

Review

Delayed Splenic Rupture Following Blunt Abdominal Injury and Its Surgical Implications

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Abstract

Spleen is the most common injured organ following blunt abdominal injury. Splenic injury may remain asymptomatic until delayed splenic rupture (DSR) occurs, which is a relatively rare complication. DSR mortality rate is also relatively high, mainly due to missed diagnosis or misdiagnosis. Treatment of DSR is classified into non-operative management (NOM) and surgical interventions; however, there are no clear guidelines in which approach is preferred in which situation. Thus, the aim of this review is to discuss the pathology, diagnosis, and treatment of delayed splenic rupture, highlighting its surgical implications. Multiple pathophysiological theories have been introduced to explain DSR such as delayed rupture of splenic sub-capsular hematoma, clot lysis, and rupture from a pseudoaneurysm. Ultrasound and computed tomography (CT) can be used in the diagnosing process of DSR; however, CT is considered the gold standard. NOM is useful for hemodynamically stable patients with splenic injury; however, it has been associated with various latent complications necessitating a surgical intervention eventually. Surgical intervention (e.g. splenectomy) proved its effectiveness in treating DSR. Current knowledge is largely derived from case reports, highlighting the need for larger, prospective studies to establish evidence-based guidelines for optimal management.

Keywords: *Delayed Splenic Rupture, Splenic Injury, Blunt Abdominal Injury, Non-operative Management, Surgical Intervention*

Introduction

Spleen is a commonly injured organ following abdominal trauma, as the incidence of splenic injury is as high as 40% to 50% among all types of abdominal trauma (1). Delayed splenic rupture (DSR) is a rare complication of abdominal trauma and is defined as the rupture of spleen days after abdominal trauma (2). High-grade splenic injury and DSR cases are mainly reported after major traumatic incidents, such as fall from height, road traffic accidents, or contact sports (3). DSR etiology has been always linked to subcapsular hematomas, splenic pseudocysts, parenchymal pseudoaneurysms, and rib fractures (4, 5). Typically, it presents with hemorrhagic shock based on the degree of injury (6).

The mortality rate from acute splenic injury is about 1%, while the DSR mortality rate is between 5%-15%, which is disproportionately higher. This high mortality of DSR is attributed to the misdiagnosis or the missed diagnosis due to the asymptomatic latent period (2, 7-10). Therefore, it is critical to keep a high level of suspicion about the potential presence of DSR, and immediate diagnosis and management are important to reduce the morbidity and mortality associated with splenic hemorrhage (2, 10). The gold standard for DSR diagnosis is a computed tomography (CT) scan with IV contrast; however, ultrasound can have a role in diagnosing it (6). Treatment of splenic injury and DSR has been debatable in recent studies. Multiple case reports reported various effectiveness of both non-operative management (NOM) and surgical intervention (3, 4, 11, 12).

The aim of this review is to explore current evidence focusing on DSR following blunt abdominal injury, its etiology, pathogenesis, diagnosis, and treatment, highlighting its surgical implications.

Methods

A comprehensive literature search was conducted in Medline (via PubMed), Scopus, and Web of Science databases up to May 5, 2025. Medical Subject Headings (MeSH) and relevant free-text keywords were used to identify synonyms. Boolean operators

(AND', OR') were applied to combine search terms in alignment with guidance from the Cochrane Handbook for Systematic Reviews of Interventions. Key search terms included: "Delayed Splenic Rupture" OR "Splenic Rupture" OR "Splenic Injury" AND "Blunt Abdominal Injury" OR "Blunt Abdominal Trauma" OR "Blunt Injury" OR "Blunt trauma" AND "Surgical Intervention" OR "Surgical Implications". Summaries and duplicates of the found studies were exported and removed by EndNoteX8. Any study that discusses the surgical implication in the treatment of DSR and published in peer-reviewed journals was included. All languages are included. Full-text articles, case series, case reports, and abstracts with the related topics are included. Comments, animal studies and letters were excluded.

Discussion

Pathology of DSR

DSR typically occurs secondary to trauma mainly a minor or trivial trauma (10, 13) such as cough, vomiting, mild hurt, slowly slipping to the floor, suddenly sitting up (14), low energy trauma (13), or colonoscopy procedure (15). DSR can also result following abdominal blunt trauma due to car accident, motorcycle accident or falling. In such cases, splenomegaly (2), conditions such as pregnancy, parturition or defecation (16), and underlying malignancy or infective pathology such as mononucleosis and lymphoma (10, 14, 17, 18) may predispose to a delayed rupture.

Although multiple pathophysiological theories have been introduced to explain DSR, the exact mechanism of it is still unclear (2, 19, 20). These theories include delayed rupture of splenic subcapsular hematoma, clot lysis, and rupture from a pseudoaneurysm. The theory of delayed rupture of splenic subcapsular hematoma starts with a splenic parenchymal injury without laceration of spleen capsule due to trauma (2, 7, 19, 21). Then a subcapsular hematoma would form due to persistent intrasplenic bleeding. The progressive increase of intrasplenic pressure may result in burst of the capsule and subcapsular hematoma rupture. This

results in intra-abdominal hemorrhage multiple days following the initial injury.

Another theory is clot lysis which can lead to progressive elevation of the intrasplenic colloid osmotic pressure. Thus, subcapsular pressure increases, leading to rupture of the capsule and the subcapsular hematoma (9, 20). The rupture of perisplenic hematoma into peritoneal cavity days following trauma as a result of compression by the surrounding organs and tamponade (7, 9, 19, 21), traumatic-induced inflammation may lead to the adherence of the greater omentum to the splenic capsule, leading to maceration of the capsule and uncontrolled hemorrhage (7, 22), and the injury of the splenic capsule and parenchyma by the edges of rib fractures due to a blunt trauma after patient mobilization all are examples of theories introduced in previous studies (16, 17, 23). A potential mechanism of DSR is the rupture from a pseudoaneurysm of intraparenchymal splenic artery branches or rupture from an asymptomatic splenic pseudocyst that may occur after the formation of an intrasplenic hematoma (9, 19).

Diagnosis of DSR

In the early 1900s, Baudet described the DSR as when patients who suffer blunt trauma but exhibit no signs of hemodynamic instability or other clinical symptoms for more than 48 hours following the initial injury (7, 16). The condition is marked by an initial absence of clinical symptoms, followed by DSR. This symptom-free interval is commonly known as the 'latent period of Baudet' (7).

DSR usually occur in 4 weeks following trauma as 90% of cases occur in this period (15, 22), while 75%–80% of cases occur in the first 2 weeks after injury (15, 24). However, it may be remarkably delayed. For instance, it occurred 5.5 years following NOM of traumatic splenic injury in a case reported by Deva and Thompson (22).

The presentation of DSR is atypical and has to be differentiated from various abdomen, chest, and musculoskeletal system diseases (14, 25). The manifestations of DSR are mainly left upper abdominal pain and tenderness, rebound tenderness,

Ballance's sign, and Kehr's sign (2, 10). A meticulous evaluation of DSR risk should be performed before selecting NOM for these patients even in the absence of any signs of it due to the seriousness of this complication (9).

Patients with DSR usually have normal vital signs and the occurrence of hemorrhagic shock in these patients is uncommon due to various mechanisms such as (1) the probability of subcapsular bleeding, thus keeping the bleeding under the splenic capsule which slows the hemorrhage, (2) a hematoma may form in some cases due to local coagulation and adhesion and this hematoma may be temporarily stable; however, it could resume bleeding if the patient experiences further trauma or engages in vigorous physical activity, (3) the rupture may be minimal, leading to slow and minimal bleeding (1).

After a blunt abdominal trauma and prolonged absence of overt symptoms, patients and physicians may consider the atypical abdominal pain as unrelated to the injury. Furthermore, atypical pain in the left hypochondrium area can also occur due to respiratory and circulatory system injuries, musculoskeletal injuries, or liver damage, resulting in more obscuring splenic injury. This may lead to delays in seeking medical care or result in NOM without imaging, which can potentially aggravate the condition.

Imaging in DSR

B-ultrasound and CT imaging are the main diagnostic methods used to diagnose DSR. Each method is used in specific situations and has advantages over the other. Ultrasound is cost-effective, more rapid, can be performed at bedside, and associated with less side effects. While CT can visualize more details and detect the extent of splenic rupture, it is also more suitable for patients whose transport is difficult. Therefore, once CT-related contraindications are excluded, it should be prioritized over ultrasound (1).

CT has shown high specificity and sensitivity in detecting splenic injury and in diagnosing DSR (23, 26) with 100% sensitivity detection of DSR (7), making CT the preferred method than ultrasound

and angiography. It was stated that the accuracy, specificity, and sensitivity of CT in diagnosing blunt abdominal injury are 97%, 100% and 85%, respectively (2, 10, 15, 23, 24, 27). Furthermore, CT proved effectiveness in reducing the incidence of unnecessary exploratory laparotomy for splenic injury (28). It also can be used in grading blunt splenic injury and in detecting and quantifying hemoperitoneum (28). Leeper et al. suggested that CT should be repeated after 48 hours of a sentinel DSR rather than 7 days as this has shown a 10% rate of progression/worsening of splenic injury (29).

It was also suggested that even in unexplained abdominal pain cases without a history of blunt trauma and negative ultrasound findings should also receive abdominal CT scan to exclude any splenic injury (8). Additionally, CT scans can detect posttraumatic splenic lesions associated with failed NOM, such as parenchymal pseudoaneurysms, subcapsular hematomas, and splenic pseudocysts (23). They are also effective in identifying features of DSR, including splenic abscesses and post-traumatic splenic artery pseudoaneurysms. However, early CT scans may miss splenic injuries if performed before a subcapsular hematoma forms or becomes large enough to detect, potentially leading to false reassurance (9). Therefore, regular follow-up CT scans in high-risk abdominal trauma patients are recommended for early DRS detection.

Treatment of DSR

NOM and surgical intervention are the main approaches for treatment of both blunt splenic injury and DSR. Multiple studies have examined the effectiveness and safety of both approaches. NOM is considered the gold standard for hemodynamically stable patients with splenic injury, primarily due to its ability to preserve the spleen's immunological and hematological functions (10, 21, 23). The increased awareness of post-splenectomy infections and the advancement in imaging techniques have contributed to the preference for NOM techniques, including splenic arterial embolization, in the treatment of splenic injuries (22, 30). NOM has been adopted by many physicians and has shown positive results in blunt splenic injuries (9, 11, 31); however, there remains

a lack of standardized NOM protocols across surgeons and institutions (29).

Furthermore, implementing NOM in DSR cases is controversial. The limited regenerative capacity of splenic tissue and the high risk of ongoing bleeding or infection often render conservative strategies ineffective (1). NOM has been associated with various complications such as DSR, splenic abscesses, septicemia, splenic vein thrombosis, infarction, and other abdominal complications, highlighting the risks associated with unjustified or poorly monitored conservative approaches (30, 32, 33).

Failed NOM has been associated with a 4% mortality rate, highlighting the importance of accurate initial assessment (33). Mahon and Sutton reported that 73% of patients who initially received NOM needed surgery eventually due to delayed hemorrhage (34), while Ward and Gillatt reported a splenectomy rate of 73% in cases of DSR (35). Furthermore, the NOM of DSR has shown failure rates ranging from 10% to 33%, based on injury severity and institutional practices (20, 36). Notably, patients with grade III or IV splenic injuries are at increased risk of NOM failure and associated complications and negative outcomes compared to those receiving early surgical intervention (30, 36).

Surgical Implications

Recently, Zhang et al. outlined the following indications for emergency surgical intervention in patients with DSR (1): (1) Patients with hemodynamic instability, at risk of shock, or failure of NOM in improving vital signs. (2) Severe damage of spleen, along with other organ damage, shown by imaging examinations such as ultrasound and CT. (3) Persistent bleeding in the abdominal cavity shown by imaging examination. (4) Splenic hilar laceration, splenic center rupture, or extensive tissue devitalization. (5) Unexplained worsening symptoms, such as worsening abdominal pain. (6) History of spleen disease.

They reported 12 cases with DSR in which all patients eventually underwent surgery and were

discharged in good health, indicating the effectiveness of surgery over NOM in managing DSR (1). Pucci et al. reported a case of DSR who underwent a total laparoscopic splenectomy post-embolization and demonstrated its feasibility (36). Additionally, Ward and Gillatt reported that DSR in adults usually requires urgent laparotomy and splenectomy in up to 73% of cases (35).

However, splenectomy is associated with different side effects, including worse immune function, worse hematological function, and gastrointestinal side effects. Splenectomy reduces immune defense against encapsulated organisms, particularly in children. However, other immune organs, including the liver and lymph nodes, may partially compensate for this immunological loss. It also can lead to alterations in the blood system, since spleen destructs the abnormal or aged red blood cells and stores platelets. Therefore, after splenectomy, hematological abnormalities such as hemolytic anemia or polycythemia may occur as well as blood coagulation and the risk of thrombosis increases (37). The impact of splenectomy on digestive system is attributed to the location of spleen adjacent to the digestive system (38). Symptoms like dyspepsia, gastroesophageal reflux, appetite loss, bloating, diarrhea, or constipation may occur. These symptoms are typically temporary; however, some patients may experience them for a longer period. During the early postoperative period, patients should carefully adjust their diet and avoid consuming oily, spicy, or other irritating foods.

Conclusion

DSR is a rare complication that occurs following blunt abdominal injury; however, a potentially fatal one, usually presenting after a symptom-free interval. Accurate and timely diagnosis is critical, with CT imaging considered the diagnostic gold standard. While NOM is effective for hemodynamically stable patients, DSR often necessitates surgical intervention due to the high risk of delayed hemorrhage and failure of conservative strategies. Importantly, current evidence is largely derived from case reports and

small case series, highlighting the need for larger, prospective studies to establish evidence-based guidelines for optimal management.

Disclosures

Author contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Ethics statement

Non-applicable.

Consent for publications

Not applicable.

Data availability

All data is provided within the manuscript.

Conflict of interest

The authors declare no competing interest.

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