INTRODUCTION

The American Cancer Society estimates 1,762,450 new cases of cancer and 606,880 cancer-related deaths in 2019. The overall cancer death rate dropped by 27% between 1991 and 2016, representing approximately 2,629,200 fewer cancer deaths.1 As survivors continue to live longer, they have a higher risk for developing cardiovascular disease. It is therefore important to establish their overall cardiovascular risk, recognizing that prior exposure to cancer therapy, such as radiation and anthracycline-based chemotherapy, may further impact their cardiovascular health. Understanding the interplay between the development of cancer, the complex treatments to combat the disease, and the baseline cardiovascular risk of any given patient is critical to optimize prevention or early detection strategies and improve overall outcomes in these vulnerable patients.

The vascular toxicity associated with all chemo- and radiotherapy treatments can be viewed in myriad ways; for this review, however, they will be summarized into thrombosis, accelerated atherosclerosis, and vasospasm.2 Since the landscape of treatment-related vascular toxicity is too broad to concisely cover in a single review, we will focus on a variety of recent cases to highlight treatment challenges and possible therapeutic options and, where appropriate, introduce the latest research findings that inform clinical decision making.

CANCER-ASSOCIATED VENOUS THROMBOEMBOLISM

It is well established that patients with cancer have a higher incidence of venous thromboembolism (VTE) than the general population, and cancer-associated VTE is a marker of poor prognosis.3 Management of cancer-associated VTE remains a substantial challenge mainly due to the high risk of recurrent thrombosis coupled with an increased risk of bleeding complications. These complicating vascular events usually result in at least temporary interruption of cancer treatment and have a negative effect on a patient’s prognosis and survival.

Establishing the best anticoagulation treatment is of utmost importance. Guidelines still recommend low-molecular-weight heparin (LMWH) for at least 6 months,4 which can be somewhat burdensome for patients because it requires daily subcutaneous injections. In addition, the most appropriate duration of treatment has not been established. Several trials have evaluated the safety and efficacy of direct oral anticoagulants (DOACs) for thromboprophylaxis and/or treatment of VTE in patients with cancer.5-9 These trials have shown that DOACs are noninferior to LMWH with regard to the rate of recurrent thrombosis. Nonetheless, DOACs are associated with a higher risk of major and clinically relevant bleeding, especially among patients with esophageal and gastric cancers. Drug-drug interactions also play a major role when deciding to use DOACs and establishing a patient’s bleeding risk. This is particularly important in patients receiving active chemotherapy and/or treatment with potent P-glycoprotein and/or CYP3A4 inhibitors or inducers (eg, ketoconazole, diltiazem, verapamil, tacrolimus, cyclosporine).

Recent trials have investigated a personalized approach to thromboprophylaxis and treatment of venous thrombosis. Khorana et al. developed the Khorana score, a model for predicting chemotherapy-associated VTE using baseline clinical and laboratory variables. The Khorana score is a validated tool that considers cancer type, pre-chemotherapy platelet count, hemoglobin level or the use of red blood cell growth factors, pre-chemotherapy leukocyte count, and body...
mass index. Although this score can help guide the decision for thromboprophylaxis based on a patient’s VTE risk, it does not include other potential risk factors for VTE. This is especially important since there is a growing number of older cancer survivors who are at risk for age-related comorbidities, specifically atrial fibrillation. In a meta-analysis by Biondi-Zoccai et al., DOACs were recommended over warfarin in patients with nonvalvular atrial fibrillation. O’Neal et al. demonstrated that early cardiology involvement is associated with increased anticoagulant prescription fills and favorable outcomes in patients with atrial fibrillation.

Patients who experience recurrent VTE despite adequate anticoagulation and those who have a contraindication to anticoagulation pose a significant challenge. There is insufficient evidence addressing the use of inferior vena cava (IVC) filters in these patients. Placement of nonretrievable filters may predispose to IVC thrombosis, especially in patients with cancers such as renal cell carcinoma. It is unclear if this practice translates into a survival benefit in a group of patients who probably have a worse prognosis. Thus, there is need for a prospective study addressing this issue.

In the case of retrievable filters, many are not retrieved due to poor clinical follow-up or significant technical challenges during retrieval. Retrieval rates are reported to be as low as 8.5%. Prolonged filter implantation is associated with numerous complications including device fracture, migration, organ penetration by device components, and elevated risk of deep vein thrombosis (DVT).

**Case 1**

A 40-year-old woman diagnosed with bilateral ovarian cancer (FIGO Stage IV) and treated with carboplatin, paclitaxel, and bevacizumab developed acute DVT in both lower extremities and subsequently bilateral pulmonary embolisms extending to multiple segments. Since there was evidence of right ventricular strain, she received tissue plasminogen activator thrombolysis. Despite treatment with full anticoagulation (enoxaparin), follow-up chest computed tomography (CT) scan with intravenous contrast demonstrated interval increase in the overall clot burden. An IVC filter was placed in addition to continuing full anticoagulation. Since the patient was receiving chemotherapy, she had a right-sided port in place. A thrombus in the right atrium was noted on a screening transthoracic echocardiogram, which was later confirmed by transesophageal echocardiogram (Figure 1). About 2 months later, a follow-up chest and abdomen/pelvis CT scan with intravenous contrast revealed thrombosis of the IVC filter (Figure 2). The patient had evidence of thrombosis in the superior vena cava (SVC) and IVC in spite of full anticoagulation.

![Figure 1](image1.png)

**Figure 1.**

Thrombus in a patient with bilateral ovarian cancer: (A) Bicaval view on transesophageal echocardiogram (TEE) showing an echodensity determined to be a thrombus (yellow arrow). (B) Color flow Doppler on TEE demonstrating partial obstruction of flow around the thrombus.
Case 2

A 27-year-old man with synovial sarcoma in his left thigh was diagnosed with bilateral pulmonary embolisms and a left lower extremity DVT involving the common femoral vein (Figure 3). The ultrasound of the left lower extremity incidentally revealed a large, vascularized mass in the mid-thigh measuring 10.2 × 10.6 cm. The patient was treated with apixaban, and a 2-month follow-up chest CT scan demonstrated a reduction in embolic burden. This case illustrates the hypercoagulable state of patients with cancer and the variability in the choice of anticoagulant when treating VTE. It further supports the fact that certain types of cancer carry a higher risk for thrombosis than others, regardless of a patient’s age and other comorbidities.

SUPERIOR VENA CAVA SYNDROME IN CANCER PATIENTS

Malignancy-associated SVC syndrome in cancer patients can present in different ways: (1) direct invasion of the tumor into the SVC; (2) external compression of the SVC by an adjacent tumor, enlarged lymph node(s), or other mediastinal structures; and (3) as a result of indwelling venous devices such as central venous catheters. In some cases, SVC syndrome may be due to more than one pathologic process and may develop at a faster
rate in cancer patients due to rapid tumor growth and lack of adequate venous collateral circulation. Common symptoms include chest discomfort, shortness of breath, facial and neck swelling, and head fullness. Management of malignancy-associated SVC syndrome is complex and should consider the severity of symptoms and the patient’s underlying condition and overall prognosis.

**Case 3**

A 39-year-old woman diagnosed with pericardial mesothelioma developed SVC syndrome within a year after her diagnosis. The patient had been treated with palliative mediastinal radiation (total of 30 Gy) and different chemotherapy and immunotherapy regimens that included cisplatin, etoposide, pemetrexed, vinorelbine, and pembrolizumab. Her symptoms consisted of headaches, head fullness especially when bending forward, and facial and upper extremity swelling. Chest, abdomen, and pelvic CT scan with intravenous contrast demonstrated narrowing of the distal SVC as it enters the right atrium (Figure 4). There was evidence of high-grade SVC obstruction with an initial gradient of 22 mm Hg, which was reduced to 1 mm Hg after SVC stenting. Her symptoms significantly improved after this intervention.

Although there are no evidence-based guidelines for management of SVC syndrome, different societies and committees have provided general recommendations. It is now recognized that endovenous recanalization with

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**Figure 3.**

Chest computed tomography demonstrates multiple bilateral pulmonary emboli (red arrows) in a 27-year-old man with synovial sarcoma in the left thigh.

**Figure 4.**

Computed tomography (CT) of patient diagnosed with pericardial mesothelioma who developed superior vena cava (SVC) syndrome. (A) Chest CT scan with intravenous contrast demonstrates narrowing of the SVC as it enters the right atrium (yellow arrow). (B) Chest CT scan with intravenous contrast demonstrates relief of SVC obstruction with stenting (yellow arrow).
SVC stent placement is an effective strategy and should be pursued in patients presenting with severe and/or life-threatening symptoms. In patients with thrombus-associated SVC obstruction, endovenous thrombolysis is an alternative that can help determine the location and extent of any venous stenosis that could be amenable to stenting. Glucocorticoids can be considered in cases caused by steroid-responsive malignancies, such as lymphoma or thymoma, but their use should be avoided in the absence of a histologic diagnosis. Radiation therapy is an effective intervention in patients with radiosensitive malignancies and stable symptoms, with improvement generally seen within 72 hours.

ANTICOAGULATION IN PATIENTS WITH BRAIN TUMORS

Intracranial hemorrhage (ICH) is common in patients with brain tumors; therefore, understanding the risks and benefits of anticoagulation in these patients is of utmost importance. The annual rate of ICH is reported to be between 0.3% and 0.6% in patients taking warfarin and between 0.1% and 0.2% in those taking DOACs. Recent trials have shown that DOACs are noninferior to warfarin regarding stroke prevention in nonvalvular atrial fibrillation and are associated with less bleeding complications, specifically less ICH. However, in cancer patients, DOACs have been shown to increase the risks of major and clinically relevant bleeding. Carney et al. recently conducted a retrospective study comparing the rates of ICH with DOACs (n = 42) and LMWH (n = 131) in patients with primary brain tumors and brain metastases. In the primary brain tumor group (n = 67), the cumulative incidence of any ICH was 0% in patients receiving DOACs versus 36.8% in those treated with LMWH, with a major ICH incidence of 18.2% in the latter group. In the group with brain metastases (n = 105), the cumulative incidence of any ICH was 27.8% in patients receiving DOACs versus 52.9% in those receiving LMWH, and 11.1% versus 17.8% for major ICH, respectively. Based on these findings, the authors concluded that DOACs appear to be safe with respect to ICH risk in patients with primary and metastatic brain tumors. A randomized controlled trial would be needed to validate these findings.

Understanding the role of oral anticoagulants in ICH is important since the majority of these events are due to diseases affecting the large or small cerebral vessels (e.g., aneurysms, arteriovenous malformations), resulting in spontaneous bleeding. Oral anticoagulants are not believed to cause ICH but, rather, to contribute to hematoma formation and expansion. Further research is warranted in this area, especially with the development of new reversal agents for DOACs.

Case 4

A 63-year-old man diagnosed with adenocarcinoma of the lung with brain metastases developed a left ventricular (LV) apical thrombus after experiencing an anterior myocardial infarction and LV systolic dysfunction with ejection fraction of 35% (Figure 5). The diagnosis of LV apical thrombus was an incidental finding during a follow-up chest CT scan. The patient ultimately

Figure 5.
Patient diagnosed with adenocarcinoma of the lung with brain metastases. (A) Transthoracic echocardiogram with contrast shows evidence of left ventricular (LV) apical thrombus (yellow arrow). (B) Transthoracic echocardiogram without contrast demonstrating LV apical aneurysm (yellow arrow).
underwent left heart catheterization and required percutaneous coronary intervention in the left anterior descending artery with a total of four drug-eluting stents. He was treated with aspirin 81 mg daily and clopidogrel 75 mg daily in addition to full anticoagulation with enoxaparin (1 mg/kg every 12 hours). A follow-up transthoracic echocardiogram after approximately 9 months of treatment showed no evidence of LV apical thrombus, and anticoagulation was discontinued. The patient was continued on dual antiplatelet therapy and had no major bleeding complications when receiving triple therapy (aspirin, clopidogrel, and enoxaparin). This case also raises the question as to whether he should continue on dual antiplatelet therapy or be switched to aspirin 81 mg daily plus rivaroxaban 2.5 mg twice daily, recognizing that the latter is neither the prophylactic nor treatment dose for VTE, and the mechanism of his LV thrombus formation is due to his anterior myocardial infarction. A randomized controlled clinical trial would be warranted to answer this question.

CARDIOVASCULAR EFFECTS OF GRAFT VERSUS HOST DISEASE

Survivors of hematopoietic stem cell transplantation (HSCT) have a higher risk of developing multiple cardiovascular risk factors and cardiovascular disease compared to the general population. Armenian et al. demonstrated that HSCT patients have a 10-year cumulative incidence of hypertension, diabetes, dyslipidemia, and multiple (≥ 2) cardiovascular risk factors of 37.7%, 18.1%, 46.7%, and 31.4%, respectively. This was primarily driven by a higher prevalence of cardiovascular risk factors in allogeneic HSCT recipients. Older age and obesity at the time of HSCT were associated with a higher risk of having these conditions. In multivariate analysis, allogeneic HSCT recipients with a history of acute graft versus host disease (GVHD) II-IV had an even greater risk for hypertension (P < .01), diabetes (P < .01), and dyslipidemia (P < .01). Conditioning with total body irradiation was associated with a higher risk of diabetes and dyslipidemia but not hypertension. Patients with pre-HSCT exposure to anthracycline therapy and/or chest radiation had a much higher incidence of cardiovascular disease.21 Tichelli et al. demonstrated that allogeneic HSCT (RR 14.5, P = .003) and at least two of four cardiovascular risk factors (hypertension, dyslipidemia, diabetes, obesity) (RR 12.4, P = .02) were associated with a higher incidence of arterial vascular events.22

HSCT-related factors can also contribute to a patient’s cardiovascular risk. Treatments such as total body irradiation can damage endothelial cells, which have also been shown to be a target of GVHD.23 Chronic use of immunosuppressive drugs, specific calcineurin inhibitors and steroids, and uncontrolled diabetes are commonly associated with a higher risk of dyslipidemia. Hypogonadism, ovulatory failure, and growth hormone deficiency are common complications in HSCT recipients.24

The development of coronary artery disease post-HSCT is a complex process and most likely results from the combination of traditional risk factors and immune-mediated mechanisms. Case reports have shown that coronary artery disease in HSCT patients appears to be due to intimal proliferation, similar to cardiac allograft vasculopathy.25 However, this can be confounded by pre-HSCT exposure to anthracycline therapy, mediastinal radiation, and/or total body irradiation.

Case 5

A 39-year-old woman diagnosed with Hodgkin lymphoma in 2001 was treated with mantle radiation as well as ABVD (adriamycin, bleomycin, vinblastine, dacarbazine) for 6 cycles and ESHAP (etoposide, methylprednisolone, cytarabine, and cisplatin) for 2 cycles and underwent autologous HSCT in 2004. Due to recurrent disease, she required allogeneic HSCT in 2006. Her case has been complicated by significant chronic GVHD involving the skin, muscles, and gastrointestinal system that requires monthly photopheresis and treatment with baricitinib and ruxolitinib. In addition, she experienced a DVT and pulmonary embolism and is on chronic anticoagulation with rivaroxaban 20 mg daily. She was referred to our clinic for cardiotoxicity monitoring and, more recently, for worsening dyspnea on exertion. Because of evidence of coronary artery calcification on routine chest CT scan, the patient was referred for a left heart catheterization that revealed severe obstructive disease involving the proximal left anterior descending artery. She required percutaneous coronary intervention with a drug-eluting stent, which improved her symptoms (Figure 6). This case illustrates the cardiac effects of radiation therapy and chronic GVHD in a young patient who was initially treated at the age of 21 years. Routine surveillance for premature coronary artery disease is highly recommended in this patient population.

ARTERIAL THROMBOSIS IN CANCER PATIENTS

Cardiovascular disease and cancer share similar risk factors, especially in older patients. For this reason, cancer patients are at risk for common cardiovascular problems, specifically coronary artery disease and peripheral arterial disease (PAD). This risk can be heightened when patients are exposed to specific cancer therapies, such as certain tyrosine kinase inhibitors.26 It is recommended that these patients undergo careful cardiovascular risk assessment and management before, during, and sometimes after therapy with agents known to increase the risk for short- and long-term vascular events.

The COMPASS (Cardiovascular Outcomes for People Using Anticoagulation Strategies) trial evaluated the effectiveness of rivaroxaban (2.5 mg twice daily) plus aspirin (100 mg daily) versus aspirin alone and rivaroxaban alone (5 mg twice daily)
for secondary cardiovascular prevention in participants with stable coronary artery disease and/or PAD. The rate of the primary outcome—a composite of cardiovascular death, stroke, or myocardial infarction—was lower by 24% with rivaroxaban plus aspirin compared to either aspirin or rivaroxaban alone. However, the rate of major bleeding was higher by 70% in the rivaroxaban and aspirin group compared to the aspirin-alone group.

Moreover, a recent analysis of this trial showed a dismal prognosis for patients with lower extremity PAD after a major adverse limb event (MALE). This was associated with a 3-fold increase in death, a 200-fold increase in the risk of subsequent amputations, and a 7-fold increase in subsequent hospitalizations. For these patients, the combination of rivaroxaban and aspirin lowered the incidence of MALE by 43% ($P = .01$), total vascular amputations by 58% ($P = .01$), peripheral vascular interventions by 24% ($P = .03$), and all peripheral vascular outcomes by 24% ($P = .02$). These findings support the combined use of rivaroxaban plus aspirin in patients with atherosclerotic vascular disease to improve their prognosis.

It is difficult to extrapolate these data to cancer patients due to their higher risk for thrombosis and bleeding and their underrepresentation in this trial. Rivaroxaban, in particular, has different doses depending on the indication, and it can sometimes be challenging to determine the most appropriate dose, such as in patients with a history of DVT (dosed to reduce the risk of recurrence) and atherosclerotic vascular disease (dosed for secondary cardiovascular prevention).

Case 6

A 65-year-old man with renal cell carcinoma diagnosed in 2014 required right radical nephrectomy followed by pazopanib. The latter had to be dose reduced and eventually discontinued due to worsening LV systolic function. The patient was then transitioned to axitinib. He was seen in clinic due to progressive dyspnea on exertion and persistent LV systolic dysfunction. Given his history of coronary artery disease requiring percutaneous coronary intervention in 2012, he was referred for left heart catheterization that revealed significant disease involving the ramus and left circumflex artery. He underwent drug-eluting stent placement and was started on dual antiplatelet therapy with aspirin 81 mg daily and clopidogrel 75 mg daily. Less than a week later, the patient presented with acute flank pain and was diagnosed with acute thrombosis of the left renal artery (Figure 7 A). In addition, a CT scan of the chest, abdomen, and pelvis revealed a thrombus in the left atrial appendage, and a venous ultrasound of the lower extremities showed evidence of acute DVT involving the left common femoral vein. The patient underwent suction thrombectomy of the left renal artery, and axitinib was discontinued (Figure 7 B). During the same hospitalization, the patient developed acute right lower extremity...
ischemia and required endarterectomy with patch angioplasty of the right common femoral artery as well as embolectomy of the right femoral, popliteal, and peroneal arteries. Although this patient had evidence of significant peripheral vascular disease at baseline, this was probably worsened by the use of pazopanib and axitinib, both of which are known to increase the risk of arterial and venous thromboembolism.

DISCUSSION

These cases highlight the complex care of patients with cancer and the dearth of evidence available to guide clinical decision making. An interdisciplinary collaboration is required in most cases to deliver individualized care to these patients due to the heterogeneity of their conditions and responses to treatment. A stepwise approach is recommended by establishing a patient’s baseline cardiovascular risk, estimating the vasotoxicity potential of the planned treatment, assessing the need for thromboprophylaxis, and understanding possible pharmacologic interactions that could result in toxicity and adverse events (Table 1). Early involvement of a cardio-oncologist will help optimize any prior therapy a patient may have been receiving for a pre-existing cardiovascular condition.

Patients with pre-existing cardiovascular risk factors may have a higher risk for vasotoxicity during and after treatment. This depends on the type of cancer therapy and any degree of risk modification achieved before initiating treatment. These patients should be considered at high risk for major cardiac events, and more stringent treatment goals should be pursued (eg, achieving low-density lipoprotein level < 70 mg/dL). This is in addition to recommending physical activity and a heart-healthy diet as dictated by the patient’s nutritional needs. It is unclear if those with a prior history of coronary artery disease should undergo risk stratification for ischemia before initiating treatment, even when asymptomatic. This decision will depend on the risk for thrombosis and ischemia based on the type of cancer and treatment plan.

The use of validated screening tools, such as the Khorana score, can assist clinicians in establishing a patient’s risk for VTE and the need to consider thromboprophylaxis. Recent trials have shown the efficacy and safety of DOACs in cancer patients and provided insight into the risk factors for clinically relevant bleeding. Nonetheless, the use of DOACs remains a challenge due to labile renal function in these patients, which requires dose adjustment, pharmacologic interaction with cancer-related (eg, ibrutinib) and noncancer-related therapies (eg, anticonvulsants, antifungals), and different dosing recommendations depending on the indication. Historically, intracranial bleeding has been a concern in patients with brain tumors or brain metastases who require anticoagulation. In a
retrospective cohort study, Carney et al. demonstrated potential safety using DOACs in these patients compared to LMWH. However, a randomized controlled trial would be needed to validate this finding.

Patients with GVHD may have a higher risk for coronary artery disease resulting from an immune process similar to that seen in transplant vasculopathy. Previous studies have also shown that patients with allogeneic HSCT have a higher risk for developing hypertension, dyslipidemia, and diabetes compared to autologous HSCT, and the risk for major cardiac events is much higher in those with pre-HSCT exposure to chest radiation or anthracyclines, as shown in the case presented here. Thus, it is important to incorporate preventative strategies such as antplatelet and statin therapy to minimize the risk of developing these conditions.

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Table 1. Evaluation and management of vascular complications in patients with cancer.
Endovascular thrombolysis is a feasible treatment option for selected patients with cancer. In a retrospective study including 178 patients with and without cancer, Kim et al. demonstrated that catheter-directed thrombolysis is a safe and effective treatment for patients with cancer and acute symptomatic DVT. The limited use of this therapy is due to a general concern for increased risk of bleeding in this population. However, in this study, only 4.9% of patients with cancer had a major hemorrhage, and none had an intracranial hemorrhage. This is reassuring when compared to the 11% incidence of major hemorrhage reported in a multicenter registry. Identifying patients with a higher risk of bleeding remains a challenge, since this is a multifactorial complication beyond the type of cancer or treatment history.

Data on the effects of cancer therapy in patients with concomitant aortic aneurysm is limited, with conflicting evidence suggesting a potential role in aneurysm enlargement and rupture. In a retrospective observational study, Leopardi et al. reported 19 consecutive cases of patients affected by abdominal aortic aneurysm and malignancy, predominantly from colorectal and urinary tract cancers. Four patients who received cancer therapy experienced a mean sac growth of 2.9 cm in 6 months, with two of them requiring urgent treatment for aortic rupture. No conclusions could be made regarding the effects of cancer therapy on the risk of aneurysm enlargement and rupture due to small sample size, although the authors suggested a potential role in this process. Moreover, Palm et al. had previously reported the case of a patient with pancreatic carcinoma who received combination therapy with gemcitabine, cisplatin, and prednisone/dexamethasone and experienced acute aneurysmal enlargement and subsequent rupture. As with the other study, a definitive association between cancer treatment and aneurysmal dilation and rupture could not be made. More recently, Martin et al. conducted a retrospective review of 91 cases between January 2000 and July 2011, and chemotherapy did not appear to increase aneurysm growth compared with patients who did not undergo treatment for malignancy. Thus, controversy persists around this topic, and further research is required given the array of therapeutic options currently available for aortic aneurysm.

CONCLUSION

Vascular complications are common in patients with cancer as a result of treatment, pre-existing cardiovascular conditions, or the cancer itself. It is thus important to recognize any risk factors and pursue aggressive interventions considering the patient’s overall prognosis and goals of care. Developing screening tools for risk stratification and understanding the effects of cancer treatment on the vascular system represent compelling areas of research within the field of cardio-oncology.

KEY POINTS

- Vascular complications in patients with cancer are common and may result from the complex treatments, baseline cardiovascular risk factors, and/or the cancer itself.
- Management of anticoagulation is one of the most significant challenges due to the risk for recurrent thrombosis, the increased rate of bleeding, and the potential pharmacologic interactions between anticoagulants and cancer therapies.
- An interdisciplinary approach to the care of cardio-oncology patients is of utmost importance and should be pursued whenever possible.

Conflict of Interest Disclosure:
Dr. Lenihan is a formal advisor for Roche, Pfizer, Bristol Myers Squibb, and Acorda Therapeutics and conducts research on behalf of Myocardial Solutions.

Keywords:
vascular toxicity, superior vena cava syndrome, arterial thrombosis, venous thromboembolism, pulmonary embolism, cardio-oncology

REFERENCES


