Vascular Oncologic Emergencies

A review of arterial and venous complications of malignancy and oncologic care that necessitate urgent transcatheter intervention.

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Vascular oncologic emergencies are common and necessitate prompt management. This review examines arterial and venous oncologic emergencies requiring acute transcatheter intervention. Arterial emergencies include hemorrhage, which may be due to the tumor itself, cancer therapy including surgery, and systemic issues. Venous emergencies include venous obstruction such as superior vena cava (SVC) syndrome and venous thromboembolism (VTE). For each type of vascular emergency, the etiology, epidemiology, diagnosis, treatment, and prognosis are discussed.

ARTERIAL ONCOLOGIC EMERGENCIES

Acute arterial bleeding can occur as a consequence of advanced malignancy or treatment. Arterial bleeding can lead to rapid, large-volume blood loss and death. Major hemorrhage occurs in approximately 10% of patients with advanced malignancy.[1] Arterial bleeds can be stratified as threatening (artery involvement without blood loss such as when a tumor encases a major artery), impending (arterial wall disruption that remains contained such as a sentinel bleed from a pseudoaneurysm prior to free rupture), or immediate (rapid, uncontained, large-volume blood loss requiring volume resuscitation followed by immediate intervention).[2,3]

Acute Tumoral Bleeding

The incidence of acute malignancy–related arterial bleeding depends on the type and stage of malignancy.[4,5] Risk factors for acute tumoral bleeding include large tumor size and proximity of the tumor to a large artery.[2] This section highlights three examples of hemorrhage due to tumors involving the gastrointestinal (GI) tract, ureter, and carotid artery.

GI bleeding. Similar to patients without cancer, the most common causes of nonvariceal GI bleeding in cancer patients are ulcers, arteriovenous malformations, and angiodysplasias. Less than 5% of upper GI bleeds are secondary to direct tumor invasion of the GI tract, most commonly gastric adenocarcinoma.[6,7] In the lower GI tract, up to a quarter of bleeds may be due to tumors, such as small bowel lymphoma, intestinal adenocarcinoma, and metastases.[8,9] Locally advanced tumors can encase large vessels and erode through the GI tract, leading to aortoenteric or iliac artery-enteric fistulas presenting with brisk bleeding and hypotensive shock.

Initial management of GI bleeding involves resuscitation and localization of the suspected source of bleeding. Upper GI bleeding normally presents with hematemesis and/or melena; lower GI bleeding presents with hematochezia. Nasogastric lavage may help distinguish these possibilities.[10] Management typically entails upper or lower endoscopy to localize the bleeding source and potential endoscopic interventions.[11,12] For patients who cannot tolerate endoscopy, noninvasive radiologic workup using CTA or tagged red blood cell (RBC) scan can be considered.[10] RBC scintigraphy is more sensitive for detecting GI bleed (94% vs 85%), but positive findings are less likely to be corroborated on percutaneous angiography (29%) than with CTA (68%).[13,14] CTA requires less preparation and can be performed more expeditiously, allowing for more rapid definitive treatment.[14,15] CTA increases the probability that sites of GI hemorrhage are localized by percutaneous angiography, as it may identify tumors' anatomic relationship with the GI tract and possible arterial targets for more selective angiography.[16]

Endovascular treatment of tumor-associated GI bleeding can be achieved using embolic agents or, less commonly, stent grafts. Embolization is well-tolerated for upper GI bleeds because of the presence of vast collateral networks.[6] The risk of ischemic complications can be reduced by superselective transarterial embolization.[6,17-19] Active extravasation may not be identified angiographically because bleeding is intermittent.[6,20,21] Even when active

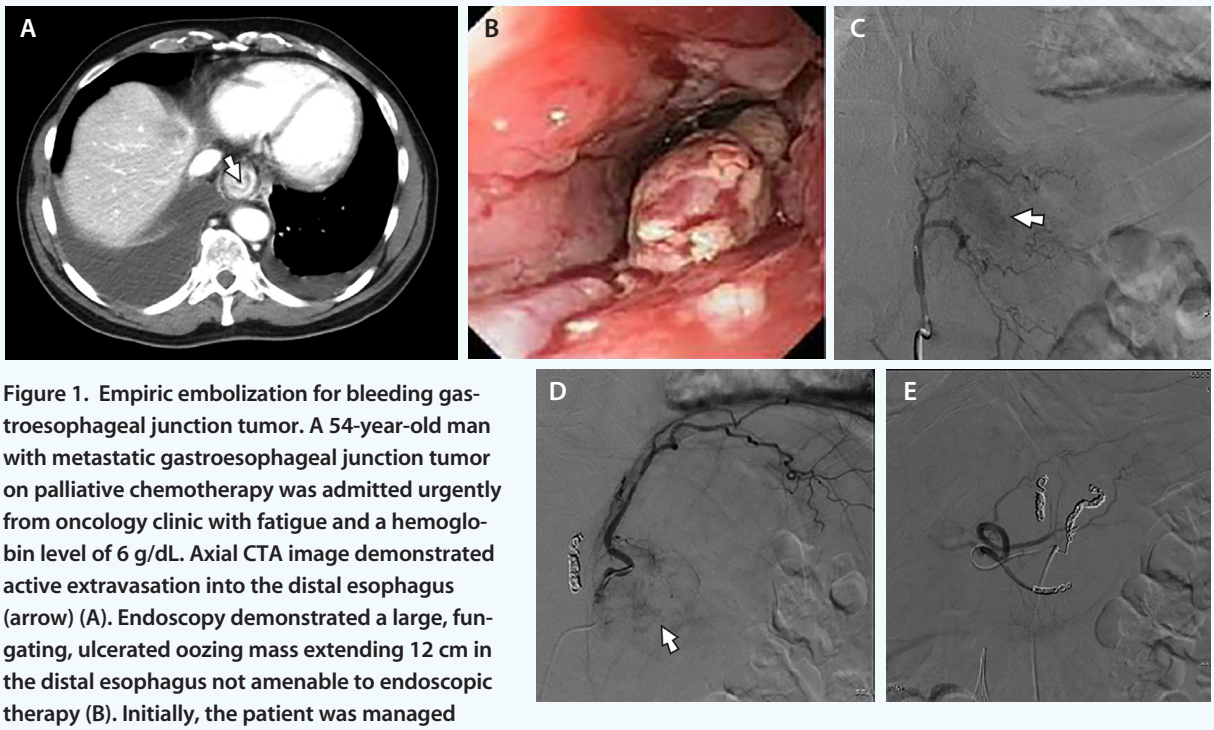

Figure 1. Empiric embolization for bleeding gastroesophageal junction tumor. A 54-year-old man with metastatic gastroesophageal junction tumor on palliative chemotherapy was admitted urgently from oncology clinic with fatigue and a hemoglobin level of 6 g/dL. Axial CTA image demonstrated active extravasation into the distal esophagus (arrow) (A). Endoscopy demonstrated a large, fungating, ulcerated oozing mass extending 12 cm in the distal esophagus not amenable to endoscopic therapy (B). Initially, the patient was managed conservatively; external radiation was not possible as the area had been previously irradiated. The patient had persistent transfusion-dependent tumoral bleeding, prompting referral for transcatheter evaluation. Visceral arteriography demonstrated tumor blush from selective left gastric (C) and left phrenic (D) arteriograms (arrows); coil embolization of these vessels and the right gastric artery was performed (E). After embolization, the bleeding stopped, and the patient was discharged home. He passed away 2 months later from progression of metastatic disease.

extravasation is not seen, abnormalities such as arterial spasm or pseudoaneurysm in the region of expected hemorrhage can identify a target vessel for embolization. Empiric embolization can be considered and has a high success rate of nearly 70% in terms of reducing hemorrhage (Figure 1).[6,21] In the setting of hemorrhage secondary to GI tumor, recurrent bleeding necessitating reintervention is not uncommon, and as such, definitive surgery often leads to the best outcomes.[22,23]

Ureteroarterial fistula. Gross hematuria frequently occurs in patients with genitourinary cancers. Rarely, massive hemorrhage results from a ureteroarterial fistula. Such communications can occur from tumor invasion of the ureter and nearby iliac artery, with gynecologic, rectal, and bladder cancers being the most common malignancies.[24] Fistulas can result from ureteral erosion in patients who have undergone ureteral stenting, surgery, or radiation.[25,26] When ureteroarterial fistulas are suspected, pelvic angiography may have a higher sensitivity than retrograde pyelography, cystoscopy, or CT.[27,28] Ureterography is not recommended because it can dislodge an existing clot or tear the fistula.[29]

Management of ureteroarterial fistulas involves close collaboration with urologists, as they may be noted during ureteral stent exchange. Severe bleeding requires both arterial and ureteral intervention, often with ureteral stent placement and occasionally ureteral repair.[24] Historically, arterial treatment involved open arterial repair or bypass, but now endovascular coverage of the fistula with stent grafts is preferred given its minimally invasive approach and ability to provide immediate bleeding control (Figure 2). Complications include recurrent hemorrhage, graft infection, and ipsilateral lower extremity morbidity.[30,31] Long-term antibiotic prophylaxis and anticoagulation are occasionally used, although there is limited evidence in support of their use.[25,32,33]

Carotid blowout. Carotid blowout, or carotid artery rupture, is an emergency most commonly presenting in patients with head and neck cancer when the skin overlying the carotid artery has lost its integrity (eg, after radical neck dissection or radiation therapy, particularly reirradiation for tumor recurrence).[3] The incidence of carotid blowout ranges from 1% to 3% in patients receiving radiation and about 4% after radical neck dissection.[34-37]

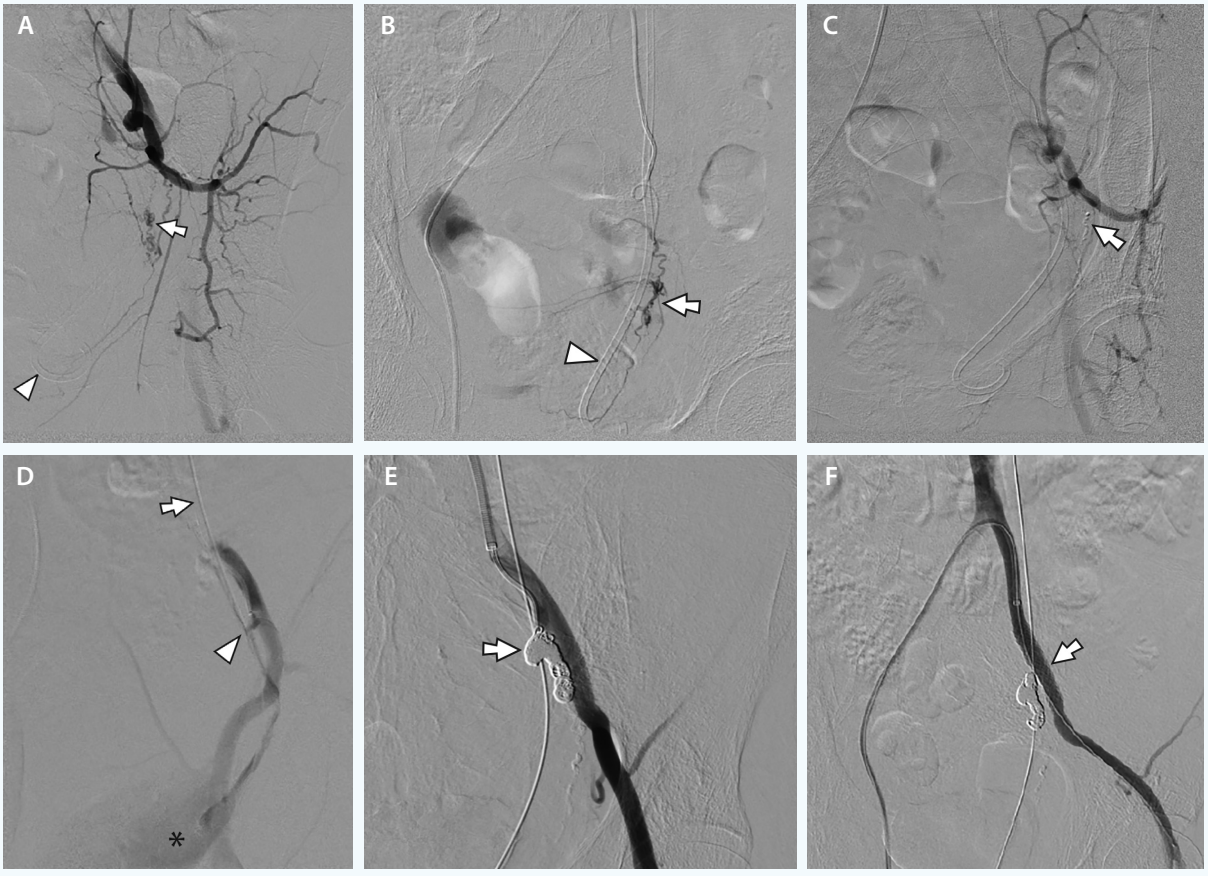

Figure 2. Ureteroarterial fistula. A 73-year-old woman with recurrent uterine carcinosarcoma developed left hydronephrosis after pelvic radiation, which was managed with a ureteral stent. During routine stent exchange, the urologist noted persistent bright red hematuria arising from the left ureteral orifice. The patient's hemoglobin decreased from 10 to 6 g/dL and she was referred for arteriography. A left internal iliac arteriogram (A) with a more selective run (B) demonstrated abnormal vasculature and extravasation (arrows) from the branches of the internal iliac artery in close proximity to the ureteral stent, which are seen subtracted out (arrowheads). The abnormal branch was embolized with glue, which is seen as a subtraction artifact (arrow), with no residual abnormal vessels in the region of the stent (C). The hemoglobin normalized with transfusion and vital signs were normal and stable. However, within 1 week, there was ongoing hematuria and persistent transfusion-dependent anemia. The ureteral stent was removed over a wire (arrow) and left internal iliac arteriography demonstrated a brisk communication between the proximal left internal iliac artery and the ureter (arrowhead), with contrast flowing into the bladder (asterisk) (D). The internal iliac artery was coil embolized, with the coil mass seen as a subtraction artifact (arrow) (E). Given the recurrent bleeding, a covered stent (arrow) was placed to entirely exclude the internal iliac artery in addition to coil embolization (F). The patient stabilized and the hematuria resolved after the second procedure.

Carotid blowout can originate from either the internal carotid or branches of the external carotid artery and most commonly occurs proximal to the carotid bifurcation.[38] Bleeding can lead to exsanguination and potentially upper airway obstruction.[2]

Carotid blowout is associated with very high mortality and must be treated emergently. Initial management includes airway protection, fluid resuscitation, direct compression with epinephrine packing, and potentially inotropic agents.[3] Historically, open surgery was required, with high mortality and neurologic morbidity.[39] Newer endovascular techniques are associated with lower complication rates. Coil embolization can be successfully used in patients with carotid blowout involving the branches of the external carotid artery.[40] For common or proximal internal carotid bleeds, covered stent grafts can immediately reconstruct the vessel wall, controlling the hemorrhage and limiting ischemic complications.[39] Covered stent grafts are successful in controlling bleeding acutely but are associated with stent infection and rebleeding.[39,40] Additionally, patients require dual antiplatelet therapy with aspirin and P2Y12 inhibitor for 1 to 3 months after stent placement,

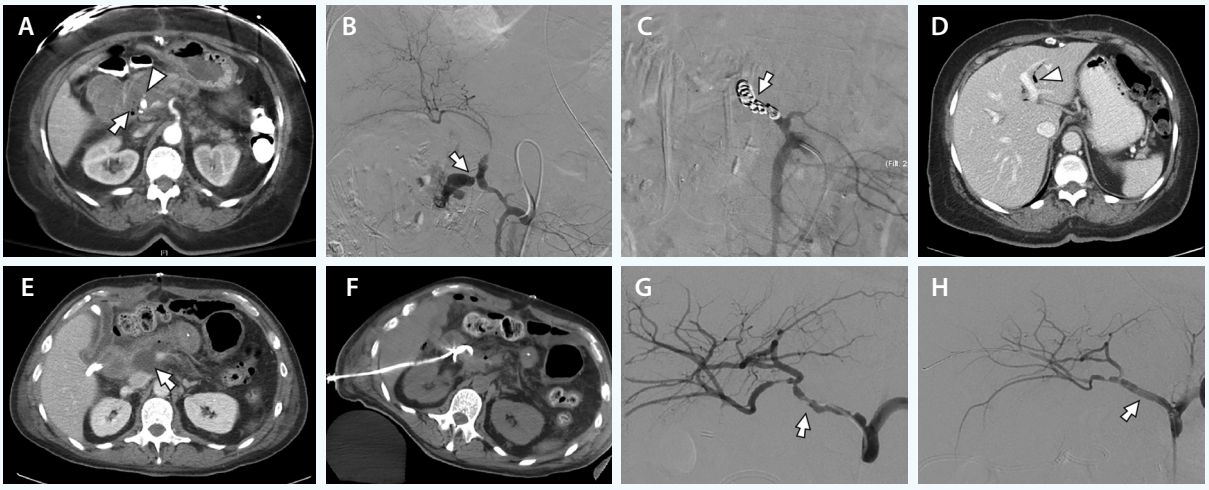

Figure 3. Gastroduodenal hemorrhage after the Whipple procedure. A 76-year-old man with pancreatic adenocarcinoma presented 1 month after a Whipple procedure with hematemesis. Upper endoscopy showed a patent anastomosis with no active bleeding. Axial CT image with contrast showed a fluid and gas collection in the pancreatic bed (arrow) and a small focus of contrast extravasation (arrowhead) (A). The patient had a bout of hematemesis and loss of consciousness with decline in systolic blood pressure to 70 mm Hg and a corresponding decline in hemoglobin from 10 to 8 g/dL. The patient was urgently intubated and transferred to interventional radiology with vasopressor support initiated. Visceral arteriography demonstrated a replaced common hepatic artery to the superior mesenteric artery, with rapid active extravasation from the gastroduodenal artery stump (arrow) (B). Given the patient's hemodynamic instability, coil embolization (arrow) of the proper to common hepatic artery was performed, covering the area of extravasation (C). Axial CT images from follow-up 6 months later showed no atrophy or evidence of prior hepatic necrosis; despite loss of arterial supply, portal venous flow supported liver function. Pneumobilia (arrowhead) was noted, as a result of the bilioenteric anastomosis portion of the Whipple procedure (D). In a companion case, a 56-year-old man with pancreatic adenocarcinoma presenting 2 weeks after Whipple procedure with fevers and leukocytosis was found to have a collection in the operative bed on contrast-enhanced axial CT (E). The patient underwent transhepatic drainage of the collection, yielding pus (F). Within 1 day, output from the drain became bloody and the patient became hypotensive. Vasopressors were initiated and the patient transferred to interventional radiology. Celiac arteriography demonstrated irregularity and spasm of the common and proper hepatic artery (arrow), although there was no active extravasation (G). A covered stent was placed across the gastroduodenal stump (arrow), with preservation of distal hepatic arterial blood flow (H). After the procedure, the patient improved with reduced pressor requirement; however, the patient ultimately expired the following day.

followed by lifelong aspirin therapy.[40,41] Coil embolization with sacrifice of the internal carotid artery is associated with a lower chance of rebleeding but a higher risk of ischemic stroke.[40]

Postoperative and Postprocedural Hemorrhage

Cancer patients undergoing surgery or other interventions may be at higher risk of bleeding due to preprocedural therapies including systemic therapy and radiotherapy. For example, bevacizumab therapy is associated with poor wound healing and potential vascular compromise.[42] High-dose radiation can result in friable tissue and lead to vessel wall disruption, rendering a site prone to bleeding. Postoperative and postprocedural bleeds are relatively uncommon but comprise a large proportion of arterial bleeds seen in cancer patients. Hemorrhage can occur after any invasive procedure, such as percutaneous biopsy, ablation, or surgery. One example of postsurgical hemorrhage occurs after pancreatectomy.

Postpancreatectomy hemorrhage (PPH). PPH is an uncommon but severe complication of pancreatic resections associated with high morbidity and mortality. PPH encompasses all postoperative bleeding episodes, the most-feared complication being massive hemorrhage due to blowout of the visceral arteries.[43,44] This most commonly involves the gastroduodenal artery and potentially the branches of the superior mesenteric artery. Pancreatic leaks lead to extravasated pancreatic enzymes, which induce an autodigestive effect on the vessel wall (Figure 3).[43-47]

According to the International Study Group of Pancreatic Surgery, early PPH occurs within 24 hours of operation and tends to result from underlying coagulopathy or technical failure of hemostasis.[44] In contrast, late PPH can occur days to weeks after the operation and results from surgical com-

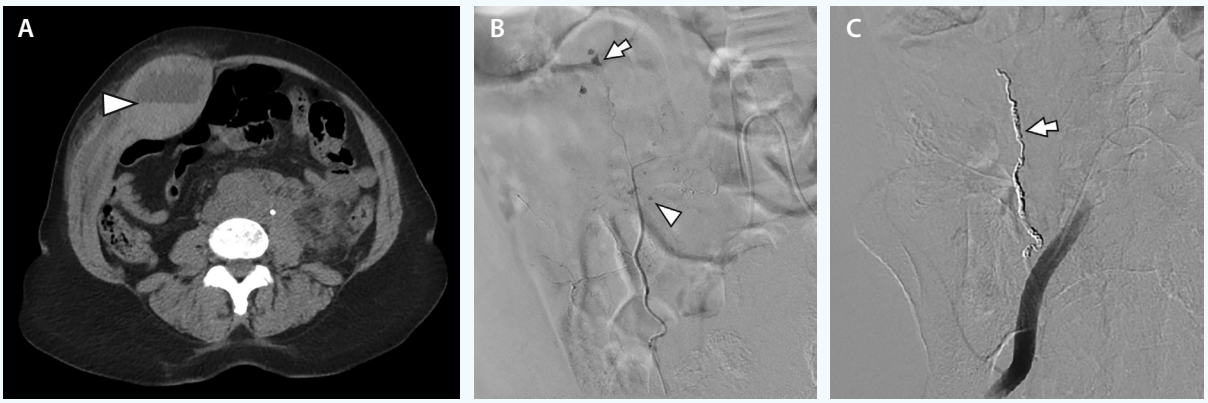

Figure 4. Anticoagulation-associated hemorrhage. A 58-year-old woman with multiple myeloma on enoxaparin for DVT and saddle PE developed severe sudden-onset mid-right abdominal pain, tachycardia, and thrombocytopenia, and hemoglobin decreased from 10 to 6 g/dL. A noncontrast axial CT image demonstrated a right rectus sheath hematoma with a hematocrit level (arrowhead) (A). A right inferior epigastric arteriogram demonstrated foci of active extravasation (arrow) with smaller puddles of contrast (arrowhead), reflecting additional small vessel tearing as the muscle enlarges due to expanding hematoma (B). The inferior epigastric artery was coil embolized (arrow), with coils placed distal to the furthest area of extravasation to the proximal aspect of the vessel to prevent backfilling of the artery (C). Additionally, an inferior vena cava (IVC) filter was placed because the patient was no longer a candidate for anticoagulation. After embolization, the patient's hemoglobin normalized after transfusion of packed RBCs and remained stable.

plications such as abscess or pseudoaneurysm development, erosion of peripancreatic vessels, and anastomotic breakdown.[2,44] PPH can be classified as intraluminal (intraenteric), manifesting as GI bleed, or extraluminal (extraenteric), presenting as bloody output from intra-abdominal surgical drains and/or a significant hemoglobin drop.[48] Arterial pseudoaneurysms, a well-known cause of extraluminal PPH, often present after an episode of sentinel bleeding.[44]

Patients with early PPH often present with extraluminal bleeding and may require immediate reoperation to correct the underlying hemostatic failure.[45,48] In late PPH, patients undergo aggressive workup with upper endoscopy or angiography to localize the bleeding source.[45] Minimally invasive endovascular treatment with coil embolization or stent graft placement can be performed to control the bleeding.[49] Coil embolization confers high rates of technical success. Occasionally, the source of PPH is the hepatic artery. Typically, embolization of the hepatic artery does not cause hepatic infarct when the portal vein is patent and there is hepatopedal flow. In the setting of a compromised portal vein, embolization of the hepatic artery can lead to hepatic infarct and/or biliary ischemia.[50] In this situation, placement of a stent graft is preferred because it may preserve hepatic arterial supply, although in small vessels, stent grafts are associated with thrombosis.[51,52]

Hemorrhage Due to Systemic Cancer-Related Issues

Oncologic patients' risk of arterial hemorrhage is increased due to systemic cancer-related issues, including thrombocytopenia as a result of chemotherapy, liver dysfunction, or malignancy. Cancer often induces a hypercoagulable state necessitating anticoagulant or antiplatelet therapy, which may cause hemorrhagic complications.[53] In the absence of trauma, spontaneous extraperitoneal hemorrhage is most often associated with anticoagulant or antiplatelet therapy.[54] The mainstay of management includes withdrawal and/or reversal of anticoagulant/antiplatelet therapy; however, when life-threatening hemorrhage occurs, particularly when active extravasation is identified by CTA, transcatheter selective embolization can prevent morbidity and mortality (Figure 4).[54]

VENOUS ONCOLOGIC EMERGENCIES

VTE is common among cancer patients and is a leading cause of death. Although most (70%) cancer patients die from progression of disease, 10% die from VTE.[55] Certain acute presentations of VTE necessitate rapid intervention that may involve peripheral therapies such as systemic thrombolysis or local therapies including thrombectomy or catheter-directed thrombolysis.

Pulmonary Embolism

Pulmonary embolism (PE) is stratified as massive or submassive.[56] Massive PE is defined by sustained hypotension (systolic blood pressure < 90 mm Hg for > 15 min), inotropic support requirement, pulselessness, or profound bradycardia with shock and is associated with a high mortality rate of 25% to 65%, therefore requiring emergent intervention including systemic thrombolysis, catheter-directed therapy, or surgical embolectomy.[57]

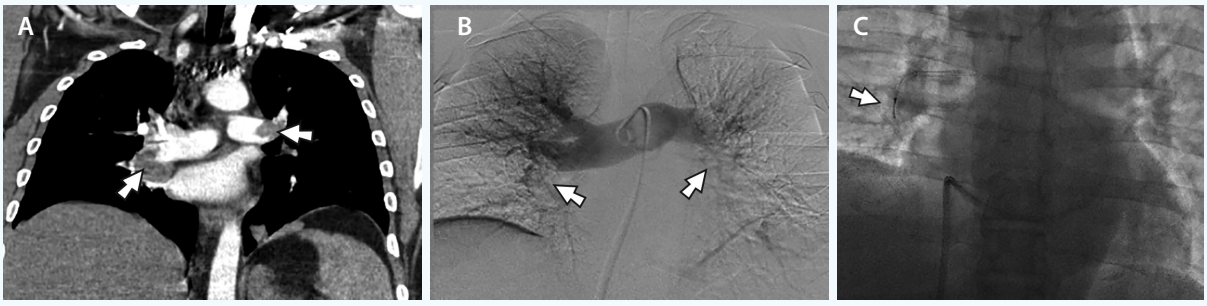

Figure 5. Submassive PE treated with mechanical thrombectomy. A 35-year-old woman with persistent vaginal bleeding due to endometrial cancer presented with dyspnea and was found to have left lower extremity DVT and PE. Contrast-enhanced coronal CT demonstrated emboli in bilateral pulmonary arteries (A). The patient had right ventricular dysfunction, elevated brain natriuretic peptide and troponin, and was tachycardic but normotensive. The sPESI was considered > 2. Because the patient was bleeding, thrombolysis was contraindicated, and the patient was referred for mechanical thrombectomy and IVC filter placement. Pulmonary arteriography demonstrated bilateral filling defects in inferior pulmonary artery branches (arrows); main pulmonary artery pressure was 35 mm Hg (B). Mechanical thrombectomy was performed with an aspiration catheter (arrow) (C). The patient developed severe cough after approximately 75% clot removal, so the procedure was terminated. Subsequently, the patient's dyspnea tachycardia resolved. She underwent pelvic radiation for the bleeding tumor such that she could eventually tolerate anticoagulation, allowing for IVC filter retrieval.

Treatment for massive PE depends on whether there are contraindications to systemic thrombolysis. Absolute contraindications to systemic thrombolysis include any active bleeding, GI bleed within 10 days, cardiovascular accident including transient ischemic attack within 2 months, and neurosurgery or intracranial trauma within 3 months.[58] Relative contraindications include cardiopulmonary resuscitation or major nonvascular surgery or trauma within 10 days, uncontrolled hypertension, puncture of noncompressible vessel, intracranial tumor, or recent eye surgery. In patients without contraindications, systemic thrombolysis with intravenous infusion of alteplase, usually administered as a dose of 100 mg over 2 hours, is the preferred treatment given the ease and rapidity of administration.[59] However, systemic thrombolytic administration entails a risk of major hemorrhage, particularly hemorrhagic stroke.[57,60]

Given the high prevalence of comorbidities in cancer patients, systemic thrombolysis is often contraindicated. In such cases or when more rapid removal of clot is deemed necessary, surgical embolectomy or catheter-directed therapy is preferred. Surgical embolectomy allows for en bloc removal of large clot volumes and can be advantageous for patients in hemodynamic shock who need to be bridged using extracorporeal membrane oxygenation.[59] Although surgical embolectomy has historically been associated with high mortality rates, better patient selection and rapid triage have led to improved outcomes data in recent years.[61-63]

Catheter-directed therapy refers to the endovascular removal of embolus by chemical or mechanical means and is recommended for the treatment of massive PE in patients who have failed systemic thrombolysis or have severe cardiovascular compromise such that systemic thrombolysis may not work quickly enough.[58] Catheter-directed therapy includes catheter-directed thrombolysis with local infusion of alteplase (typically 0.5-2 mg/hour performed over several hours) and mechanical or pharmacomechanical embolectomy. Embolectomy may be performed by rotating a pigtail catheter within the clot to fragment it or by aspiration of clot from the pulmonary artery.[64] In patients with absolute contraindications to thrombolysis, mechanical thrombectomy without local infusion of alteplase may be employed.[65] Prospective data comparing outcomes after catheter-directed thrombolysis or pharmacomechanical thrombectomy are lacking.[66]

Patients with PE who do not meet criteria for massive PE can be stratified using the simplified Pulmonary Embolism Severity Index (sPESI).[67] Submassive PE is defined by right ventricular dysfunction or myocardial necrosis demonstrated by imaging (echocardiography or CT), electrocardiographic changes (new right bundle branch block, anteroseptal ST changes, or anteroseptal T wave inversion), or elevated brain natriuretic peptide or troponin levels in the absence of sustained hypotension. The mortality rate of submassive PE is approximately 3%, which has been used a rationale to propose urgent catheter-directed thrombolysis or mechanical thrombectomy (Figure 5).[56] However, prospective data are lacking to support its use in this context.[59] Finally, patients with low sPESI score without signs of right ventricular dysfunction or myocardial necrosis are considered low to low-intermediate risk and can be managed with anticoagulation alone.[59]

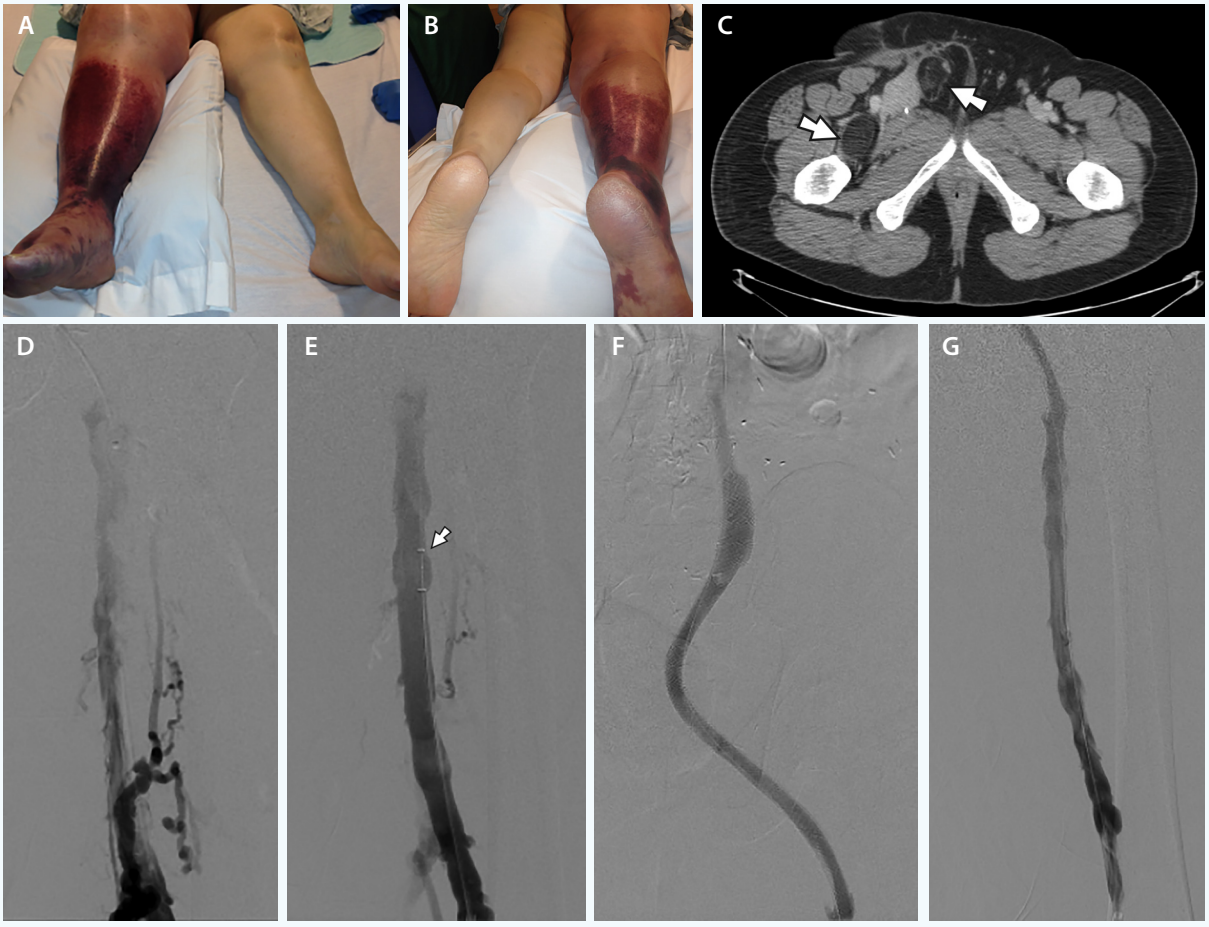

Figure 6. A patient with PCD caused by extensive lower extremity DVT (A, B) presented with severe pain, edema, and cyanosis. Due to its high morbidity and mortality, PCD is an indication for urgent catheter-directed therapy. A 48-year-old man with liposarcoma presented with worsening leg swelling despite anticoagulation. Contrast-enhanced axial CT image 3 months prior to presentation showed liposarcoma (arrows) encasing the external iliac vein (C). A right lower extremity venogram showed extensive clot in the right femoral vein (D). Pharmacomechanical thrombolysis was performed with a rheolytic infusion/aspiration catheter (arrow), seen as a subtraction artifact, with good clearance of thrombus (E). Stents were placed across the tumor-related obstruction to open the venous outflow (F). A final venogram demonstrated resolution of thrombus and patent outflow through the stents (G). The patient's symptoms improved within days of the procedure.

Deep Vein Thrombosis

Although most cases of deep vein thrombosis (DVT) can be managed in an outpatient setting, there are some cases that require immediate aggressive management. One example is phlegmasia cerulean dolens (PCD), which occurs when the venous system of an extremity becomes obstructed by an acute massive venous thrombosis (Figure 6). The most common risk factor for PCD is malignancy, which is present in about one-third of patients.[68] PCD most commonly involves the left leg.[69] Patients may first develop phlegmasia alba dolens with edema, severe pain, and blanching. Over time, fluid sequestration leads to bullae formation followed by cyanosis, venous gangrene, and circulatory collapse.[69,70] Without immediate treatment, loss of limb or death may ensue.[68]

PCD is the only instance of DVT where thrombolysis and/or thrombectomy is definitively indicated because of the urgent need to prevent limb loss or mortality.[71,72] Immediate consultation with vascular interventionalists is critical, and patients awaiting intervention should be treated with bed rest, leg elevation, fluid resuscitation, and intravenous unfractionated heparin.[68] Thrombolysis can be administered systemically or as catheter-directed therapy. Systemic thrombolysis is effective in resolving acute pain and cyanosis from PCD but is associated with high rates of postthrombotic syndrome manifested

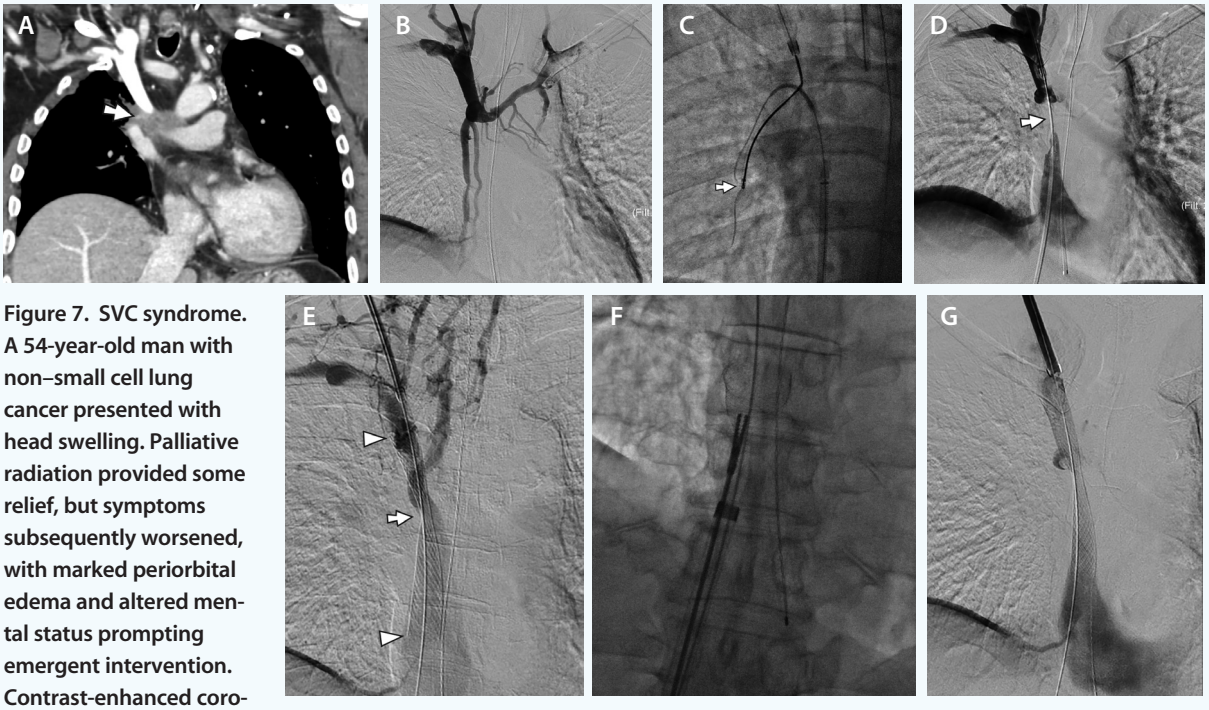

Figure 7. SVC syndrome. A 54-year-old man with non–small cell lung cancer presented with head swelling. Palliative radiation provided some relief, but symptoms subsequently worsened, with marked periorbital edema and altered mental status prompting emergent intervention. Contrast-enhanced coronal chest CT demonstrated a mediastinal soft tissue mass (arrow) obliterating the SVC (A). Initial access was obtained from the right internal jugular vein; venogram demonstrated obliteration of the SVC (B). The obstruction could not be crossed from above; therefore, additional access from the right common femoral vein was obtained. Both wires were advanced into the azygos vein, where a loop snare (arrow) was used to pull the jugular access wire down through and through (C). Venography performed by injecting contrast from both jugular and femoral access catheters demonstrated the long segment of SVC obstruction (arrow) (D). A stent was placed, with narrowing of its midportion indicative of the center of the obstruction (arrow). There is more stent below the obstruction than above, with the top and bottom of the stent indicated by arrowheads (E). The stent migrated ("watermelon seeded") into the ventricle. Forceps were used to recapture the stent, which was pulled through the sheath via the femoral access (F). A 16-mm uncovered metallic stent was then deployed with better centering on the obstruction (G). Swelling of the face and neck and mental status improved by the following day.

as massive leg edema and chronic pain.[69,73] Although catheter-directed therapy for DVT has not been shown to reduce the incidence of postthrombotic syndrome in general, direct injection of thrombolytic agents into the affected lower extremity vein results in more rapid clot lysis and allows for the use of smaller doses compared with systemic treatment, reducing the risk of bleeding.[72,74] Thrombolysis, whether systemic or catheter-directed, may also allow for more complete removal of clots, particularly from smaller vessels that may not be amenable to surgical or mechanical removal. The use of ultrasound-assisted catheter-directed thrombolysis has not been shown in prospective randomized clinical trials to improve outcomes compared with standard catheter-directed alteplase therapy.[75] As with catheter-directed treatment of PE, either single-session pharmacomechanical thrombectomy or catheter-directed thrombolysis may be employed; single-session treatment may reduce overall cost, as it obviates the need for prolonged intensive care level observation during tissue plasminogen activator infusion.[76]

Superior Vena Cava Syndrome

SVC syndrome can occur when blood flow through the SVC is obstructed due to external compression from tumor or lymph nodes, direct tumor invasion into the vessel, or venous thrombosis related to an indwelling device. Malignancy accounts for about 70% of the 15,000 cases of SVC syndrome in the United States each year, with most occurring secondary to non–small cell lung cancer, small cell lung cancer, and non-Hodgkin's lymphoma.[77,78]

The SVC drains the head, upper extremities, and upper torso and comprises one-third of the total venous return to the heart.[77] When the SVC is occluded, venous collaterals usually develop to return blood from the head and upper extremities to the heart, but this process may take weeks or be insufficient. Symptom severity depends on how rapidly the SVC becomes occluded.[78] When SVC

occlusion develops gradually, patients may present with face and neck swelling, dilated neck veins, cough, dyspnea, and upper extremity edema.[77] Symptoms are exacerbated by lying down or bending forward. If occlusion occurs more acutely, patients may present with altered mental status from elevated intracranial pressure or cerebral edema, respiratory distress from laryngeal edema or airway obstruction, or hypotension from impaired venous return.

SVC syndrome is an emergency when airway obstruction, neurologic compromise, or hemodynamic instability are present (Figure 7).[79] In such cases, intravascular stenting should be considered as it offers the most immediate relief (within 24-72 hours), coupled with high success rate and low complication rate.[80] Unlike chemotherapy and radiation, which require histologic diagnosis prior to treatment, no such information is required prior to stenting—an important consideration given that up to 60% of cases may occur in patients with undiagnosed malignancy.[77]

Although rare, SVC rupture resulting in pericardial effusion and tamponade is a catastrophic complication that can occur with stenting. In SVC syndrome, stenosis often occurs at the junction of the pericardium and the right atrium. Perforation can occur while crossing the stenosis or more commonly with balloon dilation of the stenosis.[81] Placing a stent without predilation and gradual balloon dilatation if needed after stent placement can minimize the risk of SVC rupture and hemopericardium. Self-expanding stents with a nominal diameter of at least 14 mm are used. Complications can arise with suboptimal stent choice and placement. If the stent is not well-centered on the obstruction, the force of the stenosis can cause the stent to migrate into the heart when it is opened—also known as "watermelon seeding."[82] In such cases, the stent may have to be captured in the heart using a snare.

CONCLUSION

Vascular oncologic emergencies are associated with significant morbidity and mortality. Preprocedural diagnostic workup including noninvasive imaging can help optimize minimally invasive procedural planning. Cancer patients are vulnerable to arterial hemorrhage and VTE and obstruction due to underlying oncologic morbidity. Both venous and arterial oncologic emergencies necessitate rapid diagnosis and often urgent percutaneous intervention. ■

1. Pereira J, Phan T. Management of bleeding in patients with advanced cancer. Oncologist. 2004;9:561-570. doi: 10.1634/theoncologist.9-5-561
2. Huynh TTT, Sheth RA. Management of arterial bleeding in critically ill cancer patients. In: Nates JL, Price KJ, eds. Oncologic Critical Care. Springer International Publishing; 2020:1223-1241.
3. Lu HJ, Chen KW, Chen MH, et al. Predisposing factors, management, and prognostic evaluation of acute carotid blowout syndrome. J Vasc Surg. 2013;58:1226-1235. doi: 10.1016/j.jvs.2013.04.056
4. Cartoni C, Niscola P, Breccia M, et al. Hemorrhagic complications in patients with advanced hematological malignancies followed at home: an Italian experience. Leuk Lymphoma. 2009;50:387-391. doi: 10.1080/10428190802714024
5. Harris DG, Noble SI. Management of terminal hemorrhage in patients with advanced cancer: a systematic literature review. J Pain Symptom Manage. 2009;38:913-927. doi: 10.1016/j.jpainsymman.2009.04.027
6. Loffroy R, Favelier S, Pottecher P, et al. Transcatheter arterial embolization for acute nonvariceal upper gastrointestinal bleeding: indications, techniques and outcomes. Diagn Interv Imaging. 2015;96:731-744. doi: 10.1016/j.diii.2015.05.002
7. Savides TJ, Jensen DM, Cohen J, et al. Severe upper gastrointestinal tumor bleeding: Endoscopic findings, treatment, and outcome. Endoscopy. 1996;28:244-248. doi: 10.1055/s-2007-1005436
8. Niekamp A, Sheth RA, Kuban J, et al. Palliative embolization for refractory bleeding. Semin Intervent Radiol. 2017;34:387-397. doi: 10.1055/s-0037-1608862
9. Thacker PG, Friese JL, Loe M, et al. Embolization of nonliver visceral tumors. Semin Intervent Radiol. 2009;26:262-269. doi: 10.1055/s-0029-1225667
10. Sengupta N, Cifu AS. Management of patients with acute lower gastrointestinal tract bleeding. JAMA. 2018;320:86-87. doi: 10.1001/jama.2018.5684
11. Barkun AN, Almadi M, Kuipers EJ, et al. Management of nonvariceal upper gastrointestinal bleeding: Guideline recommendations from the international consensus group. Ann Intern Med. 2019;171:805-822. doi: 10.7326/m19-1795
12. Barkun AN, Bardou M, Kuipers EJ, et al. International consensus recommendations on the management of patients with nonvariceal upper gastrointestinal bleeding. Ann Intern Med. 2010;152:101-113. doi: 10.7326/0003-4819-152-2-201001190-00009
13. Feuerstein JD, Ketwaroo G, Tewani SK, et al. Localizing acute lower gastrointestinal hemorrhage: CT angiography versus tagged rbc scintigraphy. AJR Am J Roentgenol. 2016;207:578-584. doi: 10.2214/ajr.15.15714
14. Speir EJ, Newsome JM, Bercu ZL, et al. Correlation of CT angiography and 99mtechnetium-labeled red blood cell scintigraphy to catheter angiography for lower gastrointestinal bleeding: a single-institution experience. J Vasc Interv Radiol. 2019;30:1725-1732.e1727. doi: 10.1016/j.jvir.2019.04.019
15. Hsu MJ, Dinh DC, Shah NA, et al. Time to conventional angiography in gastrointestinal bleeding: CT angiography compared to tagged rbc scan. Abdom Radiol. 2020;45:307-311. doi: 10.1007/s00261-019-02151-8
16. Jacovides CL, Nadolski G, Allen SR, et al. Arteriography for lower gastrointestinal hemorrhage: Role of preceding abdominal computed tomographic angiogram in diagnosis and localization. JAMA Surg. 2015;150:650-656. doi: 10.1001/jamasurg.2015.97
17. Barnert J, Messmann H. Diagnosis and management of lower gastrointestinal bleeding. Nat Rev Gastroenterol Hepatol. 2009;6:637-646. doi: 10.1038/nrgastro.2009.167
18. Busch OR, van Delden OM, Gouma DJ. Therapeutic options for endoscopic haemostatic failures: The place of the surgeon and radiologist in gastrointestinal tract bleeding. Best Pract Res Clin Gastroenterol. 2008;22:341-354. doi: 10.1016/j.bpg.2007.10.018
19. Zheng L, Shin JH, Han K, et al. Transcatheter arterial embolization for gastrointestinal bleeding secondary to gastrointestinal lymphoma. Cardiovasc Intervent Radiol. 2016;39:1564-1572. doi: 10.1007/s00270-016-1422-2
20. Meehan T, Stecker MS, Kalva SP, et al. Outcomes of transcatheter arterial embolization for acute hemorrhage originating from gastric adenocarcinoma. J Vasc Interv Radiol. 2014;25:847-851. doi: 10.1016/j.jvir.2014.02.005
21. Tandberg DJ, Smith TP, Suhocki PV, et al. Early outcomes of empiric embolization of tumor-related gastrointestinal hemorrhage in patients with advanced malignancy. J Vasc Interv Radiol. 2012;23:1445-1452. doi: 10.1016/j.jvir.2012.08.011
22. Lee SM, Jeong SY, Shin JH, et al. Transcatheter arterial embolization for gastrointestinal bleeding related to pancreatic adenocarcinoma: clinical efficacy and predictors of clinical outcome. Eur J Radiol. 2020;123:108787. doi: 10.1016/j.ejrad.2019.108787
23. Spanos K, Kouvelos G, Karathanos C, et al. Current status of endovascular treatment of aortoenteric fistula. Semin Vasc Surg. 2017;30:80-84. doi: 10.1053/j.semvascsurg.2017.10.004
24. Subiela JD, Balla A, Bollo J, et al. Endovascular management of ureteroarterial fistula: single institution experience and systematic literature review. Vasc Endovascular Surg. 2018;52:275-286. doi: 10.1177/1538574418761721
25. Fox JA, Krambeck A, McPhail EF, et al. Ureteroarterial fistula treatment with open surgery versus endovascular management: long-term outcomes. J Urol. 2011;185:945-950. doi: 10.1016/j.juro.2010.10.062
26. van den Bergh RC, Moll FL, de Vries JP, et al. Arterioureteral fistulas: unusual suspects-systematic review of 139 cases. Urology. 2009;74:251-255. doi: 10.1016/j.urology.2008.12.011
27. Darcy M. Uretro-arterial fistulas. Tech Vasc Interv Radiol. 2009;12:216-221. doi: 10.1053/j.tvir.2009.09.005
28. Krambeck AE, DiMarco DS, Gettman MT, et al. Ureteroiliac artery fistula: Diagnosis and treatment algorithm. Urology. 2005;66:990-994. doi: 10.1016/j.urology.2005.05.036
29. Turo R, Hadome E, Somov P, et al. Uretero-arterial fistula—not so rare? Curr Urol. 2018;12:54-56. doi: 10.1159/000489419
30. Malgor RD, Oderich GS, Andrews JC, et al. Evolution from open surgical to endovascular treatment of ureteral-iliac artery fistula. J Vasc Surg. 2012;55:1072-1080. doi: 10.1016/j.jvs.2011.11.043
31. Okada T, Yamaguchi M, Muradi A, et al. Long-term results of endovascular stent graft placement of ureteroarterial fistula. Cardiovasc Intervent Radiol. 2013;36:950-956. doi: 10.1007/s00270-012-0534-6
32. Lara-Hernández R, Riera Vázquez R, Benabarre Castany N, et al. Ureteroarterial fistulas: diagnosis, management, and clinical evolution. Ann Vasc Surg. 2017;44:459-465. doi: 10.1016/j.avsg.2017.05.001
33. Titomihelakis G, Feghali A, Nguyen T, et al. Endovascular management and the risk of late failure in the treatment of ureteroarterial fistulas. J Vasc Surg Cases Innov Tech. 2019;5:396-401. doi: 10.1016/j.jvscit.2019.06.010
34. Maran AG, Amin M, Wilson JA. Radical neck dissection: a 19-year experience. J Laryngol Otol. 1989;103:760-764. doi: 10.1017/s002221510011000x
35. McDonald MW, Moore MG, Johnstone PA. Risk of carotid blowout after reirradiation of the head and neck: a systematic review. Int J Radiat Oncol Biol Phys. 2012;82:1083-1089. doi: 10.1016/j.ijrobp.2010.08.029
36. Rühle A, Sprave T, Kalckreuth T, et al. The value of moderate dose escalation for re-irradiation of recurrent or second primary head-and-neck cancer. Radiat Oncol. 2020;15:81. doi: 10.1186/s13014-020-01531-5
37. Vargo JA, Ward MC, Caudell JJ, et al. A multi-institutional comparison of sbrt and imrt for definitive reirradiation of recurrent or second primary head and neck cancer. Int J Radiat Oncol Biol Phys. 2018;100:595-605. doi: 10.1016/j.ijrobp.2017.04.017

38. Powitzky R, Vasan N, Krempl G, et al. Carotid blowout in patients with head and neck cancer. Ann Otol Rhinol Laryngol. 2010;119:476-484. doi: 10.1177/000348941011900709
39. Shah H, Gemmete JJ, Chaudhary N, et al. Acute life-threatening hemorrhage in patients with head and neck cancer presenting with carotid blowout syndrome: follow-up results after initial hemostasis with covered-stent placement. AJNR Am J Neuroradiol. 2011;32:743-747. doi: 10.3174/ajnr.A2379
40. Wong DJY, Donaldson C, Lai LT, et al. Safety and effectiveness of endovascular embolization or stent-graft reconstruction for treatment of acute carotid blowout syndrome in patients with head and neck cancer: case series and systematic review of observational studies. Head Neck. 2018;40:846-854. doi: 10.1002/hed.25018
41. Liang NL, Guedes BD, Duvvuri U, et al. Outcomes of interventions for carotid blowout syndrome in patients with head and neck cancer. J Vasc Surg. 2016;63:1525-1530. doi: 10.1016/j.jvs.2015.12.047
42. Hang XF, Xu WS, Wang JX, et al. Risk of high-grade bleeding in patients with cancer treated with bevacizumab: a meta-analysis of randomized controlled trials. Eur J Clin Pharmacol. 2011;67:613-623. doi: 10.1007/s00228-010-0988-x
43. Wellner UF, Kulemann B, Lapshyn H, et al. Postpancreatectomy hemorrhage—incidence, treatment, and risk factors in over 1,000 pancreatic resections. J Gastrointest Surg. 2014;18:464-475. doi: 10.1007/s11605-013-2437-5
44. Wente MN, Veit JA, Bassi C, et al. Postpancreatectomy hemorrhage (PPH): an International Study Group of Pancreatic Surgery (ISGPS) definition. Surgery. 2007;142:20-25. doi: 10.1016/j.surg.2007.02.001
45. Stampfl U, Hackert T, Sommer CM, et al. Superselective embolization for the management of postpancreatectomy hemorrhage: a single-center experience in 25 patients. J Vasc Interv Radiol. 2012;23:504-510. doi: 10.1016/j.jvir.2011.12.013
46. Yekebas EF, Wolfram L, Cataldegirmen G, et al. Postpancreatectomy hemorrhage: diagnosis and treatment: an analysis in 1669 consecutive pancreatic resections. Ann Surg. 2007;246:269-280. doi: 10.1097/01.sla.0000262953.77735.db
47. Balthazar EJ. Complications of acute pancreatitis: clinical and CT evaluation. Radiol Clin North Am. 2002;40:1211-1227. doi: 10.1016/s0033-8389(02)00043-x
48. Correa-Gallego C, Brennan MF, D'Angelica MI, et al. Contemporary experience with postpancreatectomy hemorrhage: results of 1,122 patients resected between 2006 and 2011. J Am Coll Surg. 2012;215:616-621. doi: 10.1016/j.jamcollsurg.2012.07.010
49. Ching KC, Santos E, McCluskey KM, et al. Covered stents and coil embolization for treatment of postpancreatectomy arterial hemorrhage. J Vasc Interv Radiol. 2016;27:73-79. doi: 10.1016/j.jvir.2015.09.024
50. Cho SK, Kim SS, Do YS, et al. Ischemic liver injuries after hepatic artery embolization in patients with delayed postoperative hemorrhage following hepatobiliary pancreatic surgery. Acta Radiol. 2011;52:393-400. doi: 10.1258/ar.2011.100414
51. Hankins D, Chao S, Dolmatch BL, et al. Covered stents for late postoperative arterial hemorrhage after pancreaticoduodenectomy. J Vasc Interv Radiol. 2009;20:407-409. doi: 10.1016/j.jvir.2008.11.020
52. Lim SJ, Park KB, Hyun DH, et al. Stent graft placement for postsurgical hemorrhage from the hepatic artery: clinical outcome and CT findings. J Vasc Interv Radiol. 2014;25:1539-1548. doi: 10.1016/j.jvir.2014.06.023
53. Zakai NA, Walker RF, MacLehose RF, et al. Impact of anticoagulant choice on hospitalized bleeding risk when treating cancer-associated venous thromboembolism. J Thromb Haemost. 2018;16:2403-2412. doi: 10.1111/jth.14303
54. Sharafuddin MJ, Andresen KJ, Sun S, et al. Spontaneous extraperitoneal hemorrhage with hemodynamic collapse in patients undergoing anticoagulation: management with selective arterial embolization. J Vasc Interv Radiol. 2001;12:1231-1234. doi: 10.1016/s1051-0443(07)61686-8
55. Khorana AA, Francis CW, Culakova E, et al. Thromboembolism is a leading cause of death in cancer patients receiving outpatient chemotherapy. J Thromb Haemost. 2007;5:632-634. doi: 10.1111/j.1538-7836.2007.02374.x
56. Jaff MR, McMurtry MS, Archer SL, et al. Management of massive and submassive pulmonary embolism, iliofemoral deep vein thrombosis, and chronic thromboembolic pulmonary hypertension: a scientific statement from the American Heart Association. Circulation. 2011;123:1788-1830. doi: 10.1161/CIR.0b013e318214914f
57. Chatterjee S, Chakraborty A, Weinberg I, et al. Thrombolysis for pulmonary embolism and risk of all-cause mortality, major bleeding, and intracranial hemorrhage: a meta-analysis. JAMA. 2014;311:2414-2421. doi: 10.1001/jama.2014.5990
58. Kearon C, Akl EA, Ornelas J, et al. Antithrombotic therapy for VTE disease: Chest guideline and expert panel report. Chest. 2016;149:315-352. doi: 10.1016/j.chest.2015.11.026
59. Sista AK, Kuo WT, Schiebler M, et al. Stratification, imaging, and management of acute massive and submassive pulmonary embolism. Radiology. 2017;284:5-24. doi: 10.1148/radiol.2017151978
60. Fiumara K, Kucher N, Fanikos J, et al. Predictors of major hemorrhage following fibrinolysis for acute pulmonary embolism. Am J Cardiol. 2006;97:127-129. doi: 10.1016/j.amjcard.2005.07.117
61. Avgerinos ED, Chaer RA. Catheter-directed interventions for acute pulmonary embolism. J Vasc Surg. 2015;61:559-565. doi: 10.1016/j.jvs.2014.10.036
62. Tafur AJ, Shamoun FE, Patel SI, et al. Catheter-directed treatment of pulmonary embolism: a systematic review and meta-analysis of modern literature. Clin Appl Thromb Hemost. 2017;23:821-829. doi: 10.1177/1076029616661414
63. Tu T, Toma C, Tapson VF, et al. A prospective, single-arm, multicenter trial of catheter-directed mechanical thrombectomy for intermediate-risk acute pulmonary embolism: the FLARE study. JACC Cardiovasc Interv. 2019;12:859-869. doi: 10.1016/j.jcin.2018.12.022
64. Devcic Z, Kuo WT. Percutaneous pulmonary embolism thrombectomy and thrombolysis: technical tips and tricks. Semin Intervent Radiol. 2018;35:129-135. doi: 10.1055/s-0038-1642042
65. Bunc M, Steblovnik K, Zorman S, et al. Percutaneous mechanical thrombectomy in patients with high-risk pulmonary embolism and contraindications for thrombolytic therapy. Radiol Oncol. 2020;54:62-67. doi: 10.2478/raon-2020-0006
66. Avgerinos ED, Abou Ali A, Toma C, et al. Catheter-directed thrombolysis versus suction thrombectomy in the management of acute pulmonary embolism. J Vasc Surg Venous Lymphat Disord. 2019;7:623-628. doi: 10.1016/j.jvsv.2018.10.025
67. Jiménez D, Aujesky D, Moores L, et al. Simplification of the pulmonary embolism severity index for prognostication in patients with acute symptomatic pulmonary embolism. Arch Intern Med. 2010;170:1383-1389. doi: 10.1001/archinternmed.2010.199
68. Chinsakchai K, Ten Duis K, Moll FL, et al. Trends in management of phlegmasia cerulea dolens. Vasc Endovascular Surg. 2011;45:5-14. doi: 10.1177/1538574410388309
69. Oguzkurt L, Ozkan U, Demirturk OS, et al. Endovascular treatment of phlegmasia cerulea dolens with impending venous gangrene: manual aspiration thrombectomy as the first-line thrombus removal method. Cardiovasc Intervent Radiol. 2011;34:1214-1221. doi: 10.1007/s00270-010-0042-5
70. Stallworth JM, Bradham GB, Kletke RR, et al. Phlegmasia cerulea dolens: a 10-year review. Ann Surg. 1965;161:802-811. doi: 10.1097/00000658-196505000-00018
71. Thukral S, Vedantham S. Catheter-based therapies and other management strategies for deep vein thrombosis and post-thrombotic syndrome. J Clin Med. 2020;9:1439. doi: 10.3390/jcm9051439
72. Vedantham S, Piazza G, Sista AK, et al. Guidance for the use of thrombolytic therapy for the treatment of venous thromboembolism. J Thromb Thrombolysis. 2016;41:68-80. doi: 10.1007/s11239-015-1318-z
73. Tardy B, Moulin N, Mismetti P, et al. Intravenous thrombolytic therapy in patients with phlegmasia caerulea dolens. Haematologica. 2006;91:281-282.
74. Vedantham S, Goldhaber SZ, Julian JA, et al. Pharmacomechanical catheter-directed thrombolysis for deep-vein thrombosis. N Engl J Med. 2017;377:2240-2252. doi: 10.1056/NEJMoa1615066
75. Engelberger RP, Stuck A, Spirk D, et al. Ultrasound-assisted versus conventional catheter-directed thrombolysis for acute iliofemoral deep vein thrombosis: 1-year follow-up data of a randomized-controlled trial. J Thromb Haemost. 2017;15:1351-1360. doi: 10.1111/jth.13709
76. Lin PH, Zhou W, Dardik A, et al. Catheter-direct thrombolysis versus pharmacomechanical thrombectomy for treatment of symptomatic lower extremity deep venous thrombosis. Am J Surg. 2006;192:782-788. doi: 10.1016/j.amjsurg.2006.08.045
77. Friedman T, Quencer KB, Kishore SA, et al. Malignant venous obstruction: superior vena cava syndrome and beyond. Semin Intervent Radiol. 2017;34:398-408. doi: 10.1055/s-0037-1608863
78. Wilson LD, Detterbeck FC, Yahalom J. Clinical practice. Superior vena cava syndrome with malignant causes. N Engl J Med. 2007;356:1862-1869. doi: 10.1056/NEJMcp067190
79. Yu JB, Wilson LD, Detterbeck FC. Superior vena cava syndrome--a proposed classification system and algorithm for management. J Thorac Oncol. 2008;3:811-814. doi: 10.1097/JTO.0b013e3181804791
80. Lanciego C, Pangua C, Chacón JI, et al. Endovascular stenting as the first step in the overall management of malignant superior vena cava syndrome. AJR Am J Roentgenol. 2009;193:549-558. doi: 10.2214/ajr.08.1904
81. Stevens DC, Butty S, Johnson MS. Superior vena cava rupture and cardiac tamponade complicating the endovascular treatment of malignant superior vena cava syndrome: a case report and literature review. Semin Intervent Radiol. 2015;32:439-444. doi: 10.1055/s-0035-1564795
82. Funaki B. Superior vena cava syndrome. Semin Intervent Radiol. 2006;23:361-365. doi: 10.1055/s-2006-957027

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