

# Postpartum Spontaneous Splenic Rupture: A Case Report

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## Abstract

The spleen remains the most commonly affected organ in blunt injury to the abdomen in all age groups. Histologic findings (organ expansion with capsular thinning, abnormal internal architecture with reduced elasticity to the parenchyma) may help to explain why a minor trauma resulted in a major splenic injury. Such events may happen with splenomegaly due to hematologic abnormalities (eg, hereditary spherocytosis), infectious diseases (eg, malaria, infectious mononucleosis), and liver disease (eg, portal and splenic hypertension). Nonoperative or conservative treatment usually includes patients with stable hemodynamic signs, stable hemoglobin levels, minimal transfusion requirements, and younger patients. Surgical therapy is usually reserved for patients with signs of ongoing bleeding or hemodynamic instability. Prognosis is usually excellent, but those patients left asplenic by their injuries and surgery increase the risk of fatal and debilitating infection for the remainder of their lives. Spontaneous splenic rupture during pregnancy is a rare event. It occurs most commonly in the third trimester or puerperium. The lethality and devastating consequences for both mother and fetus from failure of preoperative diagnosis give this entity great importance. Absence of a history of trauma can make it difficult to reach a diagnosis and causes delay in treatment. In this case report a 24-year-old female presenting with spontaneous splenic rupture following stillbirth is presented. The rupture was apparently related to infection but whether due to brucellosis or puerperal infection is unclear. The patient made a complete recovery after surgery and antibiotic treatment.

**Keywords:** splenic rupture, pregnancy, puerperium, brucellosis, infection

## Özet

### Postpartum Spontan Dalak Ruptürü: Olgu Sunumu

Dalak, tüm yaş gruplarında künt batın travmalarında en sık etkilenen organdır. Histolojik bulgular (organda büyüme, kapsülde incelmeye, parenkim elastisitesinde azalma) minör bir travmanın niçin büyük bir dalak yaralanmasına yol açtığını açıklayabilir. Hematolojik anormallikler (herediter sferositoz), enfeksiyon hastalıkları (sıtma, enfeksiyöz mononükleozis) ve karaciğer hastalıkları (portal ve splenik hipertansiyon) gibi splenomegali yapan durumlar buna neden olurlar. Konservatif tedavi, hemodinamik bulguları ve hemoglobin düzeyleri stabil ve transfüzyon gereksinimi minimum olan genç hastalarda tercih edilir. Cerrahi tedavi ise kanamanın devam ettiği, hemodinamisi stabil olmayan hastalarda uygulanır. Cerrahi tedavi sonrası prognoz çok iyi olmakla birlikte, hastaların yaşamlarının sonraki dönemlerinde ağır seyreden ölümcül enfeksiyon riski artar. Gebelikte birlikte görülen spontan dalak ruptürü nadir görülen bir durumdur. En sık olarak üçüncü trimester ve postpartum dönemde ortaya çıkar. Preoperatif tanı konulamamasının anne ve fetus için ölümcül sonuçlar yaratması bu durumu çok önemli kılar. Öyküde travmanın olmayışı tanı konulmasını güçleştirerek tedaviyi geciktirir. Bu olgu sunumunda ölü doğum yaptıktan sonra spontan dalak ruptürü tanısı alan 24 yaşındaki olgu sunulmuştur. Bu olguda ruptürün sebebi enfeksiyonla ilgili gibi görünse de brusellozis veya puerperal enfeksiyonla olan ilişkisi belirgin değildir. Hasta cerrahi ve antibiyotik tedavisi sonrası tamamen iyileşmiştir.

**Anahtar sözcükler:** dalak ruptürü, gebelik, puerperium, brusellozis, enfeksiyon

## Introduction

The spleen is a friable and vascular organ. If the spleen is diseased or enlarged, minor trauma may result in significant bleeding (1). Spontaneous (atraumatic) rupture of the spleen is an uncommon but important clinical entity. The 'splenic emergency syndrome' during pregnancy is characterized by the onset of severe pain in the left hypochondrium or epigastrium, followed shortly by hemorrhagic shock (2). There is an increased risk in the older mul-

tiparous woman. The diagnosis is often missed due to the absence of any history of trauma and delay in the diagnosis can lead to catastrophic consequences. Since the first case of splenic rupture in pregnancy was reported in 1803 by Saxtorph (2) many case reports have been published, which suggests that the lethality for both mother and fetus from failure of preoperative diagnosis give this entity great importance.

The purpose of this article is to identify the features of atraumatic rupture of the spleen in pregnancy and to enhance awareness of this unusual condition.

## Case Report

A 24-year-old female was referred from Şanlıurfa Mater-

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nity Hospital, where she had presented complaining of severe dizziness and weakness. One week before, she had given birth to a stillborn at home. It had been her seventh pregnancy (G7 P7 A2 A0). At the maternity hospital, her complete blood count showed Hb: 6.4 g/dL, Hct: 22.4%, Plt: 224,000/ $\mu$ L, WBC: 11,100/ $\mu$ L. She was diagnosed as suffering from anaemia and transfused with two units of blood. Her condition continued to worsen and she was referred to the Şanlıurfa University Hospital.

On arrival, she was conscious but her general condition was poor and had breathing difficulties. Her temperature was 38.2°C, arterial tension was 110/60 mmHg and pulse was 100/minute. Her abdomen was swollen, but there was no problem with discharge of flatus or urine. She revealed that she had experienced dizziness and had fainted after the birth but insisted that the birth was not difficult and that the midwife had not done anything to force it. A repeat complete blood count showed Hb: 7.3 g/dL, Hct: 23.6%, Plt: 407,000/ $\mu$ L, WBC: 20,000/ $\mu$ L, PMN 56%, lymphocytes 36%, monocytes 8%.

She was hospitalised with the diagnosis of puerperal infection and anaemia, started on ceftriaxone 1 g iv bid and metronidazole 500 mg infusion bid, and given a further blood transfusion while further tests were performed.

Her biochemistry results were shown in Table 1. Serological tests for HBsAg, HBsAb, anti-HCV, anti-HDV, VDRL, Widal-AG-AH, Widal-AG-AO, Widal-AG-TH and Widal-AG-BH were all negative, while Widal-AG-TO was 1/40 and Widal-AG-BO 1/20. *Brucella* Wright agglutination test was positive at 1/1280 and the Rose Bengal test was positive. *M. tuberculosis* was negative by PCR and hybridisation. Ascitic fluid analysis showed: glucose: 1 mg/dL, total protein: 3.3 g/dL, albumin: 2.2 g/dL, globulin: 1.2 g/dL, LDH: 820 U/L, amylase: 25 U/L. The ANA and test for anti-SM were negative. A chest X-ray showed restricted inspiration at the mid-zone of the left lung, and linear atelectasis parallel to the diaphragm.

Parameter	Result	Reference range
ALP (U/L)	129	53-128
Total protein (g/dL)	5.3	6.4-8.3
Albumin (g/dL)	3.1	3.4-4.8
LDH (U/L)	500	100-190
Fe ( $\mu$ g/dL)	10	40-170
Transferrin (mg/dL)	149	185-350
PT (s)	16	10-15
aPTT (s)	33	26.5-40.0
Fibrinogen (mg/dL)	569	200-400
CRP (mg/dL)	14	0.0-0.8
ASO (IU/ml)	950	0-200



**Figure 1.** Computerized tomography view of intraparenchymal and subcapsular haematoma of spleen. The dimension of spleen was 19 cm and craniocaudal dimension of haematoma was 17 cm.

A portal vein colour Doppler was normal but an abdominal ultrasound showed an enlarged spleen (longitudinal axis 170 mm) and free fluid in the pouch of Douglas. On examination, this fluid was found to be defibrinated blood. Cytological examination showed numerous polymorphonuclear leukocytes and a few vacuolar histiocytes and mesothelial cells, consistent with infection. An abdominal CT showed splenic haematoma (Figure 1) and she underwent explorative laparotomy. Splenectomy, distal pancreatic resection and partial omentectomy were performed. Pathological examination of the material removed showed a laceration zone and hemorrhagic foci in the splenic parenchyma. Blood cultures were negative and a vaginal culture was polymicrobial with no specific pathogen present.

The patient was discharged on the 8<sup>th</sup> day after operation. At one month follow-up, she was healthy.

## Discussion

Spontaneous rupture in pregnancy without antecedent trauma is a rare condition (3), however its true incidence is unknown (2). Several pathophysiological mechanisms of splenic injury have been put forward in certain specific conditions (2). Splenic rupture in pregnancy has been attributed to the hypervolemic state, splenic enlargement, diminished peritoneal cavity volume due to the enlarged uterus and muscular contractions during pregnancy (2). Pregnancy itself can result in splenic rupture, with 150 such cases having so far been documented in the world literature. Most of these occurred in the third trimester or in the puerperium. Maternal mortality rates up to 70% have been reported (3,4). There is also an increased risk of fetal or infant death (3).

The clinical presentation of splenic injury is highly variable. In most patients with minor focal injury to the spleen, left shoulder tenderness may also be present as a result of subdiaphragmatic nerve root irritation with referred pain. With free intraperitoneal blood, diffuse abdominal pain and rebo-

und are more likely. If the intra-abdominal bleeding exceeds 5-10% of blood volume, clinical signs of early shock may manifest. Unstable patients suspected of splenic injury and intra-abdominal hemorrhage should undergo exploratory laparotomy and splenic repair or removal. A blunt trauma patient with evidence of hemodynamic instability unresponsive to fluid challenge with no other signs of external hemorrhage should be considered to have a life-threatening solid viscus (splenic) injury until proven otherwise.

Rupture of the spleen in the postpartum period poses a significant difficulty for early diagnosis, because more common entities present with similar clinical findings especially early in the course of the rupture. These include exaggerated postpartum pains, uterine rupture, intra-abdominal bleeding, and injury of a viscus. Signs and symptoms of severe shock states might be mimicked by septic shock, amniotic fluid embolus, pulmonary embolus, cardiogenic shock, and disseminated intravascular coagulopathy (5). In our case, a picture of hemorrhagic shock was noted.

Splenosis is defined as the autotransplantation of splenic tissue after disruption of the splenic capsule. It occurs most commonly in the peritoneum, omentum, and the mesentery where nodules of splenic tissue are seen. The thoracic splenosis which is an uncommon phenomenon became evident 1 to 42 years after the injury, may provide an intermittent pyrexia of unknown origin (6). However, splenosis presented with an initial complaint of fever which mimicks puerperal fever has not been reported to be associated with postpartum splenic rupture.

Brucellosis is known to occasionally cause both splenic rupture (7) and spontaneous human abortion and intrauterine fetal death (8). It is not unusual to fail to isolate *Brucella* from cultures of blood or body fluids of seropositive individuals. For example, in the series of 92 pregnant women with brucellosis reported on by Khan et al (8), only 42% had positive blood cultures. The standard treatment for brucellosis is trimethoprim-sulphamethoxazole (TMP-SMX) plus rifampicin but a third generation cephalosporin may be used instead of TMP-SMX.

In this case, the antibiotic regimen was designed with puerperal infection in mind. It is possible that ceftriaxone without rifampicin would be efficacious against *Brucella* but follow-up to ensure the absence of relapse is highly desirable.

Vaginal cultures are frequently not helpful in diagnosis, except to the extent of possibly detecting such specific pathogens as *Streptococcus pyogenes* and *Clostridium*. Though culture was not performed on pouch of Douglas fluid, even cytological staining would probably have revealed *Clostridi-*

*um* or *Enterobacter*, the two organisms which have been reported as associated with both puerperal fever and splenic rupture (though not simultaneously) (2).

Because of the immunologic function of the spleen, interest over the last century has turned to salvage of the spleen rather than splenectomy (9,10). The advent of CT scanning has made conservative management more practical and safer for victims of splenic injury (11), but deaths due to splenic rupture are still reported in hospital discharge statistics from both trauma centers and community hospitals (12,13). In this respect, splenectomy still remains the prime treatment in splenic rupture, with appropriate antibiotic treatment for any underlying bacterial infection (12,13,14).

In conclusion, the survival of patients with spontaneous postpartum splenic rupture rests on several factors, including aggressive transfusion management, early diagnosis, and splenectomy. Increased awareness and a high index of suspicion of this condition would enhance early diagnosis and effective treatment.

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