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# **Non-Operative Management of Delayed Splenic Rupture 4 Months Following Blunt Abdominal** Trauma

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	Patient: Final Diagnosis: Symptoms: Medication: Clinical Procedure: Specialty:		Female, 32-year-old Delayed splenic rupture Abdominal pain • chest pain • dyspnea — — Surgery	
Objective: Background:			<b>Challenging differential diagnosis</b> Delayed splenic rupture is a rare complication of non-operative management of a primary splenic trauma which, without proper clinical vigilance, may result in life-threatening events. It usually occurs 4-8 days after injury and, in most cases, surgery is the treatment of choice. Since non-operative management of splenic trauma, which allows splenic salvage, has become increasingly popular, the same approach could also be applied in delayed splenic rupture. We herein present a case of delayed splenic rupture that occurred 4 months after the trauma and was successfully managed non-operatively.	
Case Report: Conclusions: Keywords:		eport:	A 32-year-old woman presented with diffuse abdominal pain, chest pain, and dyspnea 4 months after sustain- ing minor thoracoabdominal blunt trauma due to a car accident. That event was inadequately investigated and was not admitted for further monitoring. Computerized tomography revealed a rupture of a splenic hemato- ma in the context of the previous splenic trauma. She was closely monitored and remained hemodynamically stable. She was discharged and followed up, with no reported relapse of her clinical condition. Delayed splenic rupture occurring 4 months after the primary splenic trauma is extremely rare. Due to its pro- longed delay, delayed rupture of the spleen can easily be overlooked and not be included in the original differ- ential diagnosis. Negligence of this event can result in dreaded complications with hemodynamic instability or even death. Furthermore, its higher mortality rate compared to primary splenic rupture highlights the impor- tance of proper clinical vigilance. Non-operative management should be attempted in hemodynamically stable patients. <b>Abdominal Injuries • Spleen • Splenic Rupture</b>	
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## Background

The spleen is one of the organs most commonly injured in acute blunt trauma. Splenic injury accounts up to 50% of all abdominal solid organ injuries [1]. Recognition of the important immunological function of the spleen renders its salvage of great significance. Whereas splenectomy has historically been the therapeutic approach of choice, in hemodynamically stable patients, non-operative management (NOM) has, during the last decades, become the standard of care when blunt splenic injury occurs. Success rates of NOM can reach >90%, completely shifting the approach to splenic injuries, regardless of the American Association for the Surgery of Trauma (ASST) score [2]. Operative management is currently reserved for hemodynamically unstable patients or in the case of concomitant intra-abdominal injury, which require surgical intervention [3]. NOM requires observation and monitoring, with or without supplementation with splenic artery embolization (SAE). These patients are admitted to a medium or intensive care unit, with close monitoring of vital signs, bed rest, consistent monitoring of hemoglobin concentration, and serial abdominal examinations [3]. However, failure of NOM occurs in 4-15% of cases [2]. Delayed splenic rupture (DSR), ongoing bleeding, and the formation of pseudoaneurysms are the potential risks of NOM. The incidence of DSR is approximately 1-2%. DSR is usually expected during the first 4-8 days after the primary injury. The longest period between trauma and delayed splenic rupture, based on published literature, was 70 days [4], making the present case, in which DSR occurred 4 months after the trauma, the longest time period reported between primary trauma and definitive trauma.

## **Case Report**

A 32-year-old woman presented with diffuse abdominal pain, chest pain, and dyspnea 24 h after insignificant activity, which also resulted in an episode of loss of consciousness. The patient reported a visit to the emergency department of another institution 4 months prior to admission because of a car accident in which she sustained minor thoracoabdominal blunt trauma. Results of a physical examination and laboratory and radiology (chest X-ray) work-up during that visit were reported to be normal. No report of previous work-up was provided by the patient and no CT examination was registered at the other institution. She was then discharged without hospital stay for observation. Vital signs upon presentation were normal: blood pressure of 100/60 mmHg, pulse of 71 beats/min, respiratory rate of 25 breaths/min, and oxygen saturation of 93% on room air, and the patient was alert and oriented. Physical examination revealed tenderness in the left lower thorax - left subcostal area. Laboratory tests revealed only elevated D-dimers (5257.3 µg/dL), 35% hematocrit, and hemoglobin 12.1 g/dL.

Cardiac work-up was immediately requested, including laboratory examination and ECHO, which was negative for cardiac events. Due to strong clinical suspicion of pulmonary embolism, based on the reported chest pain with dyspnea, the patient was immediately referred for CTA of the thorax. The decision was made to include the abdomen on the CT examination due to the pain on the left lower thorax and upper abdomen. So, in contrast to the usual work-up used in cases of right-left lower thorax or abdominal pain, to save time, ultrasound was not performed. Computed tomography (CT) of the thorax and abdomen revealed the presence of a small laceration (around 2 cm in depth) on the posterior surface of the lower pole of the spleen, classified as grade 2 injury according to the AAST Splenic Trauma Classification [2] along with hemorrhage fluid located around the spleen and liver, in the paracolic gutters, and in the pelvis, findings strongly suggestive of delayed rupture of a splenic hematoma in the context of a previous splenic trauma (Figure 1). The patient was admitted to the surgical high-dependency unit and was closely monitored. Repeat hematocrit counts were performed every 12 h, but no blood transfusion was necessary. During the observation period, the patient was hemodynamically stable with gradually increasing hematocrit. Serial CT scans were preferred to abdominal ultrasound because follow-up is more accurate and less operator-dependent. They were performed on the second (Figure 2) and the fourth day of observation (Figure 3), which showed the gradual decrease of the hemorrhagic fluid, while the second redisplayed the small rupture of the spleen, but with no blood present in the peritoneal cavity. She reported that the abdominal pain was gradually alleviated, as well as the tenderness in the left lower thorax, and she said she felt relieved. After 9 days of hospital stay, she was discharged free of pain. Upon follow-up, a CT 1 month after the episode showed complete healing of the splenic rupture (Figure 3).

#### Discussion

DSR was first described by Dr. Baudet in 1907 and was named the "latent period of Baudet" [5]. He defined DSR as rupture of the spleen at least 48 h after the trauma, with a previous asymptomatic period [5]. Cases of DSR were reported by Sizer et al in 1966; 80% occurred within 14 days and 95% within 21 days [6]. At that time, DSR referred to the delayed diagnosis of splenic injury leading to rupture. After CT was introduced in clinical practice, DSR significantly decreased, from 5-40% to 1-2% [7]. The decrease in incidence lies in the fact that through the availability of CT scans, the condition is rapidly diagnosed, allowing prompt management of patients. Reports regarding the sensitivity and specificity of CT scans for evaluation of possible splenic injuries have been reported by many to be as high as 96% and 100%, respectively [8]. The first case of DSR documented by CT scan was described in 1981 by Toombs et al [9].



Figure 1. Contrast-enhanced CT at admission. (A) Coronal plane. Hemorrhage fluid located around the spleen and liver (white arrows), in the paracolic gutters (white arrow), and the pelvis. (B) Axial plane. Perisplenic and perihepatic hematoma (black arrows).



Figure 2. Contrast-enhanced CT 24 h after admission. (A) Coronal plane. Gradual decrease of the perisplenic and perihepatic hematoma. (B) Axial plane. A small rupture of the posterior surface of the lower pole of the spleen (black circle) is shown accompanied by perisplenic hematoma (white arrow).

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Figure 3. Contrast-enhanced CT, axial plane. (A) On the 6<sup>th</sup> day of admission. The rupture of the spleen (white arrow) and the decreasing size of the perisplenic and perihepatic hematoma are shown. (B) Follow-up 1 month after the admission. Complete healing of the splenic rupture, with no presence of intraperitoneal fluid (black circle).

The exact mechanism of DSR is unclear. Several theories have been introduced to explain its occurrence. Many authors propose that perisplenic hematoma is formed as a result of a tear of the splenic capsule tamponaded by the omentum or surrounding organs, which later rupture into the peritoneal cavity [10]. Others postulate that injury of the splenic artery or vein can lead to the formation of an intraparenchymal pseudoaneurysm or splenic pseudocyst. Its irritation, growth in size and the possible rupture can develop delayed clinical symptoms [10,11]. A third theory proposes that a clot lysis or an expanding subcapsular hematoma formed after the traumatic event increases tension within the splenic capsule at the risk of rupture [10,11]. False-negative CT results during initial radiological evaluation of the spleen after trauma can contribute to delayed recognition of splenic injury. Minor contusions of the spleen, poor imaging quality, or artifacts created by the left ribs can conceal splenic injuries [12].

The clinical presentation of DSR is highly dependent on the severity of splenic injury, ranging from mild abdominal pain with normal vital signs to critically ill patients with signs of hemodynamic compromise [11]. Kehr's sign or Balance's sign can be elicited on physical examination [11].

A multicenter study by Harmon et al found the incidence of DSR was 0.4% [12]. However, in nearly half of the patients, DSR occurred in the first 48 h, in contrast to the original definition of DSR [12]. In that multicenter study, in 25% of patients, DSR occurred 48 h to 5 days after trauma, while it occurred after 7 days in only 6% [12]. A study by Liu et al sets the mean latent period of DSR at 18 days, with 58 days being the longest period between primary splenic trauma and occurrence of DSR [13]. Most patients report a history of accident

with high-injury mechanism, such as car/motorcycle accidents, causing no or minor splenic injury, and appear with a relative hemodynamic stability, as in our case. In the majority of cases, the timing of delayed post-traumatic splenic rupture has been reported to be 4-8 days after trauma [4,10,14,15]. Based on existing reports, the longest latent period was 70 days after trauma [4]; the patient presented with intense pain at the left lower thorax-left subcostal area and reported a skiing injury, with primarily minimal splenic contusion. In our case, the patient presented with the same symptoms, but DSR occurred 4 months after the primary trauma, significantly later than in previous reports in the published literature. In addition, considering that our patient was an otherwise healthy individual and reported no other injuries apart from the original car accident, we assumed that the occurrence of another mechanism of splenic rupture was highly improbable. According to Savage et al, who retrospectively analyzed a cohort of 637 patients with blunt splenic injury, approximately 20% had not documented total rupture healing within the first 3 months after the trauma [14]. DSR should be taken into consideration in the differential diagnosis, regardless of a prolonged period of time that could have elapsed after the abdominal trauma.

The importance of DSR lies in its higher mortality rate (5-15%) compared to that associated with acute splenic injury (1%) [7]. The higher mortality rate is attributed to the poor selection of patients for NOM, incorrect choice of treatment, and absence of clinical suspicion of DSR.

A number of prognostic factors regarding the failure of NOM have been investigated. A multicenter study by Peitzman et al indicated that failure of NOM was significantly affected by the grade of splenic injury, the Injury Severity Score (ISS), and the

hemodynamic status of the patient [16]. Specifically, the failure rate of NOM increased with grade of injury, from 4.8% for patients with American Association for the Surgery of Trauma grade I injury, 9.5% for grade II, 19.6% for grade III, 33.3% for grade IV, and 75.0% for grade V, and patients with an ISS >15 were significantly more likely to undergo surgery and have an eventful course if non-operative management was attempted [16]. Higher failure rates with increasing grade of splenic injury were also found by Chastang et al [17] in a prospective, multicenter study. Moreover, age greater than 55 years has proved to be an increased risk of non-operative management failure in a series of studies [18-21]. In a systematic review by Olthof et al, failure of NOM was associated with high-grade splenic injury grade (AAST grade  $\geq$ 3), Injury Severity Score  $\geq$ 25, and age  $\geq$ 40 years [3]. However, delay of operative management does not seem to affect mortality, with a 6.4% mortality rate in failure of NOM versus 16.4% in patients with immediate splenectomy [3].

Although DSR is considered a complication of NOM, it should not dissuade clinicians from conservative treatment, as nonoperative management has a success rate of 83% and should be protocol-guided [13,22]. It is important to note that application of NOM should only be attempted in properly equipped centers, with access to vigorous monitoring and/or intensive care environment, precise imaging technology, interventional radiology (in case of proceeding to SAE), surgical theaters readily available at any moment, and, more important, blood and blood products available for further support [2].

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## Conclusions

In conclusion, DSR remains a dreaded complication and an unpredictable event. High clinical suspicion is required in the presence of hemodynamic instability or simple abdominal pain, especially when a history of trauma is reported, even if there was a prolonged time after trauma. Precise treatment is pertinent and should be followed based on guidelines of primary splenic injury and in well-equipped medical centers. Surgical intervention may be necessary at any moment should the patient deteriorate. Therefore, non-operative management of DSR must be considered as an option only at institutions that offer 24-h surgical theater availability.

#### **Conflict of interests**

None.

#### **Declaration of Figures Authenticity**

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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