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Complications of Renal Surgery*

Learning Objective: At the conclusion of this continuing medical education activity, the participant will be able to describe the identification, prevention, diagnosis and management of common complications after renal surgery.

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INTRODUCTION

Surgical excision is the most oncologically effective treatment for patients with localized renal cell carcinoma. Historically the gold standard surgical management of renal cell carcinoma was radical nephrectomy. However, partial nephrectomy has become widely accepted as a nephron sparing approach in select patients with renal masses, especially those with tumors smaller than 4 cm.[1] Both radical and partial may be performed via open surgery or minimally invasive surgery (laparoscopic or robotic). The ultimate decision regarding surgical type (partial nephrectomy vs radical nephrectomy) and modality (open vs minimally invasive surgery) for excision of a renal mass is best based on patient and tumor characteristics, and the surgeon's comfort level and expertise.[2]

Regardless of the type of surgery and modality, the goal of all renal surgery is to maximize oncologic control while minimizing perioperative risks and long-term functional complications. Understanding the perioperative challenges that patients may face following renal surgery not only ensures proper preoperative risk stratification and patient selection, but also facilitates recognition and intervention of postoperative complications when those arise. In this Update we review the most common complications after renal surgery and discuss their presentation, risk factors, diagnosis and treatment.

BLEEDING COMPLICATIONS

Hemorrhage. The kidney is an extremely vascular organ, making bleeding a relatively common complication after renal surgery. This may be associated with considerable morbidity and rarely mortality. **Bleeding complications occur more commonly after PN than following RN.** Bleeding after PN, which occurs in 1.6%–8.6% of patients,[3,4] generally stems from poorly controlled vasculature within the tumor resection bed. **Risk factors for hemorrhage after PN include large tumor size, endophytic tumor anatomy and increased intraoperative blood loss.**[5] Bleeding after RN, which occurs in 0.1%–3.3% of patients,[6,7] generally arises from the renal hilum vasculature. **A major risk factor for hemorrhage after RN is the presence of advanced disease, including venous invasion (renal vein and/or inferior vena cava) and tumor extension beyond Gerota's fascia.**[8] Regardless of the type of renal surgery, the risk of bleeding can be minimized through meticulous dissection and verification of complete hemostasis.

Patients with bleeding complications of renal surgery may present with oliguria, gross hematuria, flank pain, fatigue, pallor and/or shortness of breath. If an abdominal drain was placed intraoperatively, sanguineous drain output may be observed. However, when excess bleeding occurs, the drain may clot off and be unreliable in assessing for hemorrhage. Acute loss of large volumes of blood can lead to hemodynamic instability, shock and even death.[4]

Given the potentially catastrophic sequelae, prompt diagnosis and management of bleeding after renal surgery are paramount. The diagnosis of bleeding after renal surgery starts with a strong clinical suspicion and is confirmed using serial blood count assessments. Hemodynamically stable patients with bleeding are generally managed conservatively with close monitoring of hemodynamic status, assessment of serial blood counts and administration of intravenous fluids. **Symptomatic anemia and/or significant hemodynamic changes are indications for blood transfusions**. Patients with persistent bleeding despite conservative management and/or those presenting with hemodynamic instability may undergo reexploration or renal angiography and embolization. Selective embolization of bleeding segmental and subsegmental arteries can be performed to salvage the kidney in patients who are bleeding following PN. Although reexploration is an option, it should be used only as a last resort in patients who are too unstable to undergo selective embolization or when embolization has failed, given embolization's minimally invasive nature and ability to preserve renal function.[9] Furthermore, extreme caution is warranted during reexploration as disruption of a hematoma may exacerbate bleeding and increase the risk of nephrectomy. **Patients with life-threatening hemorrhage may require reexploration or complete angioinfarction of the kidney.**

Renal arteriovenous fistula and renal artery pseudoaneurysm. **Iatrogenic vascular events such as RAVF and RAP are infrequent complications after PN that are associated with significant morbidity.** RAVF, which occurs in 0.04%–1.5% of patients undergoing PN,[10, 11] is defined as an abnormal connection between the intrarenal arterial and venous circulation without an intervening capillary bed. RAP, which occurs in 0.4%–2.3% of patients undergoing PN, is defined as a collection of blood that forms outside of the injured arterial wall but is contained within the renal parenchyma.[10, 12] Although the precise etiology of RAVF and RAP is unknown, both complications are thought to arise after transection and failure to repair an intrarenal arteriole during tumor resection and/or renorrhaphy. RAVF develops when there is subsequent fistulization of the affected artery with a nearby vein, and RAP develops when blood extravasates into the extravascular space.[11] In historical series these vascular complications have been associated with minimally invasive PN.[10]

Patients with RAVF and RAP typically present in a delayed fashion at an average of 14 days postoperatively.[10] However, these complications may occur as long as 5 months after surgery.[12] As such, maintaining an index of suspicion for these diagnoses, even several months after surgery, is important. **Patients with RAVF and RAP commonly present with gross hematuria, which develops when there is concomitant fistulization of the collecting system.**[10, 12] However, gross hematuria in this setting may be sentinel and resolve spontaneously, which should not exclude the diagnosis of RAVF and RAP. Other common presenting symptoms include flank pain, dizziness, fatigue and anemia. In severe cases patients may present with life-threatening, high output heart failure and hemorrhagic shock.[13]

Given the potentially morbid nature of these complications, suspicion for development of RAVF and/or RAP should

ABBREVIATIONS: CA (chylous ascites), CKD (chronic kidney disease), CT (computerized tomography), MIS (minimally invasive surgery), PN (partial nephrectomy), PSM (positive surgical margin), RAP (renal artery pseudoaneurysm), RAVF (renal arteriovenous fistula), RN (radical nephrectomy), SMA (superior mesenteric artery)

prompt immediate diagnosis and treatment. Currently there is no standardized method for diagnosing RAVF and RAP. **Patients for whom there is high suspicion for RAVF and/or RAP (e.g. sudden onset of gross hematuria and/or flank pain) should proceed directly to percutaneous angiography, as this may allow for prompt diagnosis (fig. 1, *A*) and treatment with concomitant angioembolization (fig. 1, *B*).** When the diagnosis of RAVF and/or RAP is unclear, patients may first undergo a CT angiogram to assess renal vascular anatomy prior to percutaneous angiography and embolization. **Angioembolization, which may be performed in a selective fashion to maximally preserve renal tissue, is the cornerstone of management.[11]** Although most cases of RAVF and RAP may be successfully treated with a single session of angioembolization, refractory cases may require multiple angioembolizations and/or rarely surgical nephrectomy. **All patients undergoing PN must be made aware of this serious complication and instructed to seek immediate medical attention in the event of severe flank pain and/or gross hematuria.**

URINARY LEAK

Urinary leaks may occur after PN when the collecting system is violated during tumor resection and the integrity of renorrhaphy is imperfect or fails to heal. Urinary leakage may occur in 1.0%–17.4%,[14] 1.6%–16.5%[15] and 0.6%–3.0%[16] of patients undergoing open PN, laparoscopic PN and robotic PN, respectively. Although the data suggest that MIS PN is associated with lower rates of urinary leakage, higher tumor complexity necessitating open PN may contribute to these differences.[17] **Nevertheless, contemporary series of MIS employing modern renorrhaphy techniques suggest that urinary leak is relatively infrequent.[18]**

Risk factors for urinary leak after PN include increased tumor size, high tumor complexity as objectified by a nephrometry scoring system, warm ischemia time, blood loss, operative time; presence of a hilar tumor, intraparenchymal renal pelvis or stage III or higher preoperative chronic kidney disease; surgeon experience; and the need for complex pelvicalyceal repair.[18–20] Historically intraoperative placement of a ureteral catheter with injection of methylene blue was utilized to identify urinary leakage and confirm successful repair. However, there are limited data to suggest that this technique is associated with a significant decrease in urinary leaks, and most surgeons have abandoned routine use of this approach.[19]

When an abdominal drain is left at the end of a PN case, urinary leaks may be suspected in patients with an elevated or persistent drain output. **After resection of complex tumors with prolonged ischemia times, acute tubular necrosis may result in low urine output from the affected renal units for the first several days following surgery.[18] As the kidney recovers and urine production increases, drain output may increase in a delayed fashion.** Therefore, a low drain output within the first few postoperative days may not fully predict the occurrence of a delayed leak. **When an abdominal drain is present, a twofold increase in creatinine level over serum level is highly suggestive of a urinary leak.[18–20]** When an abdominal drain is not present, urinary leaks may be suspected in patients with abdominal/flank pain, ileus and/or fever. In a multi-institutional review of 1791 robotic PNs, in which all abdominal drains were removed prior to discharge, symptomatic urinary leaks presented at a median of 13 days (range 3–32) postoperatively.[18] In the absence of an abdominal drain, a practice that is now common among many kidney surgeons,[21–23] urinary leaks may be diagnosed when there is contrast extravasation on CT (fig. 2), magnetic resonance imaging or retrograde pyelogram. Alternatively when a postoperative fluid collection is noted on cross-sectional imaging, fluid creatinine analysis after aspiration and/or drain placement of the fluid collection may facilitate diagnosis.[15]

The majority of urinary leaks can be managed with percutaneous drainage.[18, 20] However, in cases with persistent or high volume leakage, obstruction must be considered. Therefore,

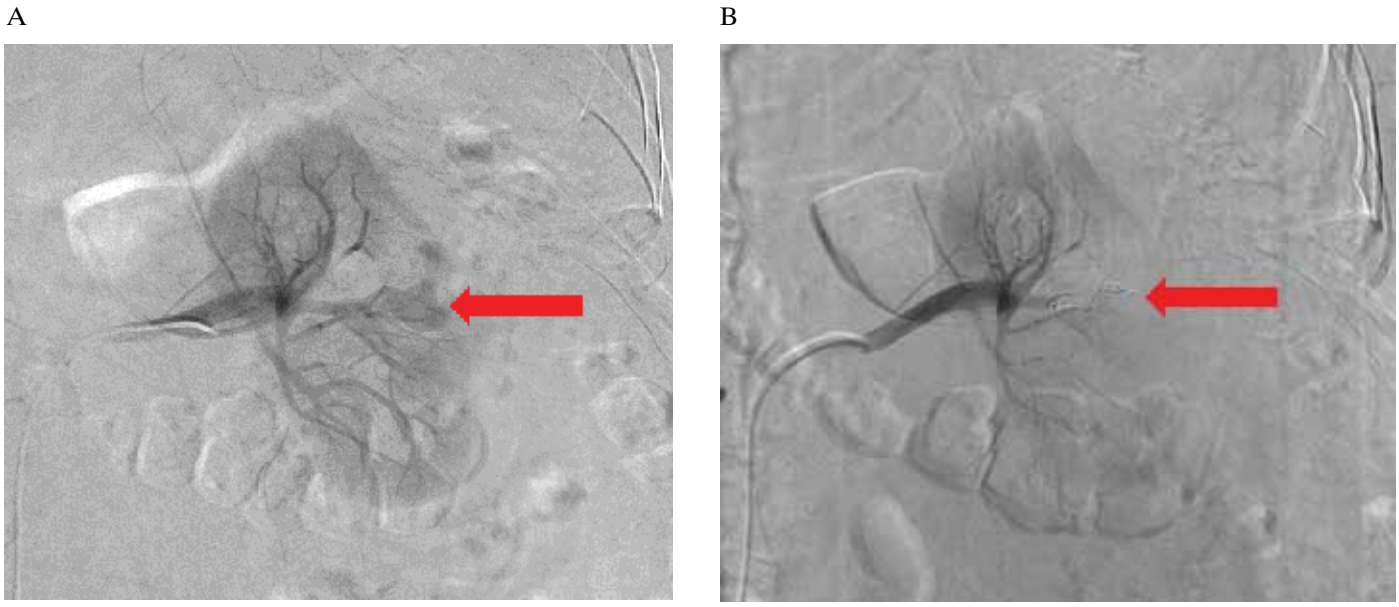

Figure 1. *A*, left renal arteriography demonstrates bilobed aneurysm formation (arrow) after left PN. *B*, post-embolization arteriography shows successful 3 ml coil (arrow) placement in middle segmental branch of left renal artery.

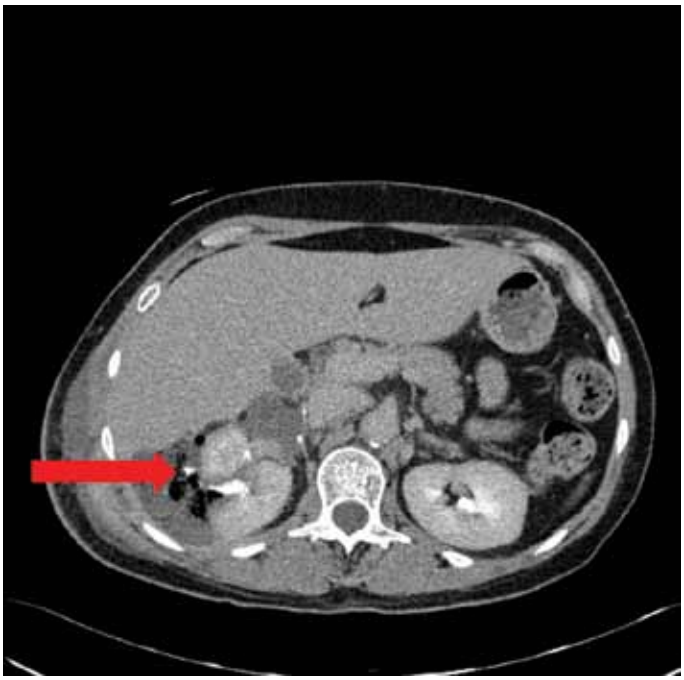

Figure 2. Delayed phase CT of abdomen with intravenous contrast shows extravasation of contrast material suggestive of urinary leak (arrow) from collecting system after right-sided PN.

retrograde pyelogram with placement of a nephroureteral stent may be performed to facilitate urinary drainage. In such cases use of a concomitant Foley catheter to reduce retrograde reflux should also be considered, especially in patients with benign prostatic hyperplasia and high pressure voiding. Rarely persistent urinary leaks may require reoperative management.[20]

LOSS OF RENAL FUNCTION

Loss of renal function is an important consideration in renal surgery. After RN loss of renal function occurs due to removal of an entire renal unit. Although the median postoperative decrease in renal function after RN has been reported to be approximately 35%,[24] this value can be difficult to accurately predict,[25] and may vary substantially depending on baseline patient characteristics and renal split function. **After PN loss of renal function may occur due to removal of healthy nephrons during tumor excision and/or ischemic insult to nephrons during temporary vascular occlusion.** Although the median postoperative decrease in renal function after PN has been reported to be approximately 10%,[26] this value varies greatly depending on baseline patient and tumor characteristics, surgical technique and quality of residual parenchymal volume following resection.[27] **Factors associated with a higher risk of renal decline include low preoperative renal function, the presence of a solitary kidney, large and endophytic tumors, longer ischemia times and utilization of warm ischemia.[28, 29] Yet most recent data suggest that residual parenchymal volume and baseline parenchymal quality are the most important predictors of ultimate renal function following PN.[27, 30, 31]**

Patients with renal function decline after RN and PN are generally asymptomatic. However, in patients with low baseline renal function renal surgery may cause clinically significant kidney dysfunction resulting in uremia, metabolic abnormalities and volume overload that may necessitate temporary or permanent dialysis.[32] Determination of renal function after renal surgery is critical as it is a metric of long-term renal functional stability and may be associated with overall survival. Zabell et al performed a retrospective review of 4283 patients who underwent renal cancer surgery and found that postoperative renal function was an independent predictor of 5-year CKD risk and non-renal cancer related mortality at 10 years.[29] **Although CKD is associated with an increased risk of cardiovascular events or all cause mortality,[32] recent data suggest that surgically induced CKD may be associated with a lower risk of functional decline and mortality than medical CKD, indicating that they may be distinct entities.[33]** Indeed, in a phase 3 international trial that randomized 541 patients with normal renal function to PN or RN, Scosyrev et al found that loss of function due to RN was not predictive of overall survival.[34]

During PN various technical modifications can help maximize postoperative renal function. **Recent studies suggest that parenchymal mass preservation is the most important determinant of functional recovery after PN, with ischemia playing a secondary role.[27]** As such, care should be taken to precisely excise the tumor and carefully reconstruct the remnant kidney to maximize nephron preservation. Some authors suggest that tumor enucleation (resecting the tumor along the tumor pseudocapsule) rather than standard resection (maintaining a rim of normal parenchyma during resection) may allow for improved renal function preservation.[35] However, Blackwell et al demonstrated that although tumor enucleation maximally spares normal parenchyma compared to standard resection, functional differences are marginal.[36] **With regard to ischemia time, recent studies suggest that most nephrons make a near complete recovery from ischemic insult after PN as long as warm ischemia time is less than approximately 25 minutes.[27, 28, 37] When ischemia time is expected to be longer than 25 minutes, utilization of cold ischemia may minimize renal function loss.[28]**

POSITIVE SURGICAL MARGIN

PSM refers to cancer cells extending to the inked surface of the resected specimen. This is more likely to occur after PN compared to RN due to the risk of leaving residual cancer in the remnant kidney. PSMs occur in 1.1%–10.7% cases after PN. Risk factors for PSMs include tumors close to the renal hilum/central location, higher tumor stage, larger tumor size and surgeon inexperience.[38] **Although some authors have suggested that MIS may be associated with higher PSM rates,[39] multiple reports have confirmed that surgical modality does not significantly affect PSM rates.[40, 41]** Similarly, although some authors have suggested that tumor enucleation compared to resection may be associated with higher PSM rates, multiple studies suggest that properly performed tumor enucleation does not significantly affect PSM rates.[41–43]

The oncologic and clinical significance of PSMs remains somewhat controversial. Petros et al demonstrated that patients with PSMs after PN compared to those with negative surgical margins experienced worse overall survival, local recurrence and metastasis.[44] Conversely in a population based analysis by Ani et al there was no statistically significant difference in 5-year cancer specific and overall survival in patients with PSMs vs negative surgical margins (cancer specific survival 90.9% vs 91.9%, and overall survival 84.4% vs 88.6%; p=0.58).[38] One proposed explanation for this discrepancy is that PSMs

are associated with disease recurrence and progression only in patients with high grade disease.[45]

Nonetheless, every effort should be made to obtain negative margins during PN, as this portends the best opportunity for complete tumor control. As the risk of local and metastatic recurrence is low in patients with PSMs, most patients with focal PSMs after PN who grossly had complete tumor removal may be managed with close surveillance. Although repeat PN or completion nephrectomy can be performed in patients with PSMs after PN, this strategy should be used extremely judiciously as only a small percentage of patients will have residual malignancy on final reoperative pathology.[45]

RHABDOMYOLYSIS

Rhabdomyolysis is a relatively rare but potentially morbid complication that occurs in 0.1%–1% of patients after renal surgery.[46, 47] It is characterized by the rapid breakdown of skeletal muscle fibers resulting in the release of myoglobin, creatinine kinase and electrolytes into the bloodstream. **During renal surgery the use of a flexed lateral decubitus position and/or a kidney bar/rest may cause prolonged compression and subsequent ischemia to the gluteal and thigh muscles. Risk factors for rhabdomyolysis include patient obesity, increased flexion of the operating table and prolonged operative times.[46]**

Rhabdomyolysis may present with excessive muscular pain out of proportion to examination occurring on the contralateral gluteal and lateral quadriceps muscles.[47] Myoglobinuria, one of the defining features of rhabdomyolysis, causes a characteristic "tea-colored" urine and can lead to acute tubular necrosis, resulting in acute kidney injury, oliguria and even renal failure. Although the precise mechanism by which myoglobinuria causes acute tubular necrosis is unclear, the combination of reduced intravascular volume and renal vasoconstriction, intraluminal cast formation and direct cytotoxicity may play a role.[48] Rhabdomyolysis may also lead to compartment syndrome. When ischemic injury affects muscles sheathed in noncompliant fascia, the resulting increase in intracompartmental pressure can lead to compromised arteriolar perfusion of muscle and nerve fibers, resulting in further tissue damage. Other rare complications of rhabdomyolysis include metabolic acidosis, cardiac arrhythmias from severe electrolyte abnormalities and disseminated intravascular coagulation.[46, 47]

The diagnosis of rhabdomyolysis is usually confirmed by an elevated plasma creatinine kinase level, although there is currently no standardized cutoff threshold for definitive diagnosis. Generally a concentration of 5 times the upper limit of normal (>5000 U/l) is used.[46] Plasma myoglobin is not as sensitive as creatinine kinase for diagnosis because of its short half-life (2–4 hours for myoglobin vs 1.5 days for creatinine kinase).[46, 47] Although urinary myoglobin may aid in diagnosis, routine testing for urinary myoglobin may be negative in up to half of patients with rhabdomyolysis.[48]

Rhabdomyolysis may be prevented by minimizing flexed positioning, avoiding use of the kidney bar/rest and ensuring all pressure points are appropriately padded during surgery. However, when rhabdomyolysis does occur, treatment consists of aggressive intravenous fluid hydration, and close monitoring and correction of electrolyte abnormalities.[47, 49] In rare cases of myoglobin induced renal failure or severe electrolyte abnormalities hemodialysis may be needed. Furthermore, in cases with associated compartment syndrome emergent fasciotomy and debridement of necrotic muscle should be performed. **Although some authors have recommended using bicarbonate to promote myoglobin washout, and mannitol to increase urinary flow and reduce myoglobin cast obstruction, the data supporting these strategies are limited.[49]** A recent study by Brown et al evaluating 382 trauma patients with rhabdomyolysis concluded that using bicarbonate and mannitol does not prevent renal failure, dialysis or mortality.[50]

HYPERTENSION

The effect of renal surgery on postoperative blood pressure has not been well characterized due to the absence of consistent findings and paucity of high quality studies. Although some reports have demonstrated that patients may develop new onset or worsening hypertension as a short-term complication after PN, these reports are limited to small case series.[51, 52] In a report by Hutchinson et al that evaluated 264 patients who underwent renal surgery those who underwent PN were more likely to be placed on new or additional antihypertensive medications postoperatively.[53] Although the precise mechanism explaining their findings is unclear, it has been proposed that temporary hypoperfusion of the kidney during PN may induce sustained activation of the renin-angiotensin-aldosterone axis. This mechanism is analogous to that of the 2-kidney, 1-clip Goldblatt model.[54] However, most other reports have failed to find an association between PN and postoperative hypertension.[55, 56]

In contrast to the results from Hutchinson et al,[53] Shah et al performed a propensity matched analysis of 13,893 patients from a national administrative database and found that RN was associated with a higher risk of new onset and worsened hypertension compared to PN.[57] The mechanism by which RN may lead to hypertension is unclear. Due to the lack of consistent findings in the literature regarding the effect of renal surgery on postoperative blood pressure, further studies are needed to elucidate this relationship.

INJURY TO SURROUNDING STRUCTURES

Bowel injury. Bowel injury occurs in 0.3%–0.5% of patients undergoing transperitoneal renal surgery.[58–60] Generally bowel injury occurs during access to the abdominal cavity or during bowel mobilization. While gaining access to the peritoneal cavity, particularly in patients with a history of abdominal surgery who may have bowel adhered to the abdominal wall, injury is possible. **During MIS inadvertent bowel injury may occur while obtaining peritoneal access using a Veress needle or inserting a trocar. Inserting trocars under direct visualization may minimize this risk**. Inadvertent thermal damage or direct laceration to bowel may occur while mobilizing bowel, particularly in patients with a history of prior abdominal surgery and perinephric inflammation.[59] In cases of thermal bowel injury presentation may be significantly delayed. Performing retroperitoneal renal surgery in patients with an extensive abdominal surgical history may lower the risk of bowel injury.[59, 60]

Prompt recognition of a bowel injury is critical as the spillage of intestinal contents may lead to sepsis, multiorgan system failure and even death. Patients with bowel injury after open surgery may present with classic signs and symptoms of an acute abdomen, such as abdominal tenderness and rigidity, leukocytosis and fever. **However, patients with bowel injury**

after MIS may present without peritonitis and with more insidious symptoms such as focal trocar site pain, low grade temperatures and leukopenia.[59] As such, a high index of suspicion should be maintained in such patients. When bowel injury is suspected, CT with oral and intravenous contrast should be obtained to evaluate for free intraperitoneal air and extraluminal contrast extravasation (fig. 3). Although a small volume of intra-abdominal air may normally be observed after MIS, a persistent or increasing volume of air in the peritoneal cavity suggests bowel injury until proven otherwise.[61]

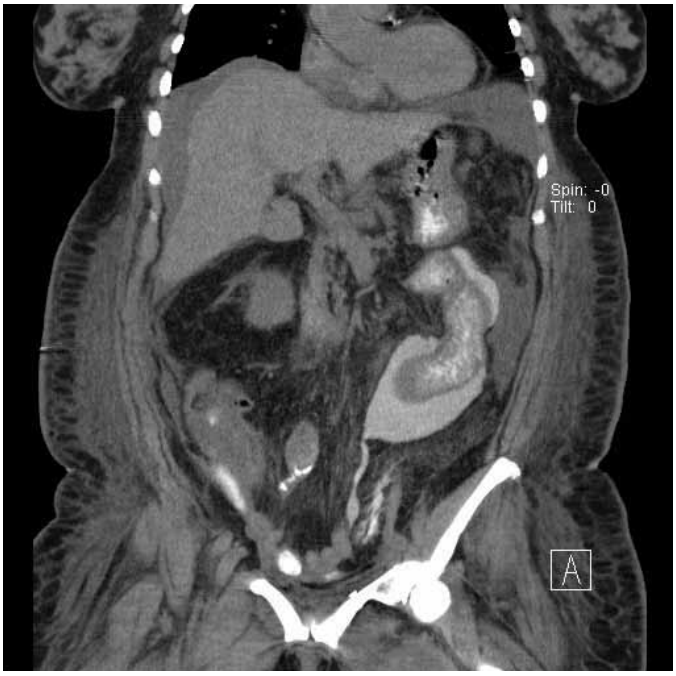

Figure 3. CT with oral contrast material demonstrates extravasation of contrast material from small bowel into peritoneum, indicating small bowel injury after left RN.

Fortunately most bowel injuries are recognized intraoperatively. Minor serosal tears may be repaired using a single layer of interrupted sutures. Full-thickness injuries may require a double-layer closure or bowel resection with re-anastomosis depending on the degree of injury.[59, 60] In cases of extensive bowel injury an intraoperative consult to general surgery is prudent. Postoperatively prompt diagnosis and intervention of bowel injury is critical.

Pleural injury. Pleural injury occurs in 0.6%–12.9% of patients undergoing renal surgery.[62] Given the proximity of the kidneys to the costodiaphragmatic recesses of the pleural spaces, pleural injury may occur while obtaining access to the retroperitoneum during open renal surgery via a flank approach, and rarely while placing trocars during retroperitoneal MIS. Also, pleural injury may occur while mobilizing the liver (right-sided cases) and spleen (left-sided cases) to expose the renal upper poles. Lastly, during right-sided MIS utilization of a liver retractor that is fixed to the diaphragm may result in pleural injury. Major risk factors for pleural injury include retroperitoneal surgery and large renal upper pole tumors.[63, 64]

Prompt diagnosis of pleural injury is critical as significant hypercarbia and tension pneumothorax (when accompanied by lung injury) may result (fig. 4). **Pleural injury may result in changes in cardiopulmonary status, including decreased oxygen saturation, increased end tidal carbon dioxide, increased airway pressure and decreased breath sounds on the affected side. During open surgery pleural leaks may be identified by filling the surgical wound with water and having the anesthesiologist deliver a large tidal volume.[63] During MIS pleural injury may be suspected in patients with diaphragmatic billowing.[64]** Regardless of surgical approach, definitive diagnosis intraoperatively is made via direct inspection of the diaphragm. Postoperatively chest x-ray may be used for diagnosis.

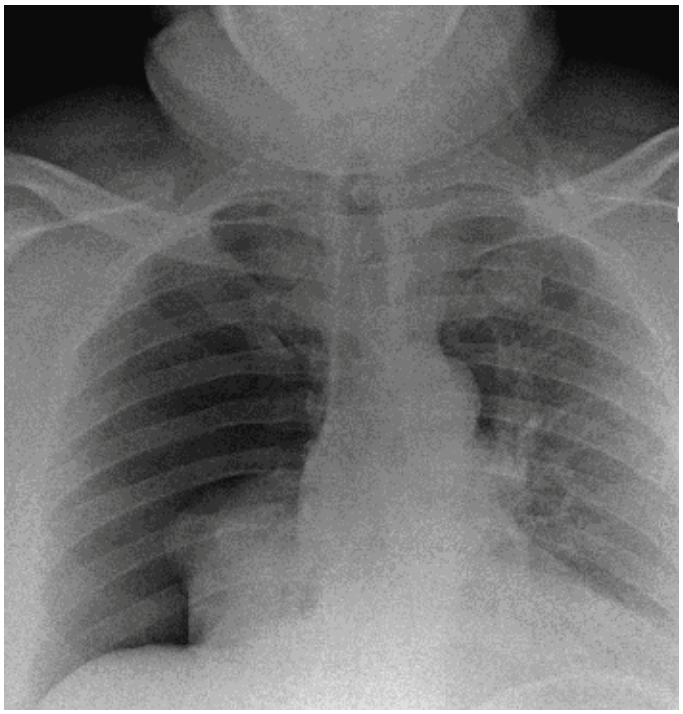

Figure 4. Chest x-ray shows right pneumothorax after right-sided PN.

When pleural injury is diagnosed intraoperatively, a catheter should be used to evacuate air from the pleural space prior to repair. One end of the catheter is placed into a cup of sterile saline and the opposite end is placed into the pleural defect. The anesthesia team is asked to deliver large tidal volumes until air bubbles cease to exit from the catheter. This mechanism allows air to exit on exhalation and prevents air from entering the pleural space on inhalation. After removing the catheter the pleural space is simultaneously closed using purse-string absorbable sutures. This technique has shown efficacy in both open surgery and MIS as long as a concomitant lung injury is not suspected.[62, 63] Small pleural injuries diagnosed postoperatively can be closely observed. In particular, pleural injuries following MIS may resorb more quickly as the chest cavity fills with carbon dioxide rather than ambient air. However, patients with significant and/or symptomatic pneumothoraces and those with concomitant lung injury may require a chest tube. **Regardless of when pleural injury is diagnosed and how it is treated, all patients should be serially monitored with upright end-expiratory chest radiographs to confirm resolution.**

Splenic injury. Splenic injury occurs in 0.5%–4.3% of patients undergoing renal surgery and primarily occurs during left-sided renal surgery. **This complication may arise due to excessive retraction of the spleen while trying to expose the left renal upper pole. As such, careful division of the splenorenal and**

splenocolic ligaments should be performed to reduce the risk of traction injuries. Less commonly, splenic injuries may also occur secondary to inadvertent laceration or thermal injury. **Risk factors for splenic injury include a history of abdominal surgery, left renal upper pole tumor, obesity and left kidney size.**[65, 66]

Due to the highly vascularized nature of the spleen, splenic injuries are usually recognized intraoperatively by a hematoma in the left upper quadrant. Superficial splenic lacerations may be treated via electrocauterization, argon beam coagulation, and/or biological and bioabsorbable hemostatic agents.[65, 66] Postoperatively splenic injuries may be diagnosed via CT. Deeper lacerations may require splenorrhaphy or splenectomy. Splenic injuries diagnosed postoperatively are generally associated with significant hemorrhage and necessitate emergent reexploration that often results in splenectomy. **As splenic macrophages are responsible for filtering and phagocytosing bacteria, patients undergoing splenectomy are at increased risk for infection by encapsulated bacteria. As such, conservative efforts should be exhausted before deciding to perform a splenectomy. If a splenectomy is performed, patients must receive postoperative pneumococcal, meningococcal and Haemophilus influenzae type B vaccinations.**[65, 66]

Pancreatic injury. Pancreatic injury occurs in 0.2%–2.1% of patients undergoing renal surgery. **It most often arises during left-sided renal surgery from aggressive retraction on the pancreas or excessive use of electrocautery during hilar dissection.** To reduce the risk of pancreatic injury, careful dissection between the tail of the pancreas and Gerota's fascia must be performed to mobilize the pancreas away from the left kidney. Risk factors for pancreatic injury include patients with prior abdominal surgeries and perinephric inflammation.[67]

Patients with pancreatic injury can have variable clinical presentations, depending on the severity of injury. Patients with superficial lacerations may be asymptomatic or present with symptoms of acute pancreatitis, such as abdominal pain radiating to the back, abdominal discomfort after eating, nausea and vomiting. Deep lacerations extending into the pancreatic ducts may lead to severe complications, including the formation of pseudocysts,[68] fistulas[69] and abscesses. **These complications may result in electrolyte abnormalities, malnutrition and even death.**[68, 69]

Postoperatively pancreatic injuries may be diagnosed via an elevated serum amylase and CT with intravenous contrast material demonstrating a capsular or ductal tear. **For minor pancreatic injuries treatment involves conservative management with bowel rest and possibly placing a nasogastric tube. If pancreatic injury is complicated by a peripancreatic collection or abscess, drainage of the collection and placement of a chronic drain may be necessary until the fluid amylase returns to normal serum levels. If pancreatic injury is recognized intraoperatively, it can generally be managed by direct suture repair. Pancreatic ductal injuries may require distal pancreatectomy.**[67–69]

Hepatobiliary injury. Hepatobiliary injury occurs in 0.1%–1.4% of patients undergoing renal surgery, and most often occurs during right-sided renal surgery due to aggressive retraction of the liver and/or inadvertent injury during bowel mobilization.[58, 70, 71] Prompt diagnosis of hepatobiliary injury is necessary to avoid life-threatening complications of hemorrhage, sepsis and liver failure. Intraoperative diagnosis involves direct inspection for bleeding and/or bile leakage. Postoperatively patients with hepatobiliary injury may present with right upper quadrant pain, hematoma, anorexia, fever and/or jaundice.[71] The diagnosis of hepatobiliary injuries may be confirmed via elevated serum liver function tests and CT with intravenous contrast. Fortunately liver injuries detected intraoperatively rarely require intervention beyond electrocautery to gain hemostasis. Postoperatively a severe liver injury resulting in hemorrhage may require reexploration. In cases of gall bladder and bile duct injuries a general surgery consultation is imperative. **Gall bladder injury may require a cholecystectomy, while biliary tree injury may require an intraoperative cholangiogram to more precisely define management options.**[71]

Lymphatic injury. Injury to the lymphatic system may result in chylous ascites, which is defined as leakage and accumulation of lipid rich lymph into the peritoneal cavity. CA may result after any renal surgery when there is injury to the cisterna chyli and its tributaries around the renal vessels.[72] **It is primarily associated with left-sided renal surgery and occurs in 0.8%–5.1% of such cases.**[72, 73] To prevent CA, all open lymphatics should be secured with clipping and/or ligation. Patients with CA may present with abdominal distention, pain, early satiety and dyspnea from limitation of diaphragmatic movement.[72] Patients may also present with a characteristic milky fluid discharge from a surgical wound.

Prompt diagnosis is critical as prolonged CA may lead to nutritional deficiency and even death. When an abdominal drain is present, drainage of milky fluid may be indicative. **When a drain is not used, the time to presentation is variable and may range from several days to several weeks, or even months after surgery.**[72, 73] As such, urologists must maintain a high index of suspicion. In such cases paracentesis is central to the diagnosis of CA. **The ascites fluid is typically milky, contains a triglyceride level greater than 200 mg/dl and tests positive for chylomicrons.**[73]

Abdominal drain placement allows for symptomatic improvement and monitoring of CA for resolution. Some practitioners, to minimize risks of infection, prefer serial paracenteses over drain placement. **Dietary modification with a high protein and low fat diet with medium chain triglycerides is usually the first intervention attempted. Medium chain triglycerides are absorbed directly from the intestine and transferred as free fatty acids and glycerol directly to the liver, reducing the production and flow of chyle.**[72, 73] Patients who fail this strategy should be placed on bowel rest with nothing by mouth and started on total parenteral nutrition. Octreotide, a synthetic somatostatin analog, may reduce lymphatic drainage and allow for resolution; yet, it is extremely costly and evidence of its efficacy is circumstantial at best.[73] When CA persists despite medical management, more invasive interventions including lymphangiography with lymphatic embolization and surgical lymphatic ligation may be indicated.

Superior mesenteric artery injury. Although only a handful of reports exist regarding SMA injury during renal surgery, this is nearly universally a devastating complication and a renal surgeon's "never event." The SMA is an unpaired artery that arises on the anterior surface of the aorta at the level of L1 and is responsible for supplying the pancreas and bowel from the lower duodenum through two-thirds of the transverse colon. Given its location, SMA injury/ligation most often arises when it is mistaken for the left renal artery. As such, the surgeon should always ask when taking the renal artery,

"Am I sure that this is not the SMA?" Risk factors include left-sided renal surgery, bulky perihilar adenopathy, perinephric inflammation and large renal mass. **If SMA injury is not recognized intraoperatively, it often results in bowel necrosis and patient death. Thus, the importance of intraoperative diagnosis and repair cannot be overemphasized. Vascular surgery should be consulted emergently when SMA injury occurs or is suspected. Reviewing preoperative cross-sectional imaging to understand the anatomical relationship between the SMA and left renal artery can help minimize the risk of SMA injury.**[74]

DID YOU KNOW?

- Being cognizant of the possible complications of renal surgery not only allows for proper preoperative risk stratification and counseling of patients, but also facilitates prompt diagnosis and management of complications when they arise.
- Postoperative hemorrhage is a major complication of PN. Risk factors for hemorrhage after PN include larger tumor size, endophytic nature of the tumor and intraoperative blood loss.
- RAVF and RAP can occur following partial nephrectomy. Large tumor size and high anatomical tumor complexity are significant risk factors for these post-surgical issues.
- Recent data suggest that surgically induced CKD may be associated with a lower risk of progression and mortality than medical CKD, indicating that they may be distinct clinical entities.
- The diagnosis of rhabdomyolysis after renal surgery is best confirmed with an elevated plasma creatinine kinase level. Plasma myoglobin is not as sensitive as creatinine kinase for diagnosis because of its short half-life (2–4 hours for myoglobin vs 1.5 days for creatinine kinase).

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Study Questions Volume 39 Lesson 39

1. A 52-year-old man is in the emergency room 14 days after a robotic right partial nephrectomy with right flank pain, dizziness, fatigue and gross hematuria. His vital signs are stable; however, his serum hemoglobin is currently 8 gm/dl from a serum hemoglobin of 12 gm/dl at discharge on postoperative day 2. The next step is
   a. bed rest, serial monitoring and transfusions as needed
   b. CT angiography
   c. percutaneous angiography
   d. emergent surgical reexploration

2. A 52-year-old woman with a healthy contralateral kidney undergoes a complex open right partial nephrectomy for a large endophytic tumor with a warm ischemia time of 25 minutes. A 15Fr abdominal drain is placed in the right retroperitoneum at the time of surgery. The abdominal drain output is scant for the first few days. On postoperative day 4 the patient's drain output significantly increases to 500 ml per day. The creatinine level of the drain output is 70 mg/dl. The best explanation for the sudden increase in drain output is
   a. insufficient size of abdominal drain
   b. improper positioning of abdominal drain
   c. delayed thermal injury to the ureteropelvic junction
   d. ipsilateral kidney function recovery after acute tubular necrosis from prolonged warm ischemia

3. A 58-year-old morbidly obese man undergoes a prolonged robotic left partial nephrectomy for a complex endophytic mass. At 12 hours postoperatively he has pain out of proportion to examination on his right gluteal and lateral quadriceps muscles, and his urine appears to be "tea-colored." The best test for diagnosis is
   a. urinary myoglobin
   b. plasma myoglobin
   c. plasma creatinine kinase
   d. basic metabolic panel

4. During mobilization of the kidney during a laparoscopic right radical nephrectomy the anesthesiologist notes decreased oxygen saturation, increased airway pressures and increased end tidal CO_2. On inspection there is billowing of the right hemidiaphragm and a 1 cm diaphragmatic defect with visible entry into the pleural cavity. The patient remains hemodynamically stable. The next step is
   a. place a chest tube and abort the procedure
   b. place a chest tube and continue the operation
   c. convert to open surgery, repair diaphragm and continue open radical nephrectomy
   d. evacuate air from the pleural cavity, repair diaphragmatic defect and continue the operation

5. A 54-year-old woman has abdominal pain after eating, nausea and vomiting 4 days after laparoscopic left radical nephrectomy. CT demonstrates a 7 cm fluid collection in the left renal fossa. A percutaneous drain is placed, which returns cloudy fluid with pH 9.8 and amylase 9000 U/l. The next step is
   a. high protein and low fat diet with medium chain triglycerides
   b. nasogastric tube and bowel rest
   c. surgical exploration with ligation of fistula site
   d. surgical exploration with distal pancreatectomy

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