Medical Management of Tumor Lysis Syndrome, Postprocedural Pain, and Venous Thromboembolism Following Interventional Radiology Procedures

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Abstract

Keywords

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- medical management

The rapid expansion of minimally invasive image-guided procedures has led to their extensive use in the interdisciplinary management of patients with vascular, hepatobiliary, genitourinary, and oncologic diseases. Given the increased availability and breadth of these procedures, it is important for physicians to be aware of common complications and their management. In this article, the authors describe management of select common complications from interventional radiology procedures including tumor lysis syndrome, acute on chronic postprocedural pain, and venous thromboembolism. These complications are discussed in detail and their medical management is outlined according to generally accepted practice and evidence from the literature.

Objectives: Upon completion of this article, the reader will be able to discuss routine medical management of select complications of common interventional radiology procedures such as tumor lysis syndrome, acute and chronic postprocedural pain, and venous thromboembolism.

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Since its inception over the last half-century, interventional radiology has developed into a medical subspecialty focusing on a wide variety of disease conditions and a proliferating number of procedures that either replaced or supplemented traditional surgical therapies. Image-guided interventional techniques have led to a significant decrease in complication rates of otherwise nonguided surgical procedures. However, given the invasive nature of the procedures, there remain important complications that still occur despite the available technology and expertise. As the frequency with which patients are treated with interventional procedures increases, it becomes necessary for physicians to become familiar with possible complications and their appropriate medical management. Rapid recognition of these complications will allow for immediate treatment and decreased patient morbidity and mortality. There is a paucity of literature focusing on the medical management of direct compli-

cations from such procedures. In this review, we describe select complications following interventional radiologic procedures, including tumor lysis syndrome (TLS), acute on chronic postprocedural pain, and venous thromboembolism. We will also provide a detailed overview of their evidence-based medical management. Complications are defined as minor (requiring overnight observation) or major (requiring therapy, hospitalization greater than 24 hours, permanent adverse sequelae, or death) based on the Society of Interventional Radiology classification system.¹

Tumor Lysis Syndrome

TLS is a major oncologic emergency encompassing an accumulation of cellular metabolites, which overwhelms the body's normal homeostatic measures, ultimately leading to acute renal failure, cardiac dysrhythmia, and seizures. Cellular death can be mediated by chemotherapy, radiotherapy, or ischemia, or alternatively may occur spontaneously as a result of rapid tumor division. Cell lysis leads to an efflux of nitrogenous proteins and electrolytes, particularly potassium and phosphorus. Binding of calcium to free phosphate can secondarily result in hypocalcemia and calcium-phosphate stone deposition. Purine degradation leads to hyperuricemia and deposition of uric acid crystals in renal collecting tubules. Together, calcium and uric acid–based calculi may lead to obstructive nephropathy. Hyperkalemia and hypocalcemia may lead to cardiac arrhythmia and arrest. These metabolic alterations may also lead to nervous system defects, life-threatening seizures, and death.

TLS is best recognized as a consequence of treatment of hematological malignancies such as leukemia and lymphoma. More rarely, it has been noted to develop in adult patients with solid tumors.^{2–4} Case reports have documented TLS following treatments of breast cancer, small cell cancer, germ cell tumors, gastrointestinal adenocarcinoma, medulloblastoma, and hepatocellular carcinoma. With regard to interventional treatments for hepatocellular carcinoma, TLS has been reported following radiofrequency ablation,⁵ thalidomide therapy,⁶ transarterial chemoembolization (TACE),^{7–9} and sorafenib infusion.^{10,11}

The most widely accepted diagnostic criteria for TLS are those proposed by Cairo and Bishop in 2004.¹² Laboratory TLS is defined by a clinically silent derangement of metabolites, with at least two criteria (► **Table 1**) met within 3 days before

the start of the intervention or 7 days afterward. Clinical TLS is defined by laboratory TLS plus one clinical manifestation of renal injury, cardiac dysrhythmia, or seizure not attributable as an adverse effect of the intervention. Renal injury is evidenced by an increase in creatinine greater than 1.5 times the upper limit of normal, or oliguria with an average urine output of < 0.5 mL/kg/h for more than 6 hours. The TLS criteria was refined by Howard et al in 2011 to include (1) having at least two metabolic abnormalities present during the same 24-hour period within 3 days before the intervention or 7 days afterward; (2) removal of the 25% change from baseline as a criterion as these are unlikely to signify clinically important changes; and (3) including any symptomatic hypocalcemia as diagnostic of clinical TLS.¹³ Spontaneous TLS is defined by laboratory or clinical TLS in patients with rapidly proliferating malignancies who have not received cytotoxic intervention.

Early stratification of patients for risk of TLS is critical to effective management and prevention of sequelae. Risk factors for TLS include rapidly proliferating tumors, bulky malignancies, large area of tumor necrosis after therapy, renal dysfunction prior to therapy, exposure to nephrotoxic agents, and baseline elevation of phosphorus, potassium, and uric acid.¹⁴ In case reports attributing TLS to interventional treatments for hepatocellular carcinoma, tumor sizes have been greater than 5 cm in diameter, tumor burden greater than 25% of the liver, and baseline renal function and electrolytes have been within normal limits. Patients are considered low to intermediate risk if without evidence of laboratory or clinical TLS but with medium to large cancer sizes, medium to high cell lysis potential, and potential for nephropathy (dehydration, acidosis, hypotension, baseline renal injury). Patients are considered high risk if with established laboratory TLS. Clinical TLS is manifested based on the criterion from Howard et al.¹³

Serum potassium, uric acid, calcium, phosphate, lactate dehydrogenase, and renal function should be closely monitored. Low-risk patients should have daily laboratories, intermediate risk should have laboratories obtained every 8 to 12 hours, high risk should have laboratories obtained every 6 to 8 hours, and established clinical TLS should have laboratories obtained every 4 to 6 hours. The mainstay of therapy includes intravenous hydration, correction of metabolic alterations, and treatment of renal failure. Aggressive hydration is recommended for all patients, as this

Table 1 Cairo–Bishop criteria for laboratory tumor lysis syndrome

Metabolic derangement	Objective criteria	Change from baseline
Hyperuricemia	≥8 mg/dL (476 mmol/L)	25% increase
Hyperkalemia	≥6.0 mEq/L (6 mmol/L)	25% increase
Hyperphosphatemia	≥4.5 mg/dL (1.45 mmol/L) in adults, or 6.5 mg/dL (2.1 mmol/L) for children	25% increase
Hypocalcemia	Corrected calcium < 7.0 mg/dL (1.75 mmol/L), or ionized calcium < 1.12 (0.3 mmol/L)	25% decrease

reduces the risk for calculi formation and obstructive nephropathy. Oral or intravenous allopurinol (110 mg/m² every 8 hours) is recommended for treatment of hyperuricemia in low- or intermediate-risk patients. Adverse effects of allopurinol include rash, dyspepsia, fever, eosinophilia, and gastrointestinal upset. Rasburicase (0.05–0.20 mg/kg), a novel uric acid oxidase agent, has been shown to be more effective than allopurinol for treatment of hyperuricemia with similar rates of adverse effects.¹⁵ However, rasburicase is financially prohibitive and contraindicated in patients with glucose-6-phosphatase dehydrogenase deficiency given an elevated risk of hemolysis. It is therefore recommended that only patients at high risk for nephropathy (uric acid levels > 8) or established clinical TLS be treated with rasburicase. For hyperkalemia, electrocardiogram should be obtained and intravenous calcium gluconate (1–3 g over 3–5 minutes) and oral sodium polystyrene (15 g four times per day) should be administered for potassium greater than 6 mEq/L. Hyperphosphatemia can be controlled with phosphate binders including sevelamer (800–1,600 mg three times a day) and oral calcium acetate (667 mg three times a day). Patients with persisting nephropathy that progresses to acute renal failure despite therapy should be transferred to a critical care unit and have hemodialysis initiated without delay.¹⁶

Postprocedural Pain

Although pain is best defined in the psychological sense, it can be clinically categorized into three etiologies: nociceptive, inflammatory, and neuropathic pain. Nociceptive pain results from activation of high threshold peripheral sensory neuron by an intense mechanical, chemical, or noxious stimulus. Inflammatory pain occurs in response to tissue injury and inflammation, most often in the acute setting. Neuropathic pain arises following injury to nerves or to sensory transmitting systems in the spinal cord and brain. A key feature of neuropathic pain is the combination of sensory loss with paradoxical hypersensitivity.

Pain remains a significant source of cancer-related morbidity, especially in advanced disease where prevalence can range from 40 to 90%.¹⁷ This can become further complicated by intervention. Generally, opiates have been the mainstay for chronic therapy in patients with cancer. In the immediate postprocedural setting, direct activation of nociceptors, inflammation, and nerve injury can lead to a clinical picture dominated by interval development of pain referred to the site of intervention and surrounding tissues. Any motion surrounding the therapeutic site or access point can evoke flares of pain. Hypersensitivity can be present anywhere along the intervened neuroanatomical region. Most patients respond well to opiates and cyclooxygenase (COX) inhibitors. If nerves are injured during the intervention, a neuropathic component might develop immediately and persist in the absence of any noxious or inflammatory stimulus. Neuropathic pain is likely to be resistant to COX inhibitors. Rather, agents such as gabapentin, pregabalin, nortriptyline, or carbamazepine should

be trialed. If systemic therapy fails, neurolysis and nerve block can achieve successful palliation of pain in the majority of patients, particularly for acute on chronic pain related to visceral tumors.¹⁸ Therefore, clinically differentiating neuropathic pain from inflammatory pain (►Table 2) is important in directing therapy. Though these general concepts can be applied to pain control in the postprocedural setting, each case should be treated individually. Frequent reassessment of the patient is necessary, and the regimen should be adjusted according to response to medication, underlying comorbidities that affect the ability of drug excretion (i.e., renal or hepatic dysfunction), baseline respiratory disease, and sensitivity to mental status changes (i.e., patients with dementia).

There is a scarcity of studies published on postprocedural pain control in interventional radiology and their effect on the oncology patient. One major study evaluated 150 patients for objective pain scores 24 hours before and after 9 different interventional procedures, observing significant increases in pain 8 hours after percutaneous biliary procedures, 6 hours after central venous catheter and gastrostomy tube insertion, and 4 hours after esophageal stenting.¹⁹ There was a significant reduction of pain after percutaneous nephrostomy. Positive predictors of acute postprocedural pain include preprocedural pain score, preprocedural analgesia, and significant increase in pain score after the procedure. There is no correlation to age, gender, or type of procedure. While preoperative pain correlates to increased intensity of acute pain, it does not necessarily correlate with chronic postoperative pain. The suggestion was pain control after interventional procedures was inadequate, and that pain medications should be prescribed more liberally after discharge. Other studies centered on pain control following uterine fibroid embolization (UFE) and TACE. The authors will discuss treatment strategies for acute and chronic pain after UFE and TACE, though this can be generally applied to other procedures as well. Furthermore, while new or worsening pain is generally a minor complication after most interventional procedures, it significantly reduces quality of life for patients and can be considered a major complication if resulting in readmission.

Table 2 Clinical presentation of neuropathic pain distinguished against inflammatory pain

Symptoms or signs	Neuropathic	Inflammatory
Spontaneous pain	Yes	Yes
Improves with heat	Rare	Frequent
Worsens to cold	Frequent	Rare
Increased threshold to pain	Frequent	No
Paroxysmal pattern	Frequent	Rare
Burning quality	Frequent	Rare
Throbbing quality	Rare	Frequent
Sensory loss	Frequent	No
Motor deficit	Frequent	No

Pain Following Uterine Fibroid Embolization

Postprocedural pelvic pain occurs in almost all patients within the first 24 to 48 hours following UFE. The pain is felt to be secondary to nonspecific ischemia of the uterus and fibroids.²⁰ Larger fibroid size was found to correlate with increased postprocedural pain.²¹ Pain control is critical to a successful procedure and overall patient satisfaction. Multi-center trials investigating UFE found postprocedural pain to be the most common adverse event necessitating readmission,²¹ and that inadequate pain control regimen on discharge is correlated to a higher incidence of readmission.²²

Inpatient and outpatient protocol has been established for pain control after UFE.^{23,24} The most commonly used medications are opiates, nonsteroidal anti-inflammatory drugs (NSAIDs), and antiemetics, a regimen which is considered adequate for up to 96% of patients. Patients should be initially placed on intravenous hydromorphone by patient-controlled analgesia. Patients should be transitioned to oral medications within the first day after the procedure if tolerable and able to withstand oral intake. If long-term opiates are required for pain control, equianalgesic tables (→Table 3) should be used to convert doses to a new opiate. Further dose calculations are required due to tolerance issues with these medications. Because of incomplete cross-tolerance between opioids, the American Pain society recommends a 25 to 50% dose reduc-

tion calculated on opioid conversion.²⁵ In patients receiving long-acting formulations, breakthrough doses of the same opioid in immediate-release form should be prescribed at 5 to 15% of the patient's 24-hour baseline dose. In general, use of opioids should be minimized given adverse effects of tolerance, respiratory depression, nausea and vomiting, constipation, urinary retention, and pruritus. For opiate-naïve patients, it is reasonable to begin with one to two tablets of oxycodone-acetaminophen 5 to 325 mg every 4 to 6 hours and ibuprofen 800 mg three times a day as needed for pain control. Tramadol represents another outpatient option, dosed at 25 to 50 mg twice a day, though this medication is associated with sedating effects and should be used near bedtime to reinforce proper rest patterns. Neuropathic agents may also be given if neuropathic pain is suspected. Superior hypogastric nerve block has also been used in some centers, with a rate of 7 of 139 (5.0%) patients requiring readmission for pain; however, these procedures are invasive and contain complications of their own.²⁶ Systemic medications should be titrated as needed to control pain to reduce the risk of readmission due to inadequate pain control. The great majority of patients experience adequate pain and symptom relief after being discharged within 5.5 hours of the procedure. Roughly, 30 to 40% of patients experience additional symptoms comprising postembolization syndrome. This manifests

Table 3 Equianalgesic dosages for opiates and suggested starting doses for opioid-naïve patients

Analgesic	Equianalgesic		Starting dose ^a	
	Parenteral	Oral	Parenteral	Oral
Morphine				
Immediate release	10 mg	30 mg	2.5 mg q4h	10 mg q4h
Controlled release (MS Contin)		30 mg		
Extended release (Avinza)		30 mg		30 mg q24h
Hydromorphone				
Immediate release	1.5–2.0 mg	7.5–8.0 mg	0.2 mg q2h	2 mg q4h
Controlled release (Hydromorph Contin)		7.5 mg		
Oxycodone				
Immediate release (Roxicodone, Percocet)		20–30 mg		5 mg q4h
Controlled release (OxyContin)		20–30 mg		10 mg q12h
Oxymorphone				
Immediate release	1 mg	10 mg	0.5 mg q4h	5 mg q4h
Extended release (Opana ER)		10 mg		5 mg q12h
Hydrocodone				
Immediate release (Lortab, Vicodin)		30–45 mg		5 mg q4h
Codeine				
Immediate release	100–130 mg	200 mg	10 mg q4h	15 mg q4h
Controlled release (Codeine)		200 mg		50 mg q12h
Meperidine	75 mg	300 mg		
Fentanyl ^b	100 µg			

^aSuggested starting doses are for opioid-naïve patients and are not equianalgesic.

^bTransdermal form converts as oral morphine 60 mg total daily dose to 25 µg/h fentanyl patch.

as pelvic pain, fatigue, fever, nausea, emesis, and transient elevations in lactate. Etiology is generally attributed to tumor necrosis and ischemic damage as a result of the embolic effect of therapy. Symptoms usually resolve in the first 3 to 4 days, and full recovery is expected within 7 to 10 days. It is hypothesized that smaller-sized particles may lead to reduced ischemic damage, though this remains in investigation through prospective trials. Recommended therapy is supportive, including analgesics and antiemetics as described above.

Pain Following Transarterial Chemoembolization

The most common major adverse effect of TACE is postembolization syndrome, occurring in roughly 80 to 90% of patients following TACE. While the overall incidence of major toxicity has decreased with the use of doxorubicin-loaded beads,²⁷ the incidence of postembolization syndrome remains nearly the same. However, prospective studies suggest the symptoms are associated with tumor response rather than collateral healthy liver damage.²⁸ Patients typically present with right upper quadrant pain, fatigue, fever, nausea, emesis, anorexia, and transient elevations of transaminases and bilirubin. Pain typically correlates with the amount of embolic material used, the number of treatments, and the baseline size of the tumor treated. Similar to the management for postembolization syndrome following UFE, intravenous hydration, intravenous analgesics with next day conversion to oral opiates, and NSAIDs are the mainstay of pain control. Antiemetics should be used to reduce symptoms of nausea and vomiting. Roughly 50% of patients can be discharged from the hospital the day following chemoembolization, with an average length of stay of 1.5 days.²⁹ Typically, reasons for longer admissions are due to serious adverse effects (i.e., liver abscess or infarction), at which point the patient should be maintained on the regimen which optimizes pain relief.

Venous Thrombosis and Thromboembolism

Venous thromboembolism, which encompasses deep venous thrombosis (DVT) and pulmonary embolism (PE), is a common life-threatening complication after any interventional procedure. Per review of several prospective and retrospective studies, the incidence of venous thrombosis varies widely (12–67%) and depends on the tumor type, presence of metastasis, prothrombotic states (i.e., factor V Leiden and prothrombin gene mutation 20210), infection, hormonal therapy, and underlying comorbidities.^{30–35} Clinically evident PE occurs at an incidence of 1 in 400 patients in the postprocedural setting. In patients with long-term central venous catheters, increased venous stasis and endothelial injury may also increase the risk of DVT and subsequent PE. Thromboembolism occurs more often in those with extended hospital stays due to major complications. Venous thromboembolism is considered provoked in the presence of temporary or reversible risk factors (surgery, trauma, immobilization, pregnancy, oral contraceptives, and hormone replacement therapy) within the last 6 weeks to 3 months prior to diagnosis.³⁶ Use of intermittent pneumatic compression devices on legs and prophylactic unfractionated

heparin (UFH) may reduce risk, but for high risk patients, low-molecular-weight heparin (LMWH) should be used at prophylactic dosing.

The most common patient presentation for pulmonary embolus includes sudden onset dyspnea (50%), pleuritic chest pain (39%), cough (23%), substernal chest pain (15%), fever (10%), hemoptysis (8%), and signs of DVT (25%).³⁷ In patients with preexisting cardiopulmonary disease, worsening dyspnea may be the only presenting symptom indicative of PE. Electrocardiogram may demonstrate the classic S1Q3T3 (S-wave in lead 1, Q wave in lead 3, and T wave inversion in lead 3) and right ventricular strain; however, these signs are more often absent. The utility of electrocardiogram is mostly in excluding other diagnoses (i.e., heart failure, myocardial infarct, or tamponade). Wells criteria or the revised Geneva score can be used to clinically stratify as low, intermediate or high risk for pulmonary embolus (►Table 4).^{38,39}

A diagnostic and therapeutic algorithm for acute PE is outlined in ►Fig. 1. D-dimer levels are elevated in plasma during any thrombotic event due to simultaneous activation of coagulation and fibrinolysis. The high negative predictive value and sensitivity (~95%) of D-dimer testing makes acute PE or DVT unlikely if testing proves negative. However, the positive predictive value and specificity (~40%) of D-dimer testing is low, making this test not useful for confirmation of PE. CT angiogram is considered diagnostic if PE is found at the segmental or more proximal level, with sensitivity of 83% and specificity of 96%.⁴⁰ With intermediate to high clinical probability of PE, the positive predictive value was 92 to 96%. Alternatively, with low or intermediate probability of PE, negative predictive value of CT was 89 to 96%, while positive predictive value is much lower (58%). The data underlie the importance of pretest stratification prior to CT imaging to prevent misdiagnosis of PE. Echocardiography may demonstrate right ventricular overload or dilation, which may aid in risk stratification in hemodynamically unstable patients. Other signs, including depressed right ventricular free wall contractility (McConnell sign) and a disturbed right ventricular ejection pattern, have demonstrated high positive predictive value for PE. However, other conditions presenting with right ventricular dyskinesia or infarction may mimic signs of PE leading to misdiagnosis. Therefore, echocardiography is not recommended for confirmation of PE in hemodynamically stable, normotensive patients with low to intermediate clinical probability.

Medical management for pulmonary embolus includes clot stabilization. Parenteral anticoagulation should be administered over the first 5 to 10 days. In patients with high or intermediate clinical probability for PE, parenteral anticoagulation should be initiated while awaiting the results of diagnostic tests. Immediate anticoagulation can be achieved with parenteral anticoagulants such as intravenous UFH, subcutaneous LMWH, or subcutaneous fondaparinux. LMWH and fondaparinux are preferred over UFH for initial anticoagulation in PE or in high-risk patients, as they carry a lower risk of inducing major bleeding and heparin-induced thrombocytopenia.^{41,42} On the other hand, UFH is recommended for patients in whom primary reperfusion is

Table 4 Modified Wells criteria for predicting pretest probability of pulmonary embolism

	Score
Clinical characteristic	
Previous PE or DVT	1.5
Heart rate >100 beats per min	1.5
Surgery or immobilization within last 30 d	1.5
Clinical signs of DVT	3
Alternative diagnosis less likely than PE	3
Hemoptysis	1
Cancer (treated within the last 6 mo)	1
Clinical probability of pulmonary embolism	
Low	0–1
Intermediate	2–6
High	≥6

considered, those with serious renal impairment ($\text{CrCl} < 30$) or severe obesity ($\text{BMI} > 40$). These recommendations are based on the short half-life of UFH (1 hour), the ease of monitoring its anticoagulant effects, and its rapid reversal by protamine. The dosing of UFH is titrated based on the

activated partial thromboplastin time. Warfarin should be initiated immediately, aiming for an International Normalized Ratio of 2.0 to 3.0 over the next 3 to 5 days. Warfarin should be continued for at least 3 months. Close follow-up is indicated as outpatient due to the increased risk of bleeding and interactions of warfarin with other medications. Catheter-directed therapy and percutaneous mechanical thrombectomy may be used as a primary therapy for an acute thrombotic event, but is reserved as salvage therapy or for thrombus involving the great vessels (i.e., vena cava). Such therapy also has a role in patients with contraindications to continuous anticoagulation or the use of thrombolytic agents. It is, however, suggested that in the absence of contraindications to the use of thrombolytic agents, mechanical thrombectomy and catheter-directed therapy be done in conjunction with pharmacological thrombolysis.⁴³

Early recurrence of thromboembolism in anticoagulated patients amounts to 2.0% at 2 weeks, 6.4% at 3 months, and 8% at 6 months.⁴⁴ Late recurrence has been reported to reach 13% at 1 year, 23% at 5 years, and 30% at 10 years.⁴⁴ For patients with repeat episodes of pulmonary thromboembolism, or patients who are poor candidates for anticoagulation, placement of an inferior vena cava filter is recommended. Retrieval filters should be removed as soon as patients are deemed safe for anticoagulant use, given 10% overall risk of filter migration, tilting or deformation, penetration of the cava wall

Fig. 1 Algorithm for diagnosis and management of suspected pulmonary thromboembolism. Modified from European Heart Journal 2014 Guidelines on the diagnosis and management of acute pulmonary embolism.

by filter limbs, fracturing of the filter and embolization of fragments, and thrombosis of the device within the inferior vena cava.⁴⁵

For treatment of DVT, patients with provoked thrombosis should be anticoagulated for only 3 to 6 months, as longer durations of anticoagulation may lead to increased risk of bleeding without significantly increased therapeutic benefit.⁴⁶ Choice of anticoagulant in high-risk patients has been studied in various trials. The CLOT trial compared six months of warfarin to subcutaneous LMWH dalteparin, and demonstrated significant reduction in recurrent thrombosis at 6 months with dalteparin and no difference in bleeding rates.⁴⁷ The LITE trial compared warfarin to tinzaparin, demonstrating reduced rates of venous thrombosis with tinzaparin but similar rates of bleeding and mortality at 12 months.⁴⁸ The CANTHANOX trial compared warfarin to enoxaparin for 3 months in patients with cancer, and found no significant difference in recurrent thromboembolism or major bleeding, though post-hoc analysis demonstrated fewer bleeding events with enoxaparin.⁴⁹ The ONCENOX trial compared enoxaparin alone to enoxaparin bridge to warfarin, and at 180 days there was no difference between the regimens in prevention of recurrent venous thromboembolism or occurrence of bleeding.⁵⁰ The authors therefore recommend a 3 to 6 months course of LMWH in high-risk patients who develop first-time provoked venous thrombosis. Contraindications for anticoagulant therapy include active major bleeding, severe uncompensated coagulopathy, thrombocytopenia < 50,000, severe platelet dysfunction, uncontrolled hypertension, and or planned surgery or invasive procedures. If patients have a central venous catheter as a source of thrombosis, routine removal of the catheter is not recommended. In stable patients without documented infection who have an ongoing need for access, it is acceptable to administer anticoagulant therapy without catheter removal, provided it continues to be well functioning and correctly positioned.⁴⁶

Conclusion

With the expansion of interventional radiology and increased available of hepatobiliary, genitourinary, and oncologic interventions, their related complications will inevitably become more common. Though these procedures are generally performed within the interventional practice, physicians should be aware of the etiology, early identification, and immediate management of these common postprocedural complications.

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