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Spontaneous Splenic Rupture in A Non-Hodgkin Lymphoma Patient, A Rare Case Report

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Abstract

Splenic rupture with no history of previous trauma is known as spontaneous splenic rupture or atraumatic splenic rupture. Most patients have high mortality rates due to missed or delayed diagnosis. In this case report, a 41-year -old male patient who presented with jaundice developed a spontaneously ruptured spleen. One of the misleading clues to this patient's clinical situation was the previous free surgical and medical history with no known predisposing factors at the time of splenic rupture diagnosis.

Keywords: *Spleen, Non-Hodgkin, Lymphoma, Spontaneous, Rupture.*

Introduction

The spleen is the largest lymphoid organ in the body; it plays an essential role in immunological defenses [1]. Commonly, splenic rupture is attributed to thoracic and abdominal blunt trauma [2]. However, atraumatic splenic rupture (ASR) may occur in 3% of total splenic rupture cases, making it a rare but fatal clinical entity.[3] The high mortality and morbidity rate of spontaneous splenic rupture is due to the delay of diagnosis and treatment as it is challenging to discover early [2,4]. Ultimately, ASR is almost always due to an underlying pathology. For example, bacterial and viral infectious diseases such as Epstein-Barr virus infection, neoplasms, connective tissue disorders, some drugs, i.e., cocaine, and inflammatory diseases such as pancreatitis, may cause spontaneous splenic rupture [3]. Among the neoplastic diseases, hematologic malignancies account for the largest group [1]. Splenic rupture is mainly caused by trauma. Nevertheless, it can also occur in some rare cases without obvious trauma, known as atraumatic splenic rupture (ASR) or spontaneous spleen rupture [4]. ASR is often life-threatening due to the delay of diagnosis and treatment [4]

Case presentation

A 41-year-old man presented complaining of scleral yellowish discoloration for one week prior to presentation. This condition started gradually and progressively increased over seven days with two-time epistaxis, anorexia, fatigue, malaise, abdominal pain, and pruritus. The patient was vitally stable by physical assessment, but jaundice, hepatomegaly, and splenomegaly were detected, along with bilateral lower limb edema. Not to mention the presence of altered urine and stool color in which urine became dark while stool became white. On the other hand, the patient denied having episodes of fever, petechiae, ecchymosis, haematochezia, or melena. As a result, our patient was admitted to the medical ward for further evaluation and assessment of these findings.

Investigations and Management

On basic blood work, low HB 10.6 (13.2-17.3) and low PLT count 55 L (150-440), PCV 29.1 (39-49) and MCV 59.6(80-99), WBC 7 was found with elevated liver transaminases. Direct Coombs test showed cold agglutinin weakly positive (anti c3d) and High INR.

Consequently, oral prednisolone, intravenous immunoglobulin (IVIG), and a therapeutic dose of folic acid were commenced for two days to improve the

platelet count. However, during this course of management, the patient started to suffer from acute severe epigastric and left-sided abdominal pain and became hemodynamically unstable with hypotension (80/60mmhg) and tachycardia (120 b/m). Resuscitation was started immediately, and the general surgery team decided to do an emergency surgery as the patient was unstable to do any imaging studies, including ultrasonography or CAT Scan. Laparotomy was done to reveal internal bleeding as a result of a ruptured spleen, and splenectomy was performed with securing hemostasis.

Outcome and Follow Up

After surgical intervention, the patient was closely monitored and followed up until full recovery from the surgical point of view. Nevertheless, subsequent hematological and histopathology studies confirmed the diagnosis of Non-Hodgkin Lymphoma; an excisional biopsy of the resected spleen showed distorted architecture with vast necrosis and hemorrhage and variable areas area revealed sinusoidal infiltration by large atypical lymphoid cells that are highly concomitant with NHL. Consequently, the patient was referred to the oncology department for treatment.

Discussion

Splenic rupture can be divided into three categories; traumatic rupture and non-traumatic rupture with an underlying pathology [2]. In other categorization, atraumatic rupture of the spleen can be classified into several subgroups, namely, infectious, neoplastic, inflammatory, congenital or structural, iatrogenic, and idiopathic [3,4]. The term spontaneous splenic rupture was introduced in the 19th century; however, the first cases were reported by Laseter *et al [4].* The incidence of atraumatic splenic rupture is clinically rare; where a published study suggests that it represents 3.2% with a twice male predominance compared to the incidence among females [3,4]. The age varies from 2 to 81 years (average = 42 years) [4]. This is similar to this reported case where the patient is male, 41 years old.

Rupture of the spleen typically presents a clinical picture of a patient in shock with profuse bleeding that requires immediate splenectomy in 85% of cases [5]. In 8% of cases, patients die before being operated on, and the diagnosis is only made at postmortem autopsy.[4] However, patients may present with splenic rupture symptoms, including left-sided abdominal pain and peritonitic abdomen. Besides, some uncommon known signs include left shoulder pain and palpable tender mass in the left upper quadrant; Kehr's and Balance's signs may elicit positively [2]. Our patient didn't initially present with these clinical symptoms or signs, as he presented with a clinical picture suggestive of the underlying pathology; non-Hodgkin lymphoma. Therefore, our patient was initially managed as a case of ITP; until he deteriorated and developed atraumatic splenic rupture.

Some studies introduce three mechanisms that contribute to ASR, elevated intra-splenic tension resulting from hyperplastic cells, compression of abdominal wall muscles during increased intra-abdominal pressure, and vascular occlusion causing infarction with or without subscapular hematoma [4]. The mechanism of splenic rupture in our case was not clear though areas of necrosis were captivated in the histopathological study.

The presenting symptoms and physical signs do not mainly confirm the diagnosis of spontaneous splenic rupture; ultrasound is considered the initial imaging modality, and a CT scan is confirmatory with high sensitivity for lesion assessment [4]. Regardless, some studies concluded that there are five identified criteria defining spontaneous splenic rupture: the absence of a history of trauma or of unusual effort that might have caused injury to the spleen, the absence of perisplenic adhesions suggestive of any previous trauma; the absence of preexisting splenic disease; and normal microscopic and macroscopic appearance of the spleen. No rise in viral antibody titers suggestive of recent infection [5]. Though our patient has an underlying neoplastic disease, at the time of being diagnosed with a ruptured spleen, no obvious and known cause could have been attributed to his condition at that moment. In that, the diagnosis of NHL was established later on. In addition, our patient was in a critical emergent condition so no imaging studies were done prior to surgery. Regarding treatment, the associated mortality with splenic rupture demands rapid diagnosis and intervention [5]. Splenectomy is the radical cure for spontaneous rupture of the spleen. However, conservative treatment might be tried to preserve the spleen [4]. In our case, our patient was hemodynamically unstable; therefore, surgical intervention ended with a splenectomy executed.

Conclusion

Though ruptured spleen remains a low incidence unclear clinical entity, its diagnosis has to be kept in mind in patients without a history of trauma but with predisposing risk factors. This relays significant consequences as early diagnosis would surely

decrease this condition's high mortality and morbidity rates.

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