Delayed splenic rupture after a minor blunt trauma: A case report and literature review

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Abstract

Delayed Splenic Rupture (DSR) is a rare but well-known manifestation of Blunt Splenic Injury (BSI), which most commonly occurs following a car accident, a fall from a great height, or a direct blow to the left thorax or abdomen. If the history of trauma is remote or unknown, the diagnosis can be difficult or missed, and a high index of suspicion is not warranted. Regardless of the time and mechanism of the inciting event, DSR should be considered in the differential diagnosis of an acute surgical abdomen. We present a case of DSR in an 81-year-old woman caused by a remote minor blunt abdominal trauma four weeks prior to her admission to our emergency department for acute abdominal pain and hemorrhagic shock (ATLS CLASS III). Starting with this case report, we conducted a literature review on the subject in order to raise awareness, knowledge, and understanding of DSR among emergency clinicians.

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Highlights

- *Delayed Splenic Rupture (DSR) is a rare but well reported clinical entity following blunt splenic injury.*
- *DSR can even occur after a trivial trauma.*
- *DSR can cause severe morbidity and mortality, regardless of the severity of the initial trauma.*
- *A detailed history of major or minor trauma must be elicited for any patients with acute abdominal pain and signs of haemodynamically instability.*

Case report

An 81-year-old woman arrived at our Emergency Department (ED) complaining of severe, sharp chest and epigastric pain. These symptoms started four hours earlier, waking her up from her sleep, and were accompanied by nausea and generalized weakness.

The past medical history – available on admission – included previous ductal adenocarcinoma of the breast successfully treated with quadrantectomy in 2007 with regular negative follow-up, asymptomatic primary polycythaemia with regular haematological follow-up and no need of phlebotomy or cytoreductive therapy in absence of splenomegaly, a mild cognitive impairment treated with amitriptyline 25 mg die and duloxetine 30 mg die and a long-term chronic use of aspirin 100 mg in primary prevention.

At admission the patient had a blood pressure of 100/60 mmHg, a heart rate of 110 beats/minute-described as 'thready'-and a respiratory rate of 26 per minute. Her body temperature was normal. Abdominal examination demonstrated guarding and tenderness with normal bowel sounds. Electrocardiogram showed sinus tachycardia with no ischemic changes. Arterial blood gas analysis revealed a metabolic acidosis (pH 7.29, pCO2 31 mmHg, pO2 78 mmHg, HCO3 18 mmol/L, BE – 1.4) with decreased haemoglobin value of 9.1 g/dL, normal glucose (115 mg/dL) and increased lactate (3.9 mmol/L, normal value < 1 mmol/L). Laboratory tests are reported in table 1 and showed neutrophil leucocytosis with a mild increase of C-reactive protein, a moderate microcitic anaemia (haemoglobin 8.3 g/dL, haematocrit 30%, MCV 77.7 fl), normal

liver function and a moderate decrease of the renal function with estimate creatinine clearance of 33.6 mL/min. Urine dip was not performed due to the subsequent rapid deterioration of the patient. Indeed, soon after the medical evaluation, the patient experienced an unbearable abdominal pain, difficulty in breathing, and left shoulder tip pain. She became pale and unconscious, and signs of circulatory shock were evident with a heart rate of 130 beats per minute and a blood pressure of 60/30 mmHg. Peripheral pulses were absent, jugular and femoral pulses were still detectable. She displayed marked tenderness with rebound and guarding on abdominal palpation. Bowel sounds were attenuated. Extended Focused Assessment with Sonography for Trauma (E-FAST) showed normal diameter of the abdominal aorta and minimal perihepatic fluid, raising the suspicion of free peritoneal fluid. No perisplenic fluid was detected but the spleen appeared with non-homogeneus parenchyma. Absence of pericardial effusion, hyperdynamic ventricular systolic function, small IVC with a collapsibility index (CI) of >50%, a normal A pattern with no signs of pleural effusion.

Arterial blood gas analysis in reservoir mask 15 Lt/min showed a metabolic acidosis (pH 7.28 pCO2 41 mmHg, pO2 139.4 mmHg, HCO3 19 mmol/L, BE – 6.7) with a significant drop in the haemoglobin value (7.3 mg/dL) and increased lactate (10.6 mmol/L). The patient was promptly treated with crystalloid infusion (1000 mL) and transfused with 2 blood units of type O RhD-negative. Clinical parameters and blood gas analysis classified an ATLS class 3 haemorrhagic shock.

The patient was promptly intubated, stabilized, and prepared for damage control surgery in the hypothesis of abdominal haemorrhage.

An enhanced contrast CT scan was performed after the patient was stabilized and intubated, and it revealed a macerated spleen with rupture, resulting in generalized haemoperitoneum over the upper abdomen and the pelvis (Figures 1 and 2). A splenectomy was required after the emergency laparotomy revealed a laceration over the spleen, which was the cause of a significant haemorrhage. The patient was moved to the Intensive Care Unit (ICU) after surgery. To rule out any other possible causes of splenic rupture, an infectious disease workup was performed. Nevertheless, serologies for viral diseases (CMV, EBV, HIV, HCV, and HBV) and blood cultures were all negative. Both a serum sample and Bronchoalveolar Lavage (BAL) showed negative EBV PCR results. No atypical lymphocytes or other abnormalities were visible in the peripheral blood smear, ruling out a haematological disorder. A contrast-enhanced total body CT was performed and excluded occult tumors. The removed spleen was 115 gr in weight and 8*7*4.5 cm in size. The superior pole of the spleen was lacerated, giving it a brownish appearance. Histological analysis revealed focal haemorrhagic infarction of the splenic parenchyma, which appeared homogeneous. Following surgery, the blood count gradually returned to normal, culminating in post-trauma reactive leukocytosis (Table 1). Unfortunately, the patient developed a hospital-acquired bloodstream infection and passed away after a lengthy (38 day) stay in the ICU.

After a second evaluation, the patient's daughter revealed that four weeks earlier, the patient had visited another ED complaining of right hip pain following a minor fall in the bathroom. She injured her right thigh after hitting the sink with her left posterior chest wall. No prior history of losing consciousness existed. There was no complaint of injury or abdominal pain. Except for hip pain, medical examinations were unremarkable. While the anteroposterior supine abdominal radiograph revealed a right ilio-pubic branch infraction, the posteroanterior and left oblique chest radiographs both revealed no rib fracture, normal lung fields, and clear costophrenic angles. The patient had been discharged home with oral paracetamol, bed rest and low molecular weight heparin at the dose of 4000 UI daily as antithrombotic prophylaxis.

Discussion

Background

DSR is characterized by splenic rupture that occurs more than 48 hours after injury in a patient who was previously haemodynamically stable. Evans published the first description of DSR in 1902.[1,2] The case of a 63-year-old dustman who passed away five days after suffering a blunt abdominal trauma was discussed by the author. Evans came to the conclusion that the capsule tear most likely happened on the fourth day following the blunt abdominal trauma after reviewing the autopsy report of an intrasplenic haematoma with a capsule laceration. Baudet later suggested the term "latent period" to describe the symptom-free period between injury and DSR in 1907.[3] The author hypothesized that it took at least 48 hours from the injury to the splenic rupture, during which there was a period of clinical quiescence known as "the latent period of Baudet." In 1932, McIndoe described 46 cases of DSR that led to a sudden circulatory collapse after a latent period of at least 48 hours.[4] The term "delayed rupture of the spleen" was suggested by the author as being vague, and he preferred "delayed haemorrhage after traumatic rupture of the spleen" as a more precise and descriptive term for the process.

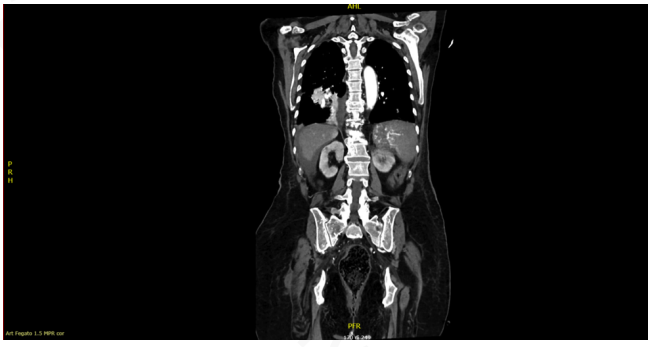

Figure 1. Coronal section of CT Abdomen showing hemoperitoneum and laceration of the spleen.

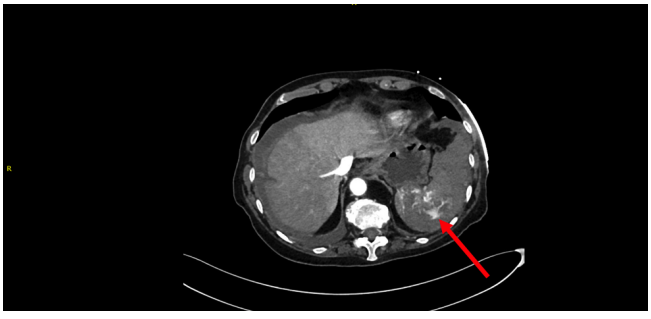

Figure 2. Transverse section of contrast-enhanced CT abdomen showing free fluid (blood) and laceration of spleen (red arrow).

Table 1. Laboratory parameters on admission, after surgical intervention and after 7 days of ICU stay.

			Admission	Post-operative	After 7 days of ICU stay
White blood cells (WBC)	10^9/L	4.8-10.8	51.6	77.1	22.6
Red blood cells (RBC)	10^{12}/L	4.2-5.4	3.86	4.63	3.84
Haemoglobin (Hb)	g/dL	12-16	8.3	11.7	9.5
Haematocrit (HTC)	%	37-47	30.0	36.3	29.5
MCV	fL	81-99	77.7	78.4	76.8
MCH	pg/Cell	27-34	21.5	25.3	24.7
MCHC	g Hb /dL	31-36	27.7	32.2	32.2
RDW	CV%	11.5-14.5	20.2	18.3	17.6
Platelets (PLT)	10^9/L	130-400	353	238	192
Neutrophils	%	40-75	68.0	NA	NA
Lymphocytes	%	20-50	2.0	NA	NA
Monocytes	%	2-15	29.0	NA	NA
Eosinophils	%	1-6	0.0	NA	NA
Basophils	%	0-2	0.0	NA	NA
Neutrophils	10^9/L	1.8-7.7	52.4	NA	NA
Lymphocytes	10^9/L	1-4.8	1.5	NA	NA
Monocytes	10^9/L	0.2-0.8	22.4	NA	NA
Eosinophils	10^9/L	0-0.45	0	NA	NA
Basophils	10^9L	0-0.2	0	NA	NA
Metamielocytes	%	2-10	1	NA	NA
Glucose	mg/dL	60-100	125	105	NA
Blood urea nitrogen	mg/dL	10-50	30	30	NA
Indirect bilirubin	mg/dL	0.01-0.8	0.71	0.60	NA
Total bilirubin	mg/dL	0.1-1	1.69	1.60	NA
Direct bilirubin	mg/dL	0.01-0.25	0.98	1	NA
Sodium	mmol/L	135-148	137.9	144.0	NA
Potassium	mmol/L	3.5-5	7.04	5.00	NA
Calcium	mmol/L	2.1-2.6	2.09	2.52	NA
Albumine	g/L	34-48	22.8	22	NA
Creatine kinase (CK)	U/L	20-170	67	105	NA
Aspartate transaminase (AST)	U/L	5-33	31	131	NA
Alanine aminotransferase (ALT)	U/L	6-41	15	52	NA
C-reactive protein (CRP)	mg/L	<6	69.8	50.9	NA
Troponine	ng/L	<14	36.2	NA	NA
Creatinine	mg/dL	0.5-1.1	1.32	1.13	NA
PT-seconds	Seconds		21.4	NA	17.6
PT-Ratio		0.9-1.18	1.65	NA	1.35
PT-INR -International Normalized Ratio-			1.67	NA	1.37
APTT-Seconds	Secondi		33.9	NA	33.3
APTT-Ratio		0.75-1.29	1.13	NA	1.11
Fibrinogen	mg/dL	150-400	619	NA	NA
D-dimer	µg/mL	0.27-0.77	13.17	NA	NA
CORONA VIRUS-esito		Neg	neg		
Anti- EBV viral capsid antigen (VCA) IgM	IU/mL		0		
Anti-EBV viral capsid antigen (VCA) IgG	IU/mL		223		
Anti- EBV Early antigen (EA) IgG	U/mL		136		
Cytomegalovirus Antibody (IgM)	U/mL	<30	0.13		
Cytomegalovirus Antibody (IgG)	U/mL	<30	181		
CMV DNA serum	IU/mL		neg		
Respiratory viruses in Bronchoalveolar lavage (BAL)					
Influenza virus A			neg		
Influenza virus B			neg		
Rhinovirus A/B/C			neg		
Adenovirus			neg		
Parainfluenza 1			neg		
Parainfluenza 2			neg		
Parainfluenza 3			neg		
Parainfluenza 4			neg		
Metapneumovirus			neg		
Bocavirus			neg		
Respiratory syncytial virus A/B			neg		
EBV DNA			neg		

Since then, there has been some discussion about the proper semantics for the clinical entity of DSR in the literature of emergency and surgical medicine. Dang *et al.* proposed in 1990 that the term DSR should only be used to describe situations in which the spleen's initial imaging was negative and the diagnosis was made 48 hours or more after the injury by surgery or additional imaging. On the basis of this premise, Kluger proposed four DSR categories and provided clinical criteria for each in 1994.[5] Based on the following clinical criteria, category I was referred to as a "true DSR": a history of blunt abdominal trauma, the absence of clinical evidence of intra-abdominal injury (peritonitis), and normal findings on the initial imaging CT scan of the abdomen. As reported in our clinical case, category II was referred to as "delayed presentation of splenic injury" and was connected to a history of blunt abdominal trauma, the lack of clinical evidence of intra-abdominal injury, and the absence of CT imaging at initial evaluation. Clinical deterioration occurs in both of the two categories more than 48 hours after the initial traumatic injury. Category III required early CT documentation of splenic injury, delayed haemorrhage, and the need for surgical intervention. It included DSR as a "failure of non-surgical management of splenic injury." Category IV DSRs were those cases that required a second operation after recurrent haemorrhage followed early surgical failure, such as partial or splenorrhaphy.

However, Kluger's classification seems to ignore the limitations of imaging technology and the realities of clinical practice by assuming that the initial imaging of the spleen must be negative in order for the term "DSR" or "true DSR" to be applied. Nowadays, small splenic capsular tears or subcapsular haemorrhages that first or second-generation scanners might have missed can be found using new spiral CT technology. Thus, the incidence of "true DSR" would have been misrepresented by strict adherence to the classification system suggested by Kluger. In fact, there has been at least one error regarding DSR due to prior technological limitations. In his series from 1931, McIndoe came to the conclusion that DSR accounted for roughly 14% of all splenic ruptures (15 out of 111 cases).[3] This incident has been mentioned numerous times in the surgical literature,[2] but it is probably inaccurate because it includes injuries that are not initially visible on imaging but would be radiographically obvious today and fail non-operative management. Thus, others have more recently discovered that the true incidence is closer to 1% using more sophisticated techniques.[2]

Traumatized patients are examined and given CT scans in many emergency departments within 30 minutes of presentation. Uncertainty exists regarding the impact of this phenomenon, or the use of CT imaging before an injury may be radiographically visible, on the entity and incidence of DSR.

The mortality rate from delayed diagnosis of splenic injury following blunt abdominal trauma ranges from 5% - 15%, in contrast to the 1% mortality rate associated with acute splenic injury.[6,7] The increased presentation of delayed splenic injury is on the rise, which poses a risk for patient morbidity and mortality. This difficulty in identifying DSR can be attributed to a number of factors, including atypical presentations that can mimic other pathologies, the absence of symptoms, or ambiguous imaging.

Regardless of the timing or mechanism of the triggering event, DSR should always be taken into consideration in the differential diagnosis of an acute surgical abdomen due to the variability in presentation and the difficulty with the initial assessment of DSR.[8] For cases of DSR where the medical history does not match the typical presentation, a differential diagnosis with the even more uncommon occurrence known as spontaneous splenic rupture is necessary.[8] A systematic review of 613 cases of splenic rupture without trauma or previously known disease was carried out between 1950 and 2011 by F. Kris Aubrey-Bassler and Nicholas Sowers.[9] The authors showed that splenic ruptures "without trauma or previously diagnosed disease are largely ignored in the emergency literature and are frequently not documented as such in journals from other fields". Splenic rupture was found to be the presenting symptom of an underlying disease in more than half of cases (n = 327). Infections (n = 143), haematologic cancers (n = 84), and non-haematologic cancers (n = 48) were the most frequently associated diseases. Only 35 instances of histologically healthy spleens spontaneously ruptured. In 23 cases, minor trauma was the primary factor leading to rupture, and medication use was a factor in 47 cases. Anticoagulants (n = 21), thrombolytics (n = 13), and recombinant granulocyte colony-stimulating factor (n = 10) were the drugs most frequently linked to ruptured spleens.

Physiopathology

The pathophysiology of DSR is not fully understood. According to Baudet, a direct abdominal trauma to the spleen can cause enough force to lacerate the splenic parenchyma but not to harm the capsule. When intrasplenic bleeding persists, a subcapsular haemorrhage develops, gradually raising the intrasplenic pressure. As a result, there may be a delay of days before the splenic capsule ruptures and causes an intraperitoneal haemorrhage.[3] The most widely held theory is this one. Another hypothesis put forth by Simpson and Ajuwon contends that a perisplenic haemorrhage and capsular tear develop at the time of injury, but are postponed from rupturing by the surrounding organs.[10] According to Hiraide *et al.*, a pseudoaneurysm of intraparenchymal splenic artery branches rupture is another possible cause of DSR.[11] Additionally, a perisplenic haematoma would stop the initial bleeding from a splenic laceration; however, bleeding would start later after the haematoma is dislodged.[10] Another potential mechanism in this situation is the rupture of an asymptomatic splenic pseudocyst that developed following blunt trauma.[11] These uncommon pseudocysts develop after an intrasplenic haemorrhage.

A generic trauma can trigger the innate immune system, according to the physiopathological mechanisms that have been extensively documented in the literature (12).[12] A clinical condition known as systemic inflammatory response syndrome (SIRS) may be brought on by severe trauma that causes extensive tissue damage.[13] It has been shown that early acute respiratory distress syndrome (ARDS) or multiple organ dysfunction syndrome (MODS) after trauma is related to excessive polymorphonuclear cells tissue influx.[14] Another risk factor for post-trauma late septic complications is excessive immune activation.[15] In surgical ICU patients, inflammatory complications cause from 50% to 80% of late mortality.[16,17]

Diagnosis

Based on the patient's haemodynamic condition, the diagnostic method should be chosen at the time of admission to the ED. E-FAST and ultrasonography (US) have replaced diagnostic peritoneal lavage (DPL) management of abdominal trauma in the present, because they are quick and effective at detecting free fluid (17-19).[17-19] Therefore, when patients are unable to undergo an abdomen enhanced CT scan, abdominal US is thought to be the best option for quickly identifying or ruling out intraperitoneal haemorrhage in haemodynamically unstable patients. A positive E-FAST result is an absolute indication for an emergency exploratory laparotomy in patients who are haemodynamically unstable. Studies have shown that for a small fluid amount, the sensitivity can reach 91% and the specificity can reach 96%.[20-21] However,

reports of 42% false negatives have been made.[22] This may be caused by the 20% of cases where there is no significant blood extravasation in splenic trauma or injuries close to the diaphragm.[18] The gold standard for trauma patients who are haemodynamically stable or stabilized is a CT scan with intravenous contrast, that has a sensitivity and specificity of 95%.[18] It is extremely uncommon for DSR to occur when the spleen appears normally on the initial post-trauma CT scan.

The extent of the injury may not have been accurately determined in our patient because only chest and pelvis X-ray were taken during the initial evaluation following the blunt trauma.

Management

Regardless of aetiology, management of DSR can be divided into non-operative and operative, depending on the degree of splenic injury as described by the AAST classification[22] and the patient's haemodynamic stability. Latest WSES classification and guidelines for splenic trauma in adult and paediatric patients divide splenic injury based on both the anatomic AAST-OIS classification (Table 2) and the hemodynamic status (Table 3) into: i) minor (WSES class I); ii) moderate (WSES classes II and III); iii) severe (WSES class IV and V).[23]

Non-operative management (NOM; conservative management and interventional radiology) has become the standard of care for haemodynamically stable patients in grades I to III, with 90% success, whereas injuries of grade IV and V, with evidence of extravasation and haemodynamically unstable patients, require operative management. NOM has been promoted mainly due to overwhelming post-splenectomy infections (OPSI), a serious complication reported in 0.5% of traumatic splenectomies and up to 20% of non-traumatic cases. Usually, OPSI occurs in the first two years after

Table 2. WSES Spleen Trauma Classification for adult and paediatric patients.

	WSES class	Mechanism of injury	AAST	Hemodynamic status[a,b]	CT scan	First-line treatment in adults	First-line treatment in pediatric
Minor	WSES I	Blunt/penetrating	I–II	Stable	Yes + local exploration in SWd	NOMc + serial clinical/laboratory/ radiological evaluation Consider angiography/ angioembolization	NOMc + serial clinical/laboratory/radiological evaluation Consider angiography/ angioembolization
Moderate	WSES II	Blunt/penetrating	III	Stable			
	WSES III	Blunt/penetrating	IV–V	Stable		NOMc All angiography/ angioembolization + serial clinical/laboratory/ radiological evaluation	
Severe	WSES IV	Blunt/penetrating	I–V	Unstable	No	OM	OM

SW: stab wound, GSW: gunshot wound. [a]Hemodynamic instability in adults is considered the condition in which the patient has an admission systolic blood pressure <90 mmHg with evidence of skin vasoconstriction (cool, clammy, decreased capillary refill), altered level of consciousness and/or shortness of breath, or >90 mmHg but requiring bolus infusions/transfusions and/or vasopressor drugs and/or admission base excess (BE) >–5 mmol/L and/or shock index >1 and/or transfusion requirement of at least 4–6 units of packed red blood cells within the first 24 h; moreover, transient responder patients (those showing an initial response to adequate fluid resuscitation, and then signs of ongoing loss and perfusion deficits) and more in general those responding to therapy but not amenable of sufficient stabilization to be undergone to interventional radiology treatments. [b]Hemodynamic stability in paediatric patients is considered systolic blood pressure of 90 mmHg plus twice the child's age in years (the lower limit is inferior to 70 mmHg plus twice the child's age in years, or inferior to 50 mmHg in some studies). Stabilized or acceptable hemodynamic status is considered in children with a positive response to fluid resuscitation: 3 boluses of 20 mL/kg of crystalloid replacement should be administered before blood replacement; positive response can be indicated by the heart rate reduction, the sensorium clearing, the return of peripheral pulses and normal skin colour, an increase in blood pressure and urinary output, and an increase in warmth of extremity. Clinical judgment is fundamental in evaluating children. [c]NOM should only be attempted in centers capable of a precise diagnosis of the severity of spleen injuries and capable of intensive management (close clinical observation and hemodynamic monitoring in a high dependency/intensive care environment, including serial clinical examination and laboratory assay, with immediate access to diagnostics, interventional radiology, and surgery and immediately available access to blood and blood products or alternatively in the presence of a rapid centralization system in those patients amenable to be transferred.

Table 3. Spleen injury scale (1994 revision).[17]

- Grade I
  - subcapsular hematoma <10% of surface area
  - parenchymal laceration <1 cm depth
  - capsular tear
- Grade II
  - subcapsular hematoma 10-50% of surface area
  - intraparenchymal hematoma <5 cm
  - parenchymal laceration 1-3 cm in depth
- Grade III
  - subcapsular hematoma >50% of surface area
  - ruptured subcapsular or intraparenchymal hematoma ≥5 cm
  - parenchymal laceration >3 cm in depth
- Grade IV
  - any injury in the presence of a splenic vascular injury or active bleeding confined within splenic capsule
  - parenchymal laceration involving segmental or hilar vessels producing >25% devascularisation
- Grade V
  - shattered spleen
  - any injury in the presence of splenic vascular injury* with active bleeding extending beyond the spleen into the peritoneum

surgery with a high mortality rate of 50% – 80%.[24] Lower hospital costs, avoidance of intra-abdominal complications and the maintenance of the immunological function of spleen are the advantages of NOM over splenectomy. On the other hand, despite the high rates of success with NOM in patients with low-grade splenic injuries, there should be a concern that current practice may develop increased morbidity and mortality in patients presenting with complications from NOM, such as haematoma, splenic pseudocyst, sepsis and DSR. A systematic review by Olthof *et al*. showed prognostic factors of NOM failure were age of 40 years or above, Injury Severity Score (ISS; Table 4) of 25 or greater, and splenic injury grade of 3 or greater, as well as transfusion of 1 or more unit of packed red blood cells.[25] In order to improve the success of NOM, clinicians must consider the following factors: admission to ICU/floor, frequency of monitoring of haemoglobin/haematocrit, vital signs, abdominal examination, resumption of diet and ambulation, repeat imaging, and length of stay.[26] Although follow-up CT scan is not routinely planned, proper discharge instructions and conditions for follow-up must be given to the patient, including laboratory observation and activity restriction may be suggested for 4–6 weeks in minor injuries and up to 2–4 months in moderate and severe injury.[27]

Despite the favorable results following ongoing monitoring in patients with the NOM of splenic injuries, a survey of medical professionals revealed that 88% of clinicians do not advise post-discharge imaging for patients who are being treated conservatively.[28,29] We are aware that patient compliance, coexisting conditions, and radiation exposure may make serial CT surveillance impractical and ineffective. On the other hand, there are not many cases of a normal spleen appearing to rupture on its own.[28] However, it is possible that in each of these cases, the history of a minor trauma was overlooked. Therefore, it is imperative to obtain a thorough history of any major or minor trauma that the patients experienced in the weeks before they presented with abdominal pain.

The use of serial CT scans as an adjunct to the NOM of splenic injury is a point of contention in the management of suspected splenic insults. Weinberg *et al.* reported that patients with pseudoaneurysms, who underwent conservative management with serial CT surveillance, had a high splenic salvage. They advocated for serial CT scans in all patients undergoing NOM for splenic injury, regardless of severity.[30] However, EAST guidelines found no evidence to support routine imaging (CT or ultrasound) in improving patients' haemodynamics and clinical outcomes.[31] As a result, alternative considerations should be given to ensure prompt diagnosis and effective DSR management, both acutely and long-term. It should be noted that Lui *et al.* compared the effectiveness of operative and non-operative management of splenic ruptures, concluding that post-splenic rupture quality of life was comparable, though patients who underwent NOM had longer hospital stays.[32]

Table 4. The Injury severity score (ISS) ranges from 0 to 75, and escalates as severity increases. If an injury is assigned with a score of 6 (incompatible-with-life injury) an ISS of 75 is automatically established.

Head and neck worst injury	
Face worst injury	
Chest worst injury	
Abdomen worst injury	
Extremity (including pelvis) worst injury	
External worst injury	
No injury	0
Minor	1
Moderate	2
Serious	3
Severe	4
Critical	5
Unsurvivable	6

Conclusions

DSR is a rare case following major traumatic events and even rarer following trivial trauma.

In our case, four weeks after a minor fall, the patient developed signs and symptoms of an acute intraperitoneal haemorrhage. Our patient experienced abdominal pain, difficulty breathing, and signs of haemorrhagic shock after the intraparenchymal haematoma ruptured and blood leaked into the peritoneal cavity.

Following stabilization and orotracheal intubation, our patient underwent a CT scan, as requested by the surgeons. An extensive panel of blood cultures and serologies for viral diseases (Table 1), the absence of cellular inclusions at the histological examination of the spleen, and a normal peripheral blood smear ruled out underlying infectious and haematological diseases. As a result, DSR was the final diagnosis.

DSR diagnosis can be difficult for even the most experienced emergency clinicians because it can mimic several other medical emergencies. The presumed triviality of the precipitating injury, an unpredictable time lag between the injury and the development of symptoms, and the possibility of atypical signs and symptoms distant from the bleeding spleen may make diagnosis difficult.

Given that not every patient with blunt abdominal trauma presents to the ED for evaluation, and that not every trauma requires a CT abdomen, it is possible that cases of spontaneous atraumatic splenic rupture are actually underdiagnosed DSR.

The most important aspect to be investigated by emergency clinicians when approaching a patient is a detailed history of any recent abdominal blunt injury, regardless of time and mechanism of the inciting event, or a history of NOM for past splenic trauma. Based on the concept that the patient assessment always begins with a complete and detailed anamnesis, a proper collection of the patient's medical history could lead to a prompt diagnosis of DSR.

The main goal of our work is to highlight the critical role of emergency clinicians in the early diagnosis of DSR, particularly after minor or trivial trauma that cannot be reported by the patient at the initial evaluation, as happened in our case report. Given the scarcity of data and the availability of evidence reported in the literature, which is mostly limited to case reports, we believe that additional research is needed to modify existing recommendations for the initial and subsequent management of splenic injuries that may be fatal in the long run.

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