitidine and review of the literature. Leuk Res. 2012;36:52-6.
27. Ghinescu C, Sallami Z, Jackson D. Choriocarcinoma of the spleen- a rare cause of atraumatic rupture. Ann R Coll Surg Engl. 2008;90:12-4.
28. Goddard SL, Chesney AE, Reis MD, Ghorab Z, Brzozowski M, Wright FC, et al. Pathological splenic rupture: a rare complication of chronic myelomonocytic leukemia. Am J Hematol. 2007;82:405-8.
29. Huckins DS, Barnett T. Occult splenic rupture presenting as acute scrotal swelling. J Emerg Med. 2012;43:438-41.
30. Jahadi MR, Bailey W, Crenshaw A. Atraumatic splenic rupture simulating ruptured sigmoid diverticulitis: report of a case and review of the literature. Dis. Colon Rectum. 1975;18:59-61.
31. Jha AK, Pokharna R, Nijhawan S, Nepalia S, Jain P, Suchismita A, et al. Atraumatic rupture of spleen. Trop Gastroenterol. 2011;32:238-40.
32. Kang C, Kim DH, Kim SC, Kim DS, Jeong CY. Atraumatic splenic rupture after coagulopathy owing to snakebite. Wilderness Environ Med. 2014;25:325-8.
33. Kocael PC, Simsek O, Bilgin IA, Tutar O, Saribeyoglu K, Pekmezci S, et al. Characteristics of patients with spontaneous splenic rupture. Int Surg. 2014;99:714-8.
34. Kroger N, Zander AR. Dose and schedule effect of G-GSF for stem cell mobilization in healthy donors for allogeneic transplantation. Leuk Lymphoma. 2002;43:1391-4.
35. Lal A, Singhal M, Sharma N, Bhalla A, Khandelwal N. Isolated splenic peliosis with spontaneous rupture after a viperine bite. Am J Emerg Med. 2014;32:194-6.
36. Lam GY, Chan AK, Powis JE. Possible infectious causes of spontaneous splenic rupture: a case report. J Med Case Rep. 2014;8:396.
37. Laseter T, Mcreynolds T. Spontaneous splenic rupture. Mil Med. 2004;169:673-4.
38. Lieberman ME, Levitt MA. Spontaneous rupture of the spleen: a case report and literature review. Am J Emerg Med. 1989;7:28-31.
39. Maqbool MS, Brush RG, Northrup M. Atraumatic splenic rupture. Ultrasound Q. 2014;30:208-10.
40. Martin TCS, Martin NK, Naresh KN, Nelson M. Atraumatic splenic rupture secondary to chronic HIV infection. Int J STD AIDS. 2013;24:989-91.
41. Mcdonald CC. Delayed splenic hematoma rupture. Am J Emerg Med. 1995;13:540-2.
42. Merrell SW, Gloviczki P. Splenic artery dissection: a case report and review of the literature. J Vasc Surg. 1992;15:221-5.
43. Mukhopadhyay M, Chatterjee N, Maity P, Patar K. Spontaneous splenic rupture: A rare presentation of dengue fever. Indian J Crit Care Med. 2014;18:110-2.
44. Ogu US, Keim G, Cleary AJ, Bloch RS. Atraumatic splenic rupture secondary to metastatic non-small-cell lung cancer. J Surg Case Rep. 2013;2013(7).
45. Piotti G, Filippin F, Rampino T, Bedino G, Bosio F, Morbini P, et al. Atraumatic spleen rupture in dialyzed patients: clinical report and review of the literature. Perit Dial Int. 2011;31:486-92.
46. Renzulli P, Schoepfer A, Mueller E, Candinas D. Atraumatic splenic rupture in amyloidosis. Amyloid. 2009;16:47-53.
47. Rice JP, Sutter CM. Spontaneous splenic rupture in an active duty Marine upon return from Iraq: a case report. J Med Case Rep. 2010;4:353.
48. Roll GR, Lee AY, Royaie K, Visser B, Hanks DK, Knudson MM, et al. Acquired amyloidosis from injection drug use presenting with atraumatic splenic rupture in a hospitalized patient: a case report. J Med Case Rep. 2011;5:29.
49. Sachithanandan A, Gleadhill I, Alexander HD, Morris TCM. Spontaneous splenic rupture in atypical (Philadelphia chromosome negative) chronic myeloid leukaemia following blastic crisis. Ir Med J. 2003;96:181-2.
50. Sajid MS, Howell P, Leaver C, Roberts K, Sains P. Spontaneous splenic rupture secondary to metastatic malignant spindle cell tumour. Singapore Med J. 2012;53:208-10.
51. Salame J, Mojddehian N, Kleiren P, Petein M, Lurquin P, Dediste A, et al. Atraumatic splenic rupture in the course of a pneumonia with Streptococcus pneumoniae. Case report and literature review. Acta Chir Belg. 1993;93:49-53.
52. Seible DM, Khatana SAM, Solomon MP, Parr JB. Hoof beats may mean zebras: atraumatic splenic rupture. Am J Med. 2013;126:778-80.
53. Smith WM, Lucas JG, Frankel WL. Splenic rupture: a rare presentation of pancreatic carcinoma. Arch. Pathol Lab Med. 2004;128:1146-50.
54. Sowers N, Bassler FK. Trivial trauma and delayed rupture of a normal spleen: a case report. J Med Case Rep. 2011;5: 591.
55. Vallabhaneni S, Scott H, Carter J, Treseler P, Machtinger EL. Atraumatic splenic rupture: an unusual manifestation of acute HIV infection. AIDS Patient Care STD. 2011;25:461-4.
56. Landingham SB, Rawls DE, Roberts JW. Pathological rupture of the spleen associated with hepatitis. Arch Surg. 1984;11:224-5.

57. Weaver H, Kumar V, Spencer K, Maatouk M, Malik S. Spontaneous splenic rupture: a rare life-threatening condition; diagnosed early and managed successfully. Am J Case Rep, 2013;14:13-5.
58. Wehbe E, Raffi S, Osborne D. Spontaneous splenic rupture precipitated by cough: a case report and a review of the literature. Scand J Gastroenterol. 2008;43:634-7.
59. Wilson CI, Inchausti B, Sendzischew H, Robinson MJ. Ceroid histiocytosis: an unusual cause of atraumatic splenic rupture. South Med J. 2001;94:237-9.

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