Thoracic vascular disease in oncologic patients

D. Varona Porres*, J. Andreu Soriano, E. Pallisa Núñez, O. Persiva Morenza, A. Roque Pérez

Departamento de Radiodiagnóstico, Hospital Vall d’Hebron, Barcelona, Spain

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Abstract Patients with oncologic disease require frequent imaging tests (predominantly computed tomography) for follow-up. These patients may have thoracic vascular disease that can influence the diagnosis, treatment, and prognosis of their cancer. Primary vascular tumors can involve the thoracic vessels, like the pulmonary arteries (pulmonary artery sarcoma), and the neoplastic disease can extend locally (lung tumor) or remotely to the thoracic vessels (pulmonary tumor embolism and pulmonary tumor thrombotic microangiopathy). Oncologic treatment results in multiple complications that involve the thoracic vessels and can even compromise the patient’s life in certain cases. CT, and especially multislice CT, makes it possible to evaluate neoplastic disease and associated thoracic vascular disease in oncologic patients.

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KEYWORDS
Thoracic diseases; Vascular diseases; Clinical oncology; Multidetector CT

Palabras clave - Enfermedades torácicas; Enfermedades vasculares; Oncología clínica; TC multicorte

Patología vascular torácica en pacientes oncológicos

Resumen La patología oncológica requiere frecuentes controles mediante pruebas de imagen, de forma predominante con tomografía computarizada (TC). En estos pacientes podemos encontrar patología vascular torácica que puede influir en el diagnóstico, el tratamiento y el pronóstico de su enfermedad neoplásica. Los tumores primarios vasculares pueden afectar a los vasos torácicos, como las arterias pulmonares (sarcoma de arteria pulmonar), y la enfermedad neoplásica se puede extender localmente (neoplasia pulmonar) o a distancia hacia los vasos torácicos (embolia pulmonar tumoral y microangiopatía trombótica tumoral pulmonar). El tratamiento oncológico es la causa de múltiples complicaciones sobre los vasos torácicos que en determinados casos llegan a comprometer la vida del paciente. La TC, especialmente con técnica multicorte, permite la evaluación de la enfermedad neoplásica y la patología vascular torácica asociada en el paciente oncológico.

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Introduction

At present, oncological radiology represents much of the activity of the departments of radiodiagnosis. It is thus important to have an extensive knowledge of oncologic pathology as well as of the possible complications that may arise from the treatment, which can often be aggressive and potentially toxic.

Oncologic disease can involve the thoracic vessels, either in the form of primary tumor or secondary to thoracic neoplasms, especially lung tumor. In addition, the treatment of oncology patients is associated with vascular complications involving the thoracic region that compromise the management and even the lives of these patients.

Neoplastic disease

Primary neoplasms

Primary pulmonary artery (PA) sarcomas (Fig. 1) are often misdiagnosed as pulmonary thromboembolic disease.

They originate from mesenchymal cells of the intima of the PA1 and may appear as polypoid intraluminal or sessile masses. In approximately half of the cases, the tumors remain intraluminal; in the other half, they spread transmurally into the adjacent lung, bronchial wall, or lymph nodes.2 Pulmonary nodules may also be seen.2

The most common symptoms are dyspnea (72%), chest pain (45%), cough (42%) and hemoptysis (24%).3 A chronic clinical course without acute dyspnea suggests PA sarcoma. The age of presentation is 13–81 years (mean 49.3 years) with no gender prevalence.3

Chest radiography most often demonstrates a unilateral hilar mass in an arterial distribution projecting into the lung. If there is parenchymal spread, the lesion can mimic lung cancer.

Computed tomography (CT) findings demonstrate a heterogeneous mass distending the PA with extraluminal extension, pulmonary condensation or subpleural nodules, pleural effusion or cardiomegaly. It may be misdiagnosed as pulmonary embolism. In 86% of cases, CT demonstrates a filling defect occupying the entire lumen of the main or proximal PA with distension of some portion of the PA involved.1 Extrapulmonary extension is an additional specific finding. The presence of a filling defect occupying the entire lumen of the main or proximal arteries may be the initial finding on CT.

Unlike pulmonary embolism, PET/CT scan may show FDG uptake.1

The prognosis is poor (five-year survival rate is 6%)4 and the mean survival time after onset of symptoms is approximately 12 months.3

Secondary neoplasms

Extrinsic compression or local invasion of the thoracic vessels

Superior vena cava syndrome (SVCS) is the result of the obstruction of the superior vena cava (SVC) or its major tributaries by intraluminal occlusion, extrinsic compression and/or invasion by malignant or benign disease.5 The most common causes are malignancies5; however, thrombosis related to catheters or pacemakers has increased in recent years. There might be direct extension of the tumor or adjacent lymph nodes or extrinsic compression. Vascular invasion may lead to thrombophlebitis and complete occlusion.5

Figure 1  (A) 68-Year-old man presents with a 3-month history of dyspnea with exertion that has progressed to dyspnea at rest over the last 15 days and chest pain from the onset of the symptoms, with palpitations, orthopnea and paroxysmal nocturnal dyspnea. Multidetector CT (MDCT) showed a filling defect involving the entire right main pulmonary artery, extending to the ipsilateral lobar branches and to the main branch of the pulmonary arteries, with increased maximum diameter and lobulated right pulmonary artery (arrows). (B) PET-CT demonstrated a linear hypermetabolic area along the trajectory of the pulmonary artery suggestive of malignancy (SUV max 12 g/ml). The patient was diagnosed with primary pulmonary artery sarcoma.
Lung neoplasm is the most common cause of SVCS (80%), being the non-small cell lung carcinoma (NSCLC) the most common (50%) (Fig. 2), followed by small cell carcinoma (SCLC) (25%) and non-Hodgkin lymphoma (NHL) (10%). Overall, 2–4% of lung neoplasms result in SVCS at some point in the disease, being more frequent in SCLC (approximately 10% at diagnosis). SVCS appears in less than 2% of patients with NSCLC, but the higher incidence of this type of tumor results in more frequent SVCS than in SCLC. SVCS appears in 2–4% of NHL, being the most common subtypes the diffuse large cell and lymphoblastic lymphoma. Diffuse large B cell lymphoma with sclerosis is the subtype that most frequently causes SVCS. Most NHLs result in SVCS secondary to extrinsic compression by lymph nodes, but intravascular proliferation can also occur (angiotropic lymphoma).

There are other malignancies associated with SVCS, including mediastinal lymph node metastases. The severity depends on the development of the collateral vascular system. In most cases, the symptoms develop gradually but sometimes this condition is asymptomatic. Dyspnea is the most common symptom. At physical examination, other symptoms include facial rubor and erythema, face, neck and chest edema, and dilated cutaneous veins over the abdomen, chest and upper extremities.

The five main collateral pathways are the:

1. Azygos-hemiazygos system.
2. Paravertebral.
3. Internal mammary.
4. Lateral thoracic and thoracoepigastric veins.
5. Anterior jugular venous system.

Some authors describe the veins around the scapula, on the back and shoulder as the most common whereas others mention the azygos vein.

Chest radiography shows abnormal findings in 84% of cases, being the most common signs the widening of the mediastinum (64%) and pleural effusion (26%).

CT is essential in the evaluation of SVCS allowing us to see the level and extent of the obstruction and to identify the collateral pathways and the underlying cause. The presence of collateral vessels is a strong indicator of SVCS. Multi-detector CT with MIP and 3D reconstructions is useful in the detection of local stenosis and to evaluate the relationship of the great vessels and the extent of collateral vessels (Fig. 2).

The treatment depends on the type and extent of the tumor, being the average life expectancy about 6 months. Chemotherapy provides good results in about 60% of SCLC and lymphomas. Radiotherapy gives a complete response in 15–60% of cases, even though with side effects. Endovascular stenting is a more effective and less invasive technique, providing good mid-term results.

Extrinsic compression or invasion from a pulmonary neoplasm can affect the pulmonary veins (PV) (Fig. 3). It has also been described the extension through the PV into the left atrium from a pulmonary neoplasm, most commonly a bronchogenic carcinoma, and to a lesser degree pulmonary sarcomas or metastases of carcinoma. This can result in cardiac arrest caused by obstruction of the mitral valve or pulmonary tumor embolism.

Extension into the great vessels of the mediastinum is considered stage T4 in NSCLC staging; therefore, surgical treatment is not the first choice. Great vessels include the aorta, SVC, inferior vena cava, main PA (pulmonary trunk), intrapericardial portions of the right and left PA and intrapericardial portions of the left and right superior and inferior PV. Invasion of more distal branches does not qualify for T4.

Extrinsic compression or invasion from lung neoplasms also can affect the PA (Fig. 4). However, PA invasion by a lung neoplasm detectable on CT is rare, but microscopic vascular invasion is common. It can be misdiagnosed with a primary PA tumor or pulmonary thromboembolism. The prognostic significance is unclear although vascular invasion has...
been described as a poor prognostic factor. A retrospective study found no correlation between macroscopic arterial invasion and histologic type and grade and lymph node staging for bronchogenic carcinoma. Lung carcinomas with polypoid growth in the main PA seem to belong to a more aggressive subtype called adenosquamous carcinoma. Although invasion of the main trunk or intrapericardial portions of the PA by a NSCLC qualify for T4 and, therefore surgical treatment is not indicated, the management of these patients is controversial. Recent articles have reported a more favourable outcome for patients undergoing extended surgery with postoperative pathological N0 disease.

Lung tumors with infiltration of the thoracic aorta are even less common than advanced tumors infiltrating the espine, the carina or the apex of the lung. There might be hemoptysis secondary to direct invasion of the aortic wall by lung cancer, resulting in an aortobronchopulmonary fistula. Direct invasion by an infectious process, an aortic aneurysm or dissection that breaks open and bleeds into the lung or erodes into a bronchus may also result in acute and massive hemoptysis. Symptoms include back pain, cough, dyspnea and hemoptysis. Hemoptysis is the most common symptom (about 95%) and is frequently massive, but can also be minor and intermittent. Minor hemoptysis may precede a fatal hemorrhage with cases described anywhere from 2 days to 1 year.

Cases of periaortic lymphoma, dissection of the ascending aorta caused by neoplastic disease and invasion of the aortic wall by a squamous cell carcinoma mimicking an intramural hematoma have also been described.

Choked radiographic findings include pulmonary condensation, widening of the mediastinum and pleural effusion (hemothorax). Infiltration of the thoracic aorta by a malignant pulmonary neoplasm has poor prognosis; however, tumor resection including the involved aorta provides long-term survival with N0 disease.

Distant spread

Pulmonary tumor embolism (PTE) is rarely diagnosed before death, probably because the clinical and radiological findings are unspecific. Autopsy findings revealed that 2–26% of patients with a known malignancy develop PTE. Breast, lung and gastric cancer are the most common neoplasms associated with PTE, but cases of

**Figure 4** 74-Year-old smoker with a 2-month history of dyspnea and cough. MDCT showed a pulmonary mass in the middle lobe invading the right pulmonary artery (arrow). Fiberbronchoscopy and biopsy were performed and the patient was diagnosed with endobronchial SCLC and died one month after diagnosis.

**Figure 5** 71-Year-old smoker and heavy drinker presented to our hospital with hemoptysis. He reported having a several-day history of hemoptysis and had four more episodes on the day of admission to the emergency department. At admission, he had a new episode of hemoptysis of fresh blood. He also reported anorexia and weight loss over the last year. The sputum cytology revealed atypical cells suspicious for malignancy. MDCT showed a lung mass and air bubbles within the lesion, probably caused by necrosis, in close contact with the descending thoracic aorta that showed irregularities in the left lateral wall indicative of infiltration (arrow). 72 h after admission, the patient had an episode of life-threatening hemoptysis that needed arteriography with embolization. Radiation therapy or surgery was not performed and the patient had a new episode of massive hemoptysis with respiratory failure and cardiopulmonary arrest. Patient died 18 days after admission.
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Figure 6  (A) 57-Year-old man with a left renal tumor waiting for surgery. Preoperative chest radiograph showed an increase in size and density of the right pulmonary hilum (arrows). (B) MDCT with multiplanar reconstruction showed a central filling defect with increase in size of the right pulmonary artery and of the lower segmental branches (white arrows). Lower image shows a renal mass (*) with thrombosis of the left renal vein (black arrow). These findings are compatible with hypernephroma with renal vein invasion and pulmonary tumor embolism. The patient underwent chemotherapy for 2 months before surgery, which reduced significantly the lesions in the right pulmonary artery and lower segmental branches on follow-up CT scan. Subsequently, the patient underwent left radical nephrectomy.

neoplasms in liver, prostate, pancreas, bone, indifferen-
tiated carcinoma, ovary, renal bladder, cervix, colorectal, kidney, mesothelioma, Wilms tumor, esophagus, parotid, melanoma, myxoma, thyroid, choriocarcinoma, vulvar carcinoma and neurogenic sarcoma have also been reported.\textsuperscript{34}

Mortality rates are very high, but early diagnosis and proper treatment (primary tumor resection) can provide a cure in selected cases (hypernephroma, myxoma and choriocarcinoma).\textsuperscript{32}

Most cases have been previously diagnosed with malignancy; however, in some cases PTE is the first manifestation. Right atrial myxoma and hypernephroma tend to embolize to the central and segmental PA\textsuperscript{33} (Fig. 6).

In many instances, distant metastases appear before the onset of respiratory symptoms. Dyspnea is the most common presenting symptom and is severe, usually acute or subacute and invariably progressive. Other symptoms are pleuritic chest pain, cough, weight loss, fatigue, syncope and hemoptysis.\textsuperscript{32}

In most cases, there is also pulmonary hypertension and right atrial overload. The presence of cor pulmonale is a poor prognostic sign often leading to death within 4–12 weeks.\textsuperscript{32}
Figure 7  (A) 38-Year-old man with a 2-month history of irritating dry cough. He had dyspnea that progressed to dyspnea with minimal exertion over the past 8 days, so he sought emergency care. MDCT scan demonstrated lymph adenopathies at the supra-aortic trunk level as well as left para-aortic (arrow), right hilar, mesenteric and retroperitoneal adenopathies. (B) MDCT also showed an
Chest radiographic findings are normal in most cases. Cardiomegaly and prominent PA are rare (less than 50%). There might be local or diffuse interstitial opacities. The absence of pulmonary opacities with dyspnea and hypoxemia suggests pulmonary vascular disease. IV contrast enhanced CT scan reveals filling defects in the main branches of the PA, obstructions and dilations of subsegmental branches, subpleural lines and wedge-shaped opacities secondary to pulmonary infarction, and signs of pulmonary venous hypertension. The presence of lymphadenopathy or lymphangitic carcinomatosis may suggest PTE. Ventilation-perfusion lung scintigraphy produces a typical pattern of multiple small, peripheral and subsegmental perfusion defects, with normal ventilation. The four main types of PTE are:

1. Large proximal tumor embolism resulting in acute pulmonary hypertension syndrome secondary to occlusion of the main PA or main lobar branches.
2. Microscopic tumor embolism involving small arteries and arterioles, with progressive dyspnea and subacute pulmonary hypertension.
3. Microvascular pulmonary invasion that may be part of the generalized lymphatic involvement, which can explain the presence of diffuse interstitial opacities.
4. Combination of the three previous mechanisms.

Pathologic findings include tumor emboli mixed with malignant cells with or without obliterator arteritis, and may coexist with lymphangitic carcinomatosis. Differential diagnosis includes pulmonary thromboembolism or pulmonary embolism secondary to other causes (septic, fat, amniotic fluid, foreign bodies and parasites). Biopsy may be indicated in those cases where the definite diagnosis is needed for treatment.

Pulmonary tumor thrombotic microangiopathy (PTTM) is a rare form of tumor embolism (Fig. 7). It is seen at autopsy in 0.9–3.3% of patients with extrathoracic malignancies. High-resolution chest CT reveals centriflobular nodules and branching linear opacities with tree-in-bud appearance. This pattern is caused not only by small airway diseases, but also by vascular abnormalities.

There are two pathogenic mechanisms:

1. Filling of centriflobular arteries with tumor cells.
2. Thrombotic microangiopathy: extensive fibrocellular intimal hyperplasia in small PA and arterioles (carcinomatous endarteritis) induced by tumor microemboli.

Histologic findings reveal arterial occlusion caused by tumor cell, peripheral arterial dilatation and extensive fibrocellular intimal hyperplasia. PTTM must be included in the differential diagnosis of dyspnea of unknown origin, particularly in case of prior diagnosis of mucin secretory adenocarcinoma. A pulmonary biopsy establishes the diagnosis, but antemortem diagnosis is uncommon. Vascular disease related to oncology treatment

Surgery

Thromboembolic events may occur in as much as 26% of patients after pulmonary resection. Vascular stumps are more vulnerable to thrombus formation. A case of post-bilobectomy stump thrombosis for lung cancer has been described; however, most cases reported in the literature refer to pneumonectomies.

Postpneumonectomy PA stump thrombosis most commonly appears during the first days after surgery (Fig. 8). In 12.4% of a series of patients, stump thrombosis was observed at postpneumonectomy CT (in 82% of patients the thrombus was present on the initial CT scan and on subsequent CT examinations). There might be reduction in thrombus size and those that remain stable have concave shape. Almost none of the patients develop propagation of the thrombus outside of the stump, suggesting a benign natural history of this condition. Stump thrombi appear with equal frequency on either side. There seems to be a relationship between stump length and the development of thrombosis, probably a consequence of a change in flow dynamics. It seems, therefore, prudent to leave the PA stump as short as possible. Pulmonary embolism secondary to deep venous thrombosis and tumor recurrence should be considered in the differential diagnosis.

Radiation therapy

Radiation-induced cardiovascular complications often develop late after therapy. Vascular complications include early stenosis of the coronary artery or calcifications of the ascending aorta. Radiation-induced vasculopathy is time and dose dependent. Venous capillaries and sinusoids are the most sensitive to ionizing radiation, being endothelial cells the most vulnerable. Radiation-induced vasculopathy usually occurs after about 10 years and is limited to the radiation field. When thrombosis and rupture occur, the damage increase in diameter of the main trunk of the pulmonary arteries (right image) and cardiomegaly at the expense of the right heart cavities (left image). (C) MDCT (lung window) showed multiple centriflobular opacities with bilateral and diffuse distribution with no clear lobar preference. (D) During hospitalization, the patient had chest pain and dyspnea with cardiopulmonary arrest and death. Post-mortem examination revealed disseminated metastatic disease of a signet-ring cell carcinoma with pancreatic immunophenotype and massive bilateral pulmonary tumor embolism. Histopathological analysis (hemotoxylin–eosin staining) revealed intimal hyperplasia related to fibroblast growth with reduced arterial size and pulmonary thrombi occluding the arterial lumen (*). These finding were compatible with pulmonary tumor thrombotic microangiopathy.
becomes clinically significant. Stenoses and occlusions are the most common lesions.

Coronary stenosis occurs after radiation therapy mainly given for Hodgkin disease and it involves the proximal portions.\textsuperscript{1}

Calcifications of the ascending aorta (Fig. 9) are secondary to radiation-induced aortitis and appear thin and well-defined.\textsuperscript{41,43} They are due to deposition of calcium salts as a sequela of a scarred intima or media after aortitis\textsuperscript{41} and are indistinguishable from atherosclerosis.

**Chemotherapy**

**Central venous catheter-related complications**

Central venous catheters (CVC) are commonly used in cancer patients allowing delivery of medication. Incidence of complications is 15\%.\textsuperscript{44,45} They can be immediate (6.2--11.7\%) or long-term (6.6\% reported in a retrospective study).\textsuperscript{45} Immediate complications include arterial puncture and hematoma (the most common), misplacement or pneumothorax-hemothorax.\textsuperscript{45} Most common late complications include infection, venous thrombosis and pulmonary embolism, mechanical (pinch off syndrome, rupture or migration) and extravasation.

CVC thrombosis occurs in 41\% of patients despite preventive measures, increasing the risk of infection.\textsuperscript{46} Thrombosis and infection are the most common late complications. Only one-third is symptomatic,\textsuperscript{46} and it can result in SVCS associated with collateral vessel formation. Complications related to CVC thrombosis include postphlebitic syndrome (15--30\%) and pulmonary embolism (11\%; only half are symptomatic).\textsuperscript{46} Risk factors include type of malignancy, type of CVC and locations of insertion site and catheter tip.\textsuperscript{45,46} Placement of the catheter tip high in the SVC and insertion from the left subclavian vein result in a higher incidence of thrombosis.\textsuperscript{46} Treatment includes medical treatment or catheter removal.

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**Figure 8** 58-Year-old man with lung cancer treated with pneumonectomy and subsequent chemo- and radiation therapy. Initial MDCT (right image) did not show pulmonary artery abnormalities, but a 2-year follow-up MDCT scan showed a concave filling defect in the stump of the left pulmonary artery post-pneumonectomy (arrow).

**Figure 9** 85-Year-old man with hypertension and ischemic cardiomyopathy (unstable angor) that was asymptomatic for years. He was diagnosed with NSCLC that was treated with radical radiation therapy during 1.5 months (70 Gy). MDCT, comparing the initial study performed with IV contrast administration (right upper image) with the post radiation therapy study without contrast administration (left upper image) performed one year later, revealed calcifications that appeared more pronounced in the aortic button on the post radiation therapy study (*). Lower images show a significant improvement in the lung mass on the post-radiation therapy scan (arrow). Later, the mass progressed and, given the cardiovascular comorbidity of the patient, it was decided to provide symptomatic treatment.
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Figure 10  (A) Gastric cancer in a 41-year-old man undergoing chemotherapy. MDCT showed a central venous catheter (CVC) in the superior vena cava (SVC) properly positioned (white arrow) and a migrated catheter located in the pulmonary arteries (black arrow). (B) MDCT oblique MIP reconstruction showed the CVC properly positioned in the SVC (white arrow) and the migrated catheter in the right main pulmonary entering the lower lobar branch, bending on itself and becoming apparent in the contralateral main pulmonary artery (black arrows). Removal of the catheter was unsuccessful; the old reservoir was retrieved confirming that the catheter was fractured 2–3 cm from it and a new CVC was placed.

Removal is indicated in case of infection, misplacement of the distal end of the catheter or irreversible obstruction.

Extravasation is a severe complication that occurs in 0.1–6.5% of cases. Its causes include rupture and migration of the catheter or perforation of the SVC wall. Extravasation of chemotherapy agents results in significant adjacent tissue damage and tissue necrosis can occur in severe cases, requiring surgery.

Fracture of a CVC is a rare complication (0.2–1%). Fracture may happen at the moment of insertion or anytime after. The pinch-off syndrome involves the compression of the catheter in the subclavian vein between the clavicle and first rib, causing the catheter to fracture and migrate. Clinical presentation of this complication may be subtle and only a minority of patients present with symptoms (chest pain, palpitations or arrhythmias). Migration of the fragmented catheter may result in PA thromboembolism and pseudoaneurysm, the removal of the catheter is thus indicated (Fig. 10).

Pulmonary embolism

The association between thromboembolic disease and cancer is well established in the literature. Along with cancer itself, chemotherapy also contributes to the activation of coagulation. Patients with malignant neoplasms and thrombosis have a lower survival rate. Moreover, patients with cancer have a four- to eightfold higher risk of dying after an acute thrombotic event.

In oncology patients, pulmonary thromboembolism occurs in three settings: synchronous diagnosis of thromboembolism and tumor, incidental diagnosis during cancer follow-up, or diagnosis in symptomatic patients.

Cancer diagnosed at the same time as an episode of venous thromboembolism is associated with advanced disease stage and poorer prognosis (Fig. 11).

Incidental pulmonary embolism is seen in 1.5% of routine CT and in 1.8–4% of oncology patients in retrospective studies. Retrospective studies reported that in 67–75% of cases the emboli were not detected at the initial CT. For this reason, careful evaluation of the PA during CT follow-up is important in these patients. Prevalence increases in hospitalized patients, in advanced stages of the disease and in patients undergoing chemotherapy. The most common malignancies associated with thrombosis are those of the breast, colon, and lung, reflecting the prevalence of these malignancies in the general population. When data were adjusted for disease prevalence, the cancers most strongly associated with thrombotic complications are those of the

Figure 11  NSCLC in a 66-year-old woman with pleural fluid cytology positive for adenocarcinoma. Initial MDCT showed a pulmonary mass in the left upper lobe (*) and pleural effusion with nodular thickening compatible with primary lung neoplasm with metastatic pleural involvement. Filling defects were seen in the upper lobar and segmental branches of the right pulmonary artery (arrows), compatible with pulmonary thromboembolism associated with the malignancy.
Figure 12  (A) 63-Year-old woman with mesothelioma undergoing chemotherapy. MDCT, comparing the initial study (left) and the two-month follow-up (right), demonstrated a filling defect corresponding to a pedunculated thrombus in the left lateral wall of the thoracic descending aorta (arrow). (B) Sagittal MDCT multiplanar reconstruction clearly demonstrated the mobile pedunculated thrombus at the origin of the thoracic descending aorta (arrow). (C) MDCT, comparing the initial study (right) and the two-month follow-up (left), shows a scar in the spleen corresponding to splenic infarction (black arrow in upper left image), and a triangular-shaped hypodense lesion in the right kidney, corresponding to a renal infarction (white arrow in lower left image). Radiological findings were compatible with a mobile thrombus in the thoracic descending aorta that caused the splenic and renal infarction secondary to visceral embolism.
pancreas, ovary, and brain. Locations are usually lobar and segmental and with a right-sided predominance.

Symptomatic pulmonary embolism has a prevalence of 11.8% on CT-angiography and a retrospective study reported that pulmonary embolism is usually central and with lower density thrombi.

Thoracic aortic mobile mural thrombus
TAMT is a rare condition that involves an aortic thrombus in a normal aorta and is a potential source of cerebral, visceral and peripheral embolism (Fig. 12).

The most common location is the descending thoracic aorta (28%) or the distal aortic arch (16%) with a preferente for the aortic isthmus, being the ascending thoracic aorta a less common location (5%).

The etiopathogenesis of TAMT is different from that of embolism associated with atherosclerotic disease. Possible causes include malignant neoplasms, hematologic diseases, therapy with exogenous steroids and estrogens, and primary endothelial dysfunction. Generalized hypercoagulability (cancer patients or patients in chemotherapy) and endothelial vasculopathy have been proposed as the most important factors in TAMT formation, with hypercoagulability seen in 40% of cases.

It can be an incidental finding, but most TAMT are detected during evaluation for distal emboli to the viscera.
or extremities. In autopsy series, the incidence was 0.45%.

Incidence of T AMT with embolism ranges between 0.8% and 9%. Embolization to the lower extremities is the most common (60%), followed by mesenteric embolism (18%). Embolization to the lower extremities originates in the abdominal aorta in 80% of cases and in the descending thoracic aorta in 20% of cases. Mesenteric emboli originate in the abdominal aorta in 56% of cases and in the thoracic aorta in 44% of cases. There can also be emboli to the renal arteries (6%), upper extremities (6%), cerebral arteries (2%) and coronary arteries (1%).

Transesophageal echography used to determine the origin of thrombi allows visualization of pedunculated thrombi floating in the aortic lumen. However, part of the aortic arch and the abdominal aorta usually cannot be visualized with transesophageal echocardiography. Magnetic resonance and multidetector CT can be useful in the diagnosis of T AMT as well as to determine the location and extent of the mural thrombus. Identification of the base of implantation can be useful to determine the optimal surgical approach and technique.

Its management is not clear and there are several therapeutic approaches. Medical treatment is the first alternative, particularly in symptomatic patients. Surgery may be performed when heparin therapy fails. Stenting can be a valid alternative to surgery.

A new embolization event may occur in 14.7% of cases and the average recurrence is about 8 months in a different location from the initial event. This fact supports the need of long-term anticoagulant therapy after surgery or endovascular treatment, and of clinical follow-up with imaging studies.

This condition should be included in the differential diagnosis of embolic events, particularly in young patients without a history of heart disease or recurrent peripheral embolism without a known cause.

Aortic dissection

An aortic dissection is a serious condition associated with hypertension. Some drugs such as bevacizumab have been associated with hypertension and worsening of preexisting hypertension, and can therefore result in aortic dissection (Fig. 13). This drug acts as an antiangiogenic and it is used in combination with cytotoxic chemotherapy. Hypertension is the most common toxicity associated with bevacizumab (up to 32%). Other antiangiogenic agents such as sunitinib and sorafenib can induce hypertension. If the medical treatment cannot control the hypertension, the antiangiogenic therapy should be discontinued. Bevacizumab is used for the treatment of colon, lung and kidney cancer, usually in the elderly, in whom the incidence of hypertension is higher.

Conclusions

Thoracic vascular disease of oncology patients can influence the treatment, management and prognosis of these patients. Multidetector CT represents an excellent tool in the diagnosis and follow-up of these patients. Due to the fast development and the emergence of new cancer therapies, further complications may arise in the near future, especially related to thoracic vascular disease.

Authorship

Responsible for the integrity of the study: DVP.

Conception of the study: DVP and JAS.

Design of the study: DVP.

Acquisition of data: DVP, JAS, OPM, ARP and EPN.

Analysis and interpretation of data: DVP, JAS, OPM, ARP and EPN.

Statistical analysis: not applicable.

Bibliographic search: DVP and JAS.

Drafting of the work: DVP.

Critical review with intellectually relevant contributions: DVP, JAS, OPM, ARP and EPN.

Approval of the final version: DVP, JAS, OPM, ARP and EPN.

Conflict of interest

The authors declare no conflict of interest.

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