Delayed Splenic Rupture after Robotic Partial Nephrectomy

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ABSTRACT

Introduction: Splenic rupture can be classified as traumatic, pathologic, or spontaneous. Most cases of spontaneous splenic rupture involve a histopathologically abnormal spleen, but in rare cases, rupture of the spleen can occur in the absence of underlying disease or trauma. We present a case of delayed spontaneous splenic rupture in the postoperative setting following a partial nephrectomy.

Case Description: A 54-y-old man presented with abdominal pain, dysuria, fever, and chills 1 week after a robotic left partial nephrectomy. An initial computed tomography scan showed no evidence of splenic injury, and he was admitted for suspected pyelonephritis. A computed tomography scan was obtained 4 d later for worsening pain and fever and revealed a 14-cm subcapsular hematoma of the spleen extending to the gastrohepatic ligament. He underwent an emergent angiogram and embolization of an actively bleeding splenic artery and inferior phrenic artery. A second embolization was required 2 d later to control ongoing bleeding. He then developed increased abdominal pain with nausea, vomiting, and continued leukocytosis secondary to a completely infarcted and necrotic spleen. A laparoscopic, hand-assisted splenectomy was performed successfully, and he was eventually discharged in stable condition.

Conclusion: Spontaneous splenic rupture is extremely rare, particularly in the postoperative setting. It is possible that some of these cases are in fact secondary to occult trauma to the spleen during surgery. Prompt diagnosis and management, often with emergent splenectomy, is critical in these cases. Minimally invasive surgery is a feasible option for splenic resection in cases of spontaneous splenic rupture.

Key Words: splenic rupture, splenectomy, nephrectomy.
Since 1958, the Orloff and Peskin criteria have been used to establish the diagnosis of true spontaneous rupture (atraumatic and idiopathic in nature); the criteria requires no antecedent trauma history, no evidence of disease of organs other than the spleen that can cause rupture, no perisplenic adhesions or scarring consistent with post-trauma, and a normal spleen on gross and histological examination to qualify as spontaneous splenic rupture. A fifth diagnostic criterion of virologic studies was added by Crate and Payne to rule out recent viral infection associated with splenic involvement. Diagnosis is based on ultrasound or abdominal computed tomography (CT) and in most cases treatment requires splenectomy.

We present a case of delayed splenic rupture in a postoperative patient, with the goal of increasing knowledge about the signs and symptoms of spontaneous splenic rupture in the postoperative setting to facilitate early diagnosis and treatment.

**CASE PRESENTATION**

A 54-y-old male with diabetes, hypertension, chronic kidney disease, and left papillary renal cell carcinoma treated initially with robot-assisted left partial nephrectomy in 2018 presented to our institution with a new left renal mass that had grown on surveillance imaging (Figure 1). He underwent a second robot-assisted partial nephrectomy in July 2019 and was discharged after an uncomplicated postoperative course. Pathology revealed a benign renal mass composed of fibroadipose tissue with fat necrosis and histiocytic giant cell reaction. He presented 1 wk postoperatively with abdominal pain, dysuria, and fever to 37.4°C. Laboratory results were significant for leukocytosis of 18,600 and urinalysis was positive for leukocyte esterase and nitrites. Pyelonephritis was suspected, but a CT scan was performed to rule out an intraabdominal abscess or pseudoaneurysm of the renal artery (Figure 2). Mild postsurgical changes were noted in the left retroperitoneum, but the spleen was normal and stable in comparison with the preoperative imaging 2 months ago. The patient’s abdominal pain, however, persisted, and he remained febrile despite broad-spectrum intravenous antibiotics.

Given the lack on improvement on intravenous antibiotics, a repeat CT scan was obtained 4 d after the initial scan. The CT imaging now revealed significant findings of a 14-cm subcapsular hematoma of the spleen extending into the gastro-

![Figure 1. Preoperative abdominal CT. This was a surveillance CT that initially showed growth of a left renal mass.](image-url)
hepatic ligament (Figure 3). The patient underwent emergent embolization of an actively bleeding splenic artery and inferior phrenic artery with two coils placed in the distal splenic artery and a lipiodol mixture placed in the left inferior phrenic artery. The patient tolerated the procedure well, but his hemoglobin steadily decreased over the subsequent 48 h. A CT angiogram showed continued bleeding from the distal splenic artery and an interval increase in the size of the hematoma to 14.7 cm. Repeat embolization of the splenic artery was performed, with seemingly successful cessation of bleeding on postintervention angiogram.

Despite successful embolization, the patient continued to clinically deteriorate. Over the following week, he developed nausea and vomiting with diffuse abdominal tenderness. He had marked leukocytosis of 30,000 and a platelet count more than 1,000,000. A final repeat CT scan was obtained 6 d following the second embolization and showed a massively enlarged spleen that was completely infarcted and necrotic and a larger hematoma within the gastrohepatic ligament compressing the gastric fundus (Figure 4). A surgical consultation was requested by the urology team, and splenectomy with hematoma evacuation was recommended. A laparoscopic hand-assisted splenectomy was undertaken.

Intraoperatively, the patient was found to have massive hemoperitoneum and a ruptured spleen with portions densely adherent to the left diaphragm and extensive clot and necrotic tissue along the greater curvature of the stomach. Hand assistance was critical to obtain exposure and remove the ruptured splenic tissue and clot. Two closed suction drains were placed in the surgical bed. Pathologic analysis confirmed necrotic spleen and organizing hematoma with granulation tissue. No additional underlying splenic abnormality was found on histopathology. His recovery was complicated by a delayed urine leak, which presented as high drain output 3 d after surgery and confirmed by elevated fluid creatinine. This required ureteral stent placement, with rapid decrease in drain output. He was eventually discharged in stable condition, with outpatient follow up for stent removal.
DISCUSSION

This report presents the case of delayed splenic rupture in the early postoperative period after a partial left nephrectomy. The etiology of the splenic rupture in this case could be traumatic secondary to occult injury during the surgery or could be deemed atraumatic idiopathic, given the initial unremarkable postoperative imaging. Atraumatic-idiopathic splenic rupture is a rare and understudied clinical entity. The atraumatic-idiopathic classification for splenic rupture is generally a diagnosis of exclusion, once trauma and pathologic causes of splenic rupture have been ruled out. Iatrogenic splenic injury from the surgery in this case was considered; however, the initial CT scan showed no injuries or abnormalities in the spleen following the partial nephrectomy. The diagnosis of a true spontaneous rupture in this case was particularly difficult given the context of the recent nephrectomy.

Idiopathic splenic rupture as a distinct clinicopathological entity has been controversial since its discovery, in part because of imprecise terminology. Historically, it has been argued that careful inquiry will always elicit a history of injury or trauma. The current literature also poses this question. In this case, the patient's prior nephrectomy and surgical history could qualify as antecedent trauma, but the initial CT after nephrectomy did not demonstrate any splenic injury and the patient's case otherwise fits Orloff and Peskin's criteria: his history includes no evidence of preexisting splenic disease and pathology confirmed a grossly and histologically normal spleen, other than necrosis induced by embolization. Pathologic analysis cannot distinguish between traumatic rupture and spontaneous rupture of a normal spleen because the pathologic features are the same (subcapsular hematoma, parenchymal tears, pedicle lacerations, fragmentation, and perisplenic hematomas). Other than abnormalities related to rupture, the spleen will be normal on both microscopic and gross examination.

Splenic injury is a well-documented complication of abdominal procedures, most commonly after colonoscopy. Splenic ruptures specifically following laparoscopic proce-
dures have been reported, with case reports hypothesizing that the traction on the spleen (either from adhesions or from the pneumoperitoneum at induction) is strong enough to cause rupture. Splenic injury has been reported after nephrectomy as well: recent studies have estimated that left nephrectomies are complicated by iatrogenic splenectomy in 4.3–13.2% of cases. In these instances, however, the mechanism of injury is attributed to either traction or injury from surgical instruments themselves. Unlike these previously reported cases, there were no clear indications of trauma to the spleen during initial or repeat nephrectomy in our patient, as evidenced by initial postoperative imaging and the patient’s initial postnephrectomy recovery. Occult trauma to the spleen during surgery is, however, a possible mechanism for postoperative delayed splenic injuries and should be considered. Injury to the inferior phrenic artery and the spleen during retraction and mobilization is even more conceivable in this case given the reoperative field.

Although rare, spontaneous splenic rupture is life threatening. A recent study reported a 12% mortality rate with spontaneous splenic rupture, and splenomegaly, age above 40 years, and neoplastic disorders were identified as risk factors. Although mortality was related to the underlying pathology in certain cases, mortality was also largely associated with a delay in diagnosis and treatment; 8% of patients died prior to surgical resection. In hemodynamically stable patients, such as ours, the gold standard for radiographic imaging is the CT scan. In splenic rupture, imaging would show subcapsular fluid collections, intraperitoneal fluid, and irregular splenic parenchyma. Abdominal CT imaging is 97% sensitive and specific in the diagnosis of splenic rupture. Management of unstable patients with splenic rupture typically requires splenectomy for hemorrhage, but in hemodynamically stable patients, conservative management or angiographic embolization can be used. Most reports of spontaneous splenic rupture (pathologic or idiopathic) required splenectomy, consistent with our patient’s hospital course. A minimally invasive approach to splenic resec-

Figure 4. Presplenectomy CT. This was the final repeat CT was obtained 6 d following the second embolization and showed a massively enlarged infarcted spleen that prompted decision for splenectomy.
tion in cases of splenic rupture is feasible: we were able to perform a hand-assisted laparoscopic splenectomy with a positive result in our patient.

CONCLUSION

Spontaneous splenic rupture is a rare but critical condition with significant associated morbidity and mortality. Whereas splenic rupture has been reported following laparoscopic surgery, rupture in most of these cases is iatrogenic secondary to traction forces transmitted to the spleen during the operation. Etiology of the splenic rupture was especially difficult to determine in this case, given the lack of clear injury during the surgery, delayed presentation, and the initial unremarkable postoperative imaging. Given that no particular pathologic risk factors were identified, we propose that the postoperative state could itself be a possible risk factor for spontaneous splenic rupture. Prompt diagnosis and management, often with emergent splenectomy, is critical in these cases. Although rare, surgeons must maintain a high index of suspicion for splenic rupture in the postoperative patient.

References: