Thoracic vascular disease in oncology patients

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Learning objectives

1. To review the causes of thoracic vascular disease in oncology patients.
2. To show the diagnostic imaging findings of these various causes, focusing on multislice CT.
3. To discuss utility of diagnostic imaging for this purpose and the clinical significance of the entities presented.

Background

Oncology patients require frequent diagnostic imaging follow-up. These patients may present vascular disease, which is clearly depicted by multislice CT. In this exhibit we will describe the causes, imaging findings, and clinical significance of thoracic vascular disease diagnosed in oncology patients.

Imaging findings OR Procedure details

1. NEOPLASTIC DISEASE

1.1. PRIMARY TUMORS

Primary sarcomas of the pulmonary artery (Fig. 1-7) are frequently diagnosed as pulmonary thromboembolism, and are derived from mesenchymal cells from the intima of the pulmonary artery. They can be intraluminal or sessile polypoidal masses.
originating in the intima of the pulmonary artery. Approximately half are intraluminal and the other half are transmurally spread to the adjacent lung, bronchial wall or lymph glands. There frequently exist pulmonary nodules. The most common symptoms are dyspnea (72%), thoracic pain (45%), coughing (42%), and hemoptysis (24%). A chronic clinical development without severe dyspnea suggests this diagnosis. Between 13-81 yrs. (average of 49.3 yrs.). No sexual predominance.

In chest radiograph, the most characteristic presence is that of a unilateral hilar mass with the distribution of the pulmonary artery projected towards the lung. If the mass extends to the pulmonary parenchyma, it can simulate pulmonary neoplasm.

The CT shows a heterogeneous mass that expands the pulmonary artery with extravascular invasion, condensation or subpleural nodules, pleural effusion or cardiomegaly, often confused with pulmonary embolism. In 86%, there exists a filling defect that occupies the totality of the lumen of the main or proximal pulmonary artery and expansion of a portion of the affected pulmonary artery. The extraluminal extension is another specific find. The presence of a filling defect in the whole of the lumen of the main or proximal pulmonary arteries can be the initial find in the CT.

FDG PET/CT can be useful in diagnosis, as the FDG detects it, as opposed to pulmonary embolism.

Poor prognosis (survival of 6% after 5 yrs). The average survival expectancy after symptoms begin is approximately 12 months.

1.2. SECONDARY TUMORS

1.2.1. EXTRINSIC COMPRESSION OR LOCAL INVASION OF THORACIC MALIGNANCIES

1.2.1.1. VEINS

The Superior Vena Cava Syndrome (SVCS) is due to obstruction of the SVC or its principal tributary veins by intraluminal occlusion or extrinsic compression and/or invasion by benign or malignant disease. The most common cause is malignant neoplasm, although thrombosis related to catheters or pacemakers has increased in recent years. There may be a direct extension of the tumor or compression (Fig. 8 and 9). Vascular invasion can lead to thromboflebitis and occlusion.

The most common malignant neoplasm is pulmonary neoplasm (80%), although there are other causes such as lymphoma or metastasis. Non-small cell lung cancer (NSCLC) is the most common (50%), followed by that of small cell lung cancer (SCLC) (25%) and Non Hodgkin's Lymphoma (NHL) (10%). 2-4% of pulmonary neoplasm develop SVCS
at some stage of the disease, the most frequent being in SCLC (approximately 10% at the onset of the disease) *(Fig. 10-12).* There may be SVCS in less than 2% of NSCLC, although the incidence is higher given that this syndrome is more common in SCLC.

In NHL there may exist SVCS in 2-4% of cases, the most common subtype being the diffuse of large cells and lymphoblastic lymphoma. B cell lymphoma with sclerosis is the most frequently cause of SVCS. Many NHLs cause SVCS due to extrinsic compression caused by adenopathies, although there may exist intravascular extension (angiotropic lymphoma) *(Fig. 13 and 14).*

There are other malignant tumors associated with SVCS, including mediastinal lymph node metastasis *(Fig. 15).*

Severity depends upon the development of the collateral vascular system. In the majority of cases, the symptoms develop gradually and may be asymptomatic. The most common symptom is dyspnea. Clinical findings: Facial flushing and erythema, edema of the face, neck and thorax; dilation of thoracoabdominal and upper extremity cutaneous veins.

There may be 4 principal routes of collateral circulation:

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

Some authors refer to the veins around the scapula, back or shoulder as the most common and others the azygos vein.

The chest radiograph is abnormal in 84% of cases, the most frequent signs being: mediastinal widening (64%) and pleural effusion (26%).

CT is essential for evaluation, allowing visualization of: level and extent of the obstruction, identification of collateral routes and the underlying cause. The presence of collaterals is a strong indicator of SVCS. MSCT with MIP and 3D volume rendering is useful for the detection of focal stenosis and visualization of the location of the large vessels and the extension of the collateral vessels.

Treatment depends upon the type of cancer and its extension. Average life expectancy is approximately six months. Chemotherapy treatment gives good results in approximately 60% of SCLCs and lymphomas. Radiotherapy gives a successful response in 15-60% of cases, although there are many side effects and it usually fails in the following year. Endovascular stent is very effective and only slightly invasive, with good medium term results.

The **Pulmonary Veins (PV)** can be affected by extrinsic compression *(Fig. 16)* or invasion in cases of pulmonary neoplasm *(Fig. 17 and 18).* Extension to the left atrium
through the PV has been described, above all in bronchogenic carcinomas, the remaining cases being primary or secondary sarcomas. Heart failure due to mitral obstruction or pulmonary tumor embolism may result. In cases where invasion of the PV by the tumor is suspected, ligation of these veins may be useful as soon as possible during the operation and as close as possible to the entry to the left auricle, performing an intrapericardial pneumonectomy.

1.2.1.2. ARTERIES

The **Pulmonary Arteries (PA)** can be affected by pulmonary neoplasm due to extrinsic compression *(Fig. 19 and 20)* or invasion *(Fig. 21 and 22)*. Invasion of the pulmonary neoplasm into the pulmonary artery is rarely seen, although microscopic vascular invasion is frequent. It can be confused with a primary tumor or pulmonary embolism.

The resulting prognosis is not clear, although the vascular invasion has been described as a negative prognostic factor. In a retrospective study, there was no detected correlation between microscopic arterial invasion by bronchogenic carcinoma and the type and histological grade and lymph stadiation. In cases of polypoidal growth in the main PA, a more aggressive histological subtype, denominated adenosquamous carcinoma, was observed. Some authors give a more favourable prognosis for patients with extensive surgery and NO disease.

The **Thoracic Aorta** is more rarely affected by advanced pulmonary tumors than the spinal column, the carina or the pulmonary apexes *(Fig. 23-25)*. There may be hemoptysis due to direct invasion of the aortic wall by pulmonary neoplasm, causing aortobronchial pulmonary fistula. Direct invasion by an infectious process, an aortic aneurism or aortic dissection breaking towards the lung or eroding a bronchus can also lead to acute and massive hemoptysis. The aortobronchial pulmonary fistulas are observed in 85% of hemoptysis with aneurysms of the lower thoracic aorta. The symptoms can be: back pain, coughing, disnea and hemoptysis. Hemoptysis is the most common symptom (approximately 95% of cases) and is usually massive, although it can be minor and intermittent. Minor hemoptysis can precede a fatal haemorrhage in 2 days to 1 year.

Cases of periaortic lymphoma, dissection of the ascending aorta due to infiltration by neoplastic disease, and invasion of the aortic wall by squamous cell carcinoma simulating an intramural hematoma have been reported.

In chest radiograph, the findings are: pulmonary condensation, mediastinal widening and pleural effusion (hemothorax).
Infiltration of the thoracic aorta by malignant pulmonary neoplasm has a poor prognosis, although surgery on the tumor and the affected aorta can achieve long term survival with N0 disease.

1.2.2. DISTANT EXTENSION

1.2.2.1. PULMONARY TUMOR EMBOLISM (PTE)

PTE is infrequently diagnosed before death, probably due to unspecific clinical and radiological findings. Between 2-26% in autopsies with a known malignant disease. Frequently associated neoplasms are breast, lung and gastric; although there are other published cases: liver, prostate, pancreas, bone, undifferentiated carcinoma, ovary, urinary bladder, cervix, colorectal, kidney, mesothelioma, Wilms tumor, oesophagus, parotid, melanoma, mixoma, thyroids, coriocarcinoma, vulvar carcinoma and neurogenic sarcoma. The mortality rate is high, although early diagnosis and appropriate treatment (surgery on the primary tumor) can achieve an apparent cure in selected cases of hypernephroma, mixoma and coriocarcinoma.

The majority of cases are diagnosed prior to a malignant disease, although there are cases in which this is the initial state. Mixoma of the right auricle and the hypernephroma tend to embolize in central and segmental arteries (Fig. 26-30).

In many cases there exists metastasis in other organs before any respiratory symptoms. Dyspnea is the most commonly occurring symptom, being severe, usually acute or subacute and invariably progressive. Other symptoms are: pleural thoracic pain, coughing, weight loss, fatigue, syncope and hemoptysis.

In the majority of cases there exists pulmonary hypertension and right ventricular overload. The presence of cor pulmonale is a bad sign and often leads to death within 4-12 weeks. The PaO2 is mainly reduced to below 50 mmHg and the alveolar-arterial oxygen (A-aO2) gradient is increased. The high pulmonary artery pressure suggests a progressive sub-acute process, given that an acute rise above 50 mmHg is incompatible with life.

The chest radiograph is normal in the majority of cases. Cardiomegaly and prominent pulmonary arteries are infrequent (less than 50%). There may be localized or diffused interstitial opacities. The absence of pulmonary opacities in the presence of dyspnea and hypoxemia suggests pulmonary vascular disease.

CT can show:

1. Subpleural lines and wedge-shaped opacities in areas of pulmonary infarction.
2. Adenopathies, pulmonary venous hypertension or carcinomatous lymphangitis.
3. Filling defects in main branches and dilation of subsegmentary branches.

Pulmonary scan with ventilation/perfusion gives a typical pattern with multiple, small, peripheral and subsegmentary perfusion defects, with usually normal ventilation.

The 4 basic types of PTE are:

1. Large pulmonary tumor embolism: Acute pulmonary hypertension syndrome due to occlusion of the main pulmonary arteries or lobar branches.
2. Microscopic tumor embolism: Small arteries and arterioles. In the majority of cases, progressive dyspnea and sub-acute pulmonary hypertension.
3. Microvascular invasion from generalized lymphatic affectation: In some cases, diffuse interstitial opacities.
4. Combination of the 3 previous mechanisms.

The pathological findings include thrombosis mixed with tumor cells, with or without obliterator artheritis.

The differential diagnosis includes pulmonary thromboembolis or pulmonary embolism due to other causes (septic, fat, amniotic fluid, foreign bodies and parasites).

The majority of cases present severe pulmonary arterial hypertension from the start. Biopsy could be suitable in cases in which a definitive diagnosis is necessary for treatment.

1.2.2.2. THROMBOTIC MICROANGIOPATHY OF PULMONARY TUMORS (TMPT)

TMPT is a rare form of pulmonary tumor embolism (Fig. 31-38). In autopsies, 0.9-3.3% show extrathoracic neoplasia.

In HRCT, centrilobular nodules connected to branching linear opacities can be appreciated, related to a tree-in-bud pattern. This tree-in-bud pattern is usually caused by small airway diseases, but can also be due to vascular anomalies.

There are 2 pathogenic mechanisms:

1. Filling of centrilobular arteries by tumor cells.
2. Thrombotic microangiopathy: Extensive intimal fibrocellular hyperplasia of small pulmonary arteries (carcinomatous endarteritis) induced by tumoral microembolism.

Histology detects arterial occlusion by tumor cells, peripheral arterial dilation and extensive intimal fibrocellular hyperplasia.
PTTM must be included in a differential diagnosis of disnea of unknown origin, above all with diagnosis prior to mucino-secretory adenocarcinoma.

The diagnosis is performed by pulmonary biopsy, although it is rarely diagnosed pre-mortem.

1. **VASCULAR DISEASE RELATED TO ONCOLOGICAL TREATMENT**

2.1. **SURGERY**

Thrombotic episodes can appear in 26% of pulmonary resections. Vascular stumps may be more vulnerable to the formation of thrombus. A case of post-bilobectomy by pulmonary neoplasm has been reported, although the majority of cases refer to pneumonectomies (Fig. 39 and 40).

**Postpneumonectomy pulmonary artery stump thrombosis (Fig. 41 and 42)**

Postpneumonectomy pulmonary artery stump thrombosis takes place in the first few days of post-surgery. In one series, CT detected postpneumonectomy in 12.4% (82% in the initial CT and the rest in later CTs). There may be a reduction of thrombus and, in those that remain stable it has a concave form. There was no propagation of thrombus outside the stump in any of the patients, so the nature of this complication seems to be benign. There is no left or right predominance. The length of the stump seems to contribute to thrombus formation, probably due to changes in the dynamics of blood flow, and so it seems prudent to leave the pulmonary artery stump as short as possible.

The diagnostic differential should be performed with pulmonary embolism due to deep vein thrombosis or tumor recurrence.

2.2. **RADIATION THERAPY**

Cardiovascular complications due to radiotherapy are often late in appearing. Among vascular complications, we can find premature coronary artery stenosis or ascending aortic calcification.

Radiation-induced vasculopathy is time and dose dependent. The venous capillaries and the sinousoids are the most sensitive to ionizing radiation, the endothelial cells being the most vulnerable. Usually appears in approximately 10 years and is limited to the field of radiation. It can reach the stage of thrombosis and rupture, thus it is clinically significant. The most frequent lesions are occlusions and stenosis.

**Post-radiotherapy coronary stenosis** is seen above all in Hodgkin's lymphomas and affects the proximal portions.
Ascending aortic calcification (Fig. 43 and 44) can appear with radiation induced aortitis, and is fine and well defined. It is due to calcium salt deposits on the tissue as a consequence of scarring on the intima or media by aortitis. It is indistinguishable from atherosclerosis.

2.3. CHEMOTHERAPY

2.3.1. COMPLICATIONS OF CENTRAL VENOUS CATHETERS

Central venous catheters (CVC) are frequently used in oncological patients for the administration of medication. The incidence of complications is 15%. These can be immediate (6.2-11.7%) or late (6.6% in a retrospective study). Amongst the immediate we find: arterial puncture and hematoma (the most frequent), malplacement or pneumothorax-hemothorax. The most common amongst the late complications are: infection, venous thrombosis and pulmonary embolism, mechanics ("pinch off" syndrome, breakage or migration) and extravasation.

Thrombosis can appear in 41% of cases, in spite of preventive measures, conditioned by a greater risk of infection. Thrombosis is the most common late complication alongside infection.

Only a third of cases have symptoms. The complications of thrombosis related to catheters can be postphlebitic syndrome (15-30%) and pulmonary embolism (11%; only half being symptomatic).

Risk factors include: type of neoplasia, type of catheter and location of the insertion into the distal extremity. The high placement of the distal extreme catheter in the SVC (Fig. 45 and 46) and insertion in the Subclavian vein are related to a higher incidence of thrombosis.

Treatment can be medical or the removal of the catheter. Removal is recommended in cases of infection, malplacement of the distal extreme or irreversible obstruction.

Extravasation is a severe complication that can be observed in 0.1-6.5% of cases. The causes can be breakage and migration of the catheter or perforation of the SVC wall (Fig. 47-49). The extravasation of the chemotherapy of these patients causes severe damage to the adjacent tissue, leading to tissue necrosis which may require surgery.

Breakage is a rare complication (0.2-1%). It can be produced during the insertion of the catheter or later on. "Pinch-off syndrome" consists of the compression of the catheter between the clavicle and the first rib, located in the subclavian vein, which can break and migrate. Clinical presentation is usually subtle and only a minority of cases show symptoms (thoracic pain, palpitations or disrhythmias). The migration of the fragmented catheter can lead to a thromboembolism, thus removal is recommended (Fig. 50-52).
2.3.2. PULMONARY EMBOLISM

The association between thromboembolic disease and neoplasia is well established in medical literature. As well as the tumor itself, chemotherapy also contributes to the activation of coagulation. Patients with malignant neoplasm and thrombosis have a lower survival rate. Furthermore, patients with cancer have a 4-8 times higher risk of death due to an acute thrombotic episode.

In oncological patients, we can observe pulmonary thromboembolism in three types of situation: diagnosed alongside the tumor, incidental diagnosis during follow-up on neoplastic disease, or diagnosis in symptomatic patients.

Neoplasm diagnosed at the same time as a venous thromboembolic episode has been seen to be associated with an advanced stage of disease and a worse prognosis (Fig. 53 and 54).

**Incidental pulmonary embolism** has been noted in 1.5% of routine CTs and, in oncological patients, it has a prevalence of between 1.8-4% in retrospective studies. In a study it was not detected in an initial CT, therefore it is important to carefully evaluate the pulmonary arteries in routine CTs of oncological patients. This prevalence increases in hospitalized patients in an advanced stage of their disease. The risk is also increased for patients undergoing chemotherapy treatment. The most common neoplasms are: breast, colon and lung, reflecting the prevalence of these malignant neoplasms in the general population. When the data are adjusted by the prevalence of disease, the neoplasms which are most strongly associated are: pancreas, ovary and brain. The location is usually lobular and segmentary with predominance on the right (Fig. 55).

**Symptomatic pulmonary embolism** is prevalent in angioCT, at 11.8%, and has been reported in a retrospective study to be usually central and with thrombus of lower density (Fig. 56).

2.3.3. MOBILE THROMBUS IN THORACIC AORTA

**TAMT (Thoracic Aortic Mobile Mural Thrombus)** (Fig. 57-59) is a rare occurrence defined as an aortic thrombus in a normal aorta and it is a potential source of cerebral, visceral and periferal embolism.

It is most frequently located in the descending aorta (28%) or in the distal aortic arch (16%) with a predilection for the aortic isthmus. 5% in the ascending aorta.

TAMT has a different pathogeny to the embolism associated with atherosclerotic disease. Possible pathogeny: malignant neoplasm, haematological disease, steroids
and exogene estrogens, and primary endothelial disease. Generalized hypercoagulation (eg. neoplastic patients on chemotherapy treatment) or vascular endothelial disease have been proposed as the most important factors in the formation of TAMT. There is hypercoagulability in almost 40% of cases.

It may be an incidental finding, but the majority of cases are discovered during the evaluation of visceral or extremity distal embolisms. The incidence is 0.45% at autopsy. TAMT incidence with embolism varies between 0.8-9%. Embolism of the lower extremities is the most frequent (60%), followed by mesenteric embolism (18%). 80% of lower extremity embolism originates in the abdominal aorta and 20% in the descending thoracic aorta. 56% of mesenteric embolisms originate in the abdominal aorta and 44% in the thoracic aorta. Embolisms may also occur in the renal arteries (6%), upper extremities (6%), cerebral arteries (2%) and coronary arteries (1%).

Evaluation of the origin of embolisms by transesophageal ultrasound allows the visualization of pedunculated thrombus floating in the aortic lumen. Transesophageal ultrasound have the disadvantage of not being able to visualize portions of the aortic arch and the abdominal aorta. MRI and MSCT can be useful in the diagnosis, the exact location and the definition of the extension of the aortic mural thrombus. The location of the base of the implantation is necessary in order to determine the optimum route and surgical technique.

Handling is not clear and there are various therapeutic options. Medical treatment is the first alternative, above all in asymptomatic cases. Surgery can be considered in the event of the failure of medical treatment with heparin. Treatment by means of an endovascular stent can reduce morbidity and mortality in those patients treated with surgery.

There can be a relapse in 14.7% of cases, and the average time of reappearance is approximately 8 months. This supports the need for anticoagulant treatment during a long post surgery or an endovascular treatment and clinical follow up with imaging technology.

This pathology should be considered in the differential diagnosis for embolism episodes, above all in young patients without heart disease or recurrent peripheral embolisms of undetected cause.

**2.3.4. AORTIC DISSECTION**

**Aortic dissection** is a serious pathology related to arterial hypertension. Some medications such as bevacizumab have been related to the appearance of arterial hypertension or the worsening of pre-existing arterial hypertension, and therefore may potentially provoke aortic dissection *(Fig. 60 and 61)*. This medication acts as an antiangiogenic and is used in combination with cytotoxic chemotherapy. The most common toxicity is arterial hypertension (in up to 32%). Other antiangiogenics also provoke hypertension (sunitinib and sorafenib). Medical treatment does not usually
control hypertension, and so the antiangiogenic treatment should be removed. The use of bevacizumab is recommended in colonic, lung and renal neoplasias, and these patients are usually of an advanced age, in which the incidence of hypertension is higher.

1. **MISCELLANEA**

3.1. **ACQUIRED DISEASE**

3.1.1. **ATHEROSCLEROSIS AND RELATED CONDITIONS**

**Vascular atherosclerosis** is very common in patients with pulmonary neoplasm, given that the two conditions present common risk factors such as smoking and chronic respiratory disease (Fig. 62 and 63). The presence of comorbidity in non-small cell lung cancer (NSCLC) is considered an important prognostic factor, affecting the survival and morbidity in patients who are operated on for pulmonary neoplasia with associated cardiovascular disease.

**Thoracic aortic aneurysms** are associated with atherosclerosis as the most frequent cause, and can therefore be associated with pulmonary neoplasm and simulate mediastinal masses in chest radiograph (Fig. 64 and 65). The presence of calcification in the intima and the proximity of the predicted position in the aorta help diagnosis.

3.1.2. **MYCOTIC AORTIC ANEURYSM**

**Infected or mycotic aortic aneurysms** (Fig. 66 and 67) are uncommon, (0.7-2.6% of all aortic aneurysms) and consist of the breakage of the arterial wall with the formation of a blind pouch adjacent to the arterial lumen. Prevalence of mycotic aortic aneurysms treated: 0.7-1%. Delay in treatment or absence of it often leads to fulminant sepsis, spontaneous arterial rupture and death.

The locations of mycotic aneurysms in order of frequency are: aorta, peripheral arteries, cerebral arteries and visceral arteries. *Staphylococcus* and *Streptococcus* are the most frequent germs which cause this.

There can be precipitant causes: arterial trauma (29%), immunodepression (24%), concurrent sepsis, bacterial endocardytis (17%), congenital cardiovascular defects (10%) or primary (3%). Immunodepression can be caused by: malignant solid or haematological neoplasm, lymphoproliferative diseases, alcoholism, steroids or chemotherapy, chronic renal failure, autoimmune diseases and diabetes.
Mycotic aneurysms can rupture during surgery in 53% to 75% of cases. Mortality 16-40%. In 7-24% of infected aortic aneurysms, there is free rupturing (63-100% mortality) and in 47-61% of cases there is contained rupturing at discovery.

MSCT is the examination of choice for diagnosis. Normally, the aorta is affected in different locations to atherosclerosis. The most commonly affected parts are: descending thoracic, thoracoabdominal and upper abdominal. The radiological findings are: periaortic edema (fat stranding or hypodense concentric ring), concentric or exocentric inflammatory soft tissue, periaortic gas (infrequent), focal saccular dilation, absence of calcification in the mycotic aneurism, and extravasation of the contrast with hematoma in the case of rupture. The periaortic soft tissue and fat stranding are the most common findings (48%).

The treatment of small and asymptomatic mycotic aneurysms consists of intravenous antibiotic treatment for a period of four to six weeks and monitoring by image tests. Large and symptomatic aneurysms require urgent surgery in combination with antibiotic therapy. Residual aneurysms or those that grow during follow up image monitoring are also surgically treated.

3.2. CONGENITAL DISEASE

Congenital vascular anomalies can be discovered incidentally in oncological patients and are usually asymptomatic. However, there are situations in which their diagnosis is important: insertion of central venous catheter (left superior vena cava), vascular anomalies that simulate pulmonary neoplasm or mediastinal adenopathies (pulmonary arteriovenous malformations, right aortic arch and congenital venous anomalies), vascular anomalies that condition atypical manifestations of pulmonary neoplasm (azygos lobe), and congenital venous anomalies that could complicate pulmonary neoplasm surgery (partial anomalous pulmonary venous drainage).

Persistent left superior vena cava can simulate mediastinal mass on the left edge at the level of the aortic arch and must also be identified before the insertion of a central venous catheter (above all if there is no superior vena cava on the right side) it being possible to simulate malplacement in the chest radiograph (Fig. 68 and 69).

Right aortic arch has the appearance of a right paratracheal mass in the chest radiograph (Fig. 70 and 71). The most common form is that associated with an aberrant left subclavian artery and is usually asymptomatic.

Anomalous unilateral single pulmonary vein is a rare entity consisting of a unilateral pulmonary vein that includes all the veins of a lung drained in the left atrium. In chest radiograph, it can simulate a pulmonary nodule, especially in the lateral projection of retrocardiac location (Fig. 72 and 73).
Azigos lobe is an anatomical variant found in 1% of autopsies and in approximately 0.4% of chest radiographs. It is produced due to the penetration of the azygos vein in the right superior lobe during the embryological development that drags the two pleural layers, trapping a portion of the right superior lobe. The azygos vein lobe can contain or present adjacent diseases, pulmonary neoplasm being amongst these, giving rise to a dense azygos vein lobe (Fig. 74 and 75).

Partial anomalous pulmonary venous drainage is observed in approximately 0.3-0.5% of cases. In the left superior lobe, the pulmonary veins drain into a vertical vein which runs towards the left brachiocephalic vein. Usually asymptomatic when not associated with cardiac disease. If it is located in the lobe of a pulmonary neoplasm during surgery by lobectomy, no problems should arise (Fig. 76). In the event of it being located in a different lobe, this anomaly should be treated in order to avoid possible right cardiac failure due to increased blood flow through the anomalous vein.

Images for this section:
**Fig. 1:** Man 68 yrs. Stress dyspnea (3 m. of evolution), thoracic pain, palpitations, orthopnia and nocturnal paroxystic dyspnea. Chest radiograph showed peripheral nodular opacity in right lung. (\*), increase in size of right pulmonary hilum (white arrows) and loss of volume of right lung.
Fig. 2: MSCT. Filling defect affecting the totality of the main right pulmonary artery with extension to haemolateral lobe branches and trunk of pulmonary artery. The maximum diameter of the Right Pulmonary Artery was increased, with lobular morphology.
Fig. 3: MSCT. Loss of volume in right lung and septal intralobular thickening on right lung due to collateral circulation through intercostal arteries.
**Fig. 4:** MRI. Endoluminal tumor in Right Pulmonary Artery and lobular branches that extend to the main pulmonary artery with slight enhancement of contrast except for the component of the main pulmonary artery.
Fig. 5: PET-CT. Lineal hypermetabolic area that follows the trajectory of the right pulmonary artery, suggestive of malignancy (SUV maximum 12g/ml).
Fig. 6: MSCT at 10 m. Increase in size of the arterial lesion in right lung, extending to main trunk and left pulmonary artery, affecting segmentary branches of left upper lobe.
**Fig. 7:** MSCT at 10 m. Pulmonary nodule in parahilar left lower lobe of 8 mm. diameter, new appearance, probably a metastasis. Final diagnosis was primary sarcoma of pulmonary artery.
**Fig. 8:** MSCT with previous study of 8 months (images on the right). Man 72 yrs. with right pneumonectomy by NSCLC. Tumor can be appreciated with soft tissue density mass compressing the superior vena cava and the left atrium (red lines).
Fig. 9: MSCT (MIP reconstructions). Visualization of the compression of the superior vena cava although it remains permeable, with collateral circulation through azygos vein.
Fig. 10: Man of 56 yrs. with SCLC. MSCT with adenopathies in right paratracheal, precarinal, subcarinal regions and right hilum. Probable invasion of superior vena cava with irregularity of right lateral wall and important compression with sharpening of right pulmonary artery.
Fig. 11: MSCT with MIP reconstructions (upper images) showing the obstruction of superior vena cava with collateral circulation through azygos vein and diaphragmatic vein that drains into inferior vena cava. Focal hepatic lesions compatible with metastasis (bottom left image).
Fig. 12: MSCT at 2 months after chemotherapy according to cisplatin/etoposide scheme. Marked radiological improvement of mediastinal lesions seen in previous study and clinical feature of superior vena cava syndrome.
Fig. 13: Man 35 yrs. with superior vena cava syndrome presenting type B large cell mediastinal primary lymphoma. MSCT showed anterior mediastinal mass (*) and thrombosis in innominate vein and superior vena cava (red arrows).
**Fig. 14:** MSCT with multiplanar reconstructions (coronal and sagittal). Mediastinal mass (*) and thrombosis in superior vena cava (red arrows). The patient was treated with chemotherapy with marked radiological and clinical improvement.
Fig. 15: Man 64 yrs. with mesothelioma with pleural and mediastinal extension. MSCT showed mediastinal mass compressing superior vena cava (red arrows) with infiltration of mediastinal fat and bilateral pleural effusion.
Fig. 16: Man 54 yrs. with NSCLC. MSCT comparing initial study (images right.) with post-chemotherapy CT (images left). Pulmonary mass in left lower lobe with left hilar and mediastinal (predominantly subcarinal) extension associated with left pleural effusion. Bilateral compression of the pulmonary veins that improve with post-chemotherapy CT (red arrows).
Fig. 17: Patient 73 yrs., ex-smoker. MSCT showing pulmonary mass in left lower lobe (*) invading pulmonary veins. Thrombus in Left Superior Pulmonary Vein (red arrows) and amputation of Left Lower Pulmonary Vein (white arrow).
Fig. 18: MSCT with multiplanar reconstruction (oblique) showing the presence of thrombus in Left Superior Pulmonary Vein. Left pneumonectomy and resection of left atrium were performed. Histology showed the presence of poorly differentiated squamous cell carcinoma, with invasion of the left atrium wall.
Fig. 19: Patient 48 yrs, ex-smoker. Dry cough and left thoracic pain (2 weeks’ evolution), which is the reason he went to emergency department. Chest radiograph showed central pulmonary mass with loss volume of the left superior lobe.
Fig. 20: MSCT. Left hilar mass compressing Left Pulmonary Artery. Final diagnosis was disseminated lung neoplasm (by retroperitoneal metastasis).
Fig. 21: Man 74 yrs. Smoker with dyspnea and cough for two months. Chest radiograph showed large pulmonary mass located in middle lobe.
Fig. 22: MSCT. Pulmonary mass in middle lobe invading the right pulmonary artery (red arrows). The patient was diagnosed with pulmonary neoplasm by fibrobronchoscope and biopsy of an endobronchial lesion in the middle lobe and died a month after diagnosis.
Fig. 23: Man 71 yrs. Smoker and heavy drinker who arrived for hemoptysis refers to various hemoptysis episodes days before and the day of arrival at hospital has 4 coughing episodes. At emergency department, he presented hemoptoic expectoration with fresh blood. Anorexia and weight loss occurred in the last year. The cytology of sputum showed atypical cells suspicious of malignancy. MSCT showed pulmonary mass with air bubbles that suggest necrosis, in close contact with descending thoracic aorta.
Fig. 24: MSCT shows irregularity in the left lateral wall of descending thoracic aorta in contact with pulmonary mass of the left lower lobe, suggestive of infiltration of the vessel wall (red arrows).
Fig. 25: After 72 hours of admission, the patient presents threatening hemoptysis that requires arteriography with embolization. The arteriography shows irregularity around the aorta on left side that later is shown to be compatible with an aortic ulcer (red arrows). The patient died 18 days after admission.
**Fig. 26:** Man 57 yrs. with left renal tumor infiltrating inferior vena cava. Pre-surgical chest radiograph showed increase in size and density of right pulmonary hilum.
Fig. 27: MSCT. Central filling defect with increase in size of right pulmonary artery and lower segmentary branches.
**Fig. 28**: MSCT with multiplanar reconstruction. The upper images showed filling defects in the right pulmonary artery and lower segmentary branches. The bottom image shows the renal tumor (*) with thrombosis of the left renal vein (red arrow). The findings are compatible with hypernephroma with invasion of the renal vein and pulmonary tumor embolism.
Fig. 29: MSCT. Centrilobular opacities in left lower lobe probably related to thrombotic microangiopathy of pulmonary tumor.
**Fig. 30:** This patient was treated with chemotherapy prior to surgery for 2 months, significantly reducing the lesions to the right pulmonary artery and lower segmentary branches in follow-up CT (images on the left). Later, a radical nephrectomy was performed.
Fig. 31: Man 38 yrs. with irritating dry cough for two months. In the last 8 days, progressive dyspnea. Chest radiograph showed increase in size of pulmonary hila and cardiomegaly.
Fig. 32: MSCT. Supraaortic, left paraaortic and right hilum adenopathies.
Fig. 33: MSCT. Increase in width of the main trunk of pulmonary arteries and right cardiac chambers.
Fig. 34: MSCT. Multiple lung nodular opacities of centrilobular location and diffuse and bilateral distribution with some isolated area of ground-glass.
Fig. 35: MSCT with multiplanar reconstructions (coronal). Centrilobular lung opacities with diffuse bilateral distribution. No clear predominance of any pulmonary lobe.
Fig. 36: MSCT. Small mesenteric and retroperitoneal adenopathies.
Fig. 37: During their admission, the patient presented thoracic pain and dyspnea with cardiorespiratory arrest and death. Necropsy showed metastatic disease of pancreatic carcinoma. In pulmonary parenchymas, massive bilateral tumor embolism. Histology: Tumor thrombus occluding the arterial lumen.
**Fig. 38:** Histology: Hyperplasia of the intima layer, due to fibroblastic growth, producing a reduction of the width of the arterial lumen. The findings are compatible with thrombotic microangiopathy of pulmonary tumor.
**Fig. 39:** Man 76 yrs, ex-smoker. Right lower lobectomy due to lung carcinoma. MSCT compares the first post surgical study (right images) with a study after 6 months (left images). Filling defect in the right pulmonary artery (red arrows) resolved in the later CT.
Fig. 40: MSCT with multiplanar reconstructions (coronal and oblique). Loss of volume of the right lung due to a lower lobectomy and filling defect in the right pulmonary artery (red arrows) in relation to a thrombosis of the post-surgical stump.
**Fig. 41:** Man 58 yrs. with lung neoplasm treated by means of pneumonectomy and later by chemotherapy and radiation therapy. Initial MSCT (right image) does not showed any pulmonary artery alterations. However, in follow-up MSCT after 2 years we can appreciate a filling defect with a concave form in the post-pneumonectomy stump of the left pulmonary artery (red arrow).
Fig. 42: MSCT with multiplanar reconstruction (oblique). Filling defect with a concave form in the stump of the left pulmonary artery related to a thrombosis (red arrow).
Fig. 43: Man 85 years with arterial hypertension and asymptomatic unstable angor for several years. NSCLC treated by means of radical radiation therapy for 1.5 months (70 Gy). MSCT comparing initial study with endovenous contrast (right images) with post-radiation therapy non-enhanced examination (left images), performed a year after the previous study. Marked calcifications in the thoracic aorta in the post-radiation therapy CT.
**Fig. 44:** MSCT with multiplanar reconstructions (coronal) comparing the study before radiation therapy with the post-radiation therapy study (upper images). We can see more marked calcifications on the thoracic aorta in the post-radiation therapy study (*). In the lower images, marked improvement to the pulmonary mass in the post-radiation therapy study (red arrow). Subsequently, the pulmonary mass progressed and, given the cardiovascular comorbidity of the patient, a symptomatic treatment of the neoplasia was decided.
Fig. 45: Woman 74 yrs. with colon neoplasm in treatment with chemotherapy. MSCT showed thrombus in relation to the catheter, located in the superior vena cava.
**Fig. 46:** MSCT with multiplanar reconstruction (oblique). Filling defect in the superior vena cava due to thrombus in relation to the catheter, which presents a distal end in the superior vena cava (red arrows).
Fig. 47: Man 81 yrs. with rectal neoplasm under treatment with chemotherapy. Patient arrived at emergency department with sudden disnea. Chest radiograph showed an increase in the cardiac silhouette and probable bilateral pleural effusion. The central venous catheter is apparently located at the union of the superior vena cava and the right atrium.
**Fig. 48:** MSCT performed with clinical suspicion of pulmonary thromboembolism. Pericardial and bilateral pleural effusion. The central venous catheter is exteriorized by the front wall of the superior vena cava and with surrounding hematoma (red arrows).
**Fig. 49:** MSCT (coronal oblique and sagittal planes). Exteriorization of the catheter through the front wall of the superior vena cava (red arrows).
Fig. 50: Man 41 yrs. with gastric neoplasm under treatment with chemotherapy. Chest radiograph (on the right) showed the central venous catheter in the superior vena cava with distal end in the union of the superior vena cava and the right atrium. Another catheter was migrated in the right pulmonary artery. Previous chest radiograph from 2 months earlier (left image) showed a catheter in the superior vena cava with the distal end in the right atrium.
**Fig. 51:** MSCT showing the catheter correctly placed in the superior vena cava and the migrated catheter in the right main pulmonary artery, towards the lower lobar branch.
**Fig. 52:** MSCT with MIP reconstructions (oblique). Migrated catheter in the right main pulmonary artery introduced in the inferior lobar branch and the left pulmonary artery (left image). The withdrawal of the loose catheter was attempted without success and the old reservoir was removed, confirming that the catheter had been seccionated at 2-3 cm from the reservoir. A new catheter was inserted.
**Fig. 53:** MSCT showed filling defects in lobar and segmentary branches of the right pulmonary artery (red arrows) compatible with pulmonary thromboembolism associated with the pulmonary neoplasm.
**Fig. 54:** Woman 66 years with NSCLC and cytology of the pleural liquid positive for adenocarcinoma. Initial diagnosis MSCT showed pulmonary mass in the left superior lobe and pleural effusion with associated nodular pleural thickening compatible with primary pulmonary neoplasm with metastatic pleural extension.
Fig. 55: Man 71 years old with urinary bladder neoplasm treated with surgery and chemotherapy. MSCT showed filling defects in lower lobar and segmentary branches of the right pulmonary artery in relation to incidental pulmonary embolism.
Fig. 56: Woman 52 years with breast neoplasm and left thoracic pain. MSCT was performed and showed bilateral filling defects in main and lower lobar branches of pulmonary arteries related to pulmonary embolism, left pleural effusion, and ground-glass area in the left inferior pulmonary lobe probably due to pulmonary infarction.
Fig. 57: Woman 63 yrs. with mesothelioma under treatment with chemotherapy. MSCT (initial study on right images and 2 months later on left images). Filling defect related to a pendunculated thrombus adjoining the left lateral wall of the descending thoracic aorta (red arrows).
Fig. 58: MSCT with multiplanar reconstructions (coronal and sagittal). Mobile pendunculated thrombus at the beginning of the descending thoracic aorta (red arrows).
Fig. 59: MSCT comparing the initial study (right images) and those of two months later (left images). In the upper image, reduction of the left pleural effusion previously seen. In the central images, scar on the spleen corresponding to a post-infarction lesion (white arrow). In the lower images, hypodense lesion of a triangular morphology in the right kidney due to infarction (red arrow). The radiological findings are compatible with a mobile thrombus in the descending thoracic aorta with right renal and splenic infarctions due to visceral embolism.
**Fig. 60:** Woman 79 yrs. with renal neoplasm treated with nephrectomy, subsequently presenting distant metastasis. Treatment with chemotherapy, including sorafenib. MSCT in follow-up studies (initial, 3 m., 5 m. and 8 m.) showed progressive aortic dissection located in the descending thoracic aorta.
Fig. 61: MSCT with multiplanar reconstruction (oblique) showing dissection in the descending thoracic aorta (arrow). Treatment with sorafenib was suspended.
Fig. 62: Man 80 yrs. ex-smoker 12 years ago with intermittent claudication for 1-2 years. Chest radiograph showed pulmonary mass in the right lower lobe and calcification of the thoracic aorta.
**Fig. 63:** MSCT showed pulmonary mass in the right lower lobe in relation to pulmonary neoplasm and calcifications due to extensive atheromatosis in coronary arteries and thoracic aorta.
**Fig. 64:** Man 66 yrs. ex-smoker with peripheral athereiopathy. Chest radiograph showed pulmonary mass projected onto the left pulmonary hilum (\*\) and nodular opacity in a left paraspinal location (red arrows).
Fig. 65: MSCT shows a small aneurysm in the descending thoracic aorta, causing nodular opacity of left paraspinal location on the chest radiograph (red arrows).
Fig. 66: Man 69 yrs. smoker with thoracic pain, fever and progressive disnea. Chest radiograph showed left pulmonary mass.
Fig. 67: The patient presented sepsis due to S. Aureus, resistant to cloxicylin, and was diagnosed with NSCLC. MSCT presented pulmonary mass in the left lower lobe due to pulmonary neoplasm (*) and focal dilation in the descending thoracic aorta with absence of calcification (white arrow) and periaortic soft tissue compatible with mycotic aneurysm in the descending thoracic aorta.
**Fig. 68:** Man 54 yrs. with pulmonary neoplasm in treatment with chemotherapy. Chest radiograph showed the central venous catheter with entry through the right subclavian artery which runs towards the left mediastinal edge.
Fig. 69: MSCT showed the presence of the left superior vena cava with catheter inside, without appreciating the vena cava on the right side
Fig. 70: Woman 43 yrs. diagnosed with breast neoplasm prior to surgery. Chest radiograph showed right paratracheal opacity that displaces the trachea without appreciating the aortic button to the left.
Fig. 71: MSCT showed a right aortic arch without aberrant subclavian artery (*).
Fig. 72: Woman 43 yrs. with breast neoplasm. Chest radiograph showed retrocardiac nodular opacity in lateral projection (red arrow).
**Fig. 73:** MSCT with multiplanar reconstruction and 3D showing an anomalous unilateral single left pulmonary vein.
Fig. 74: Man 48 yrs. smoker with cervical pain for 4 months and peripheral facial paralysis in the last week. Chest radiograph showed well defined opacity along the right edge of paratracheal location (red arrow).
**Fig. 75:** MSCT showed pulmonary mass originating in the azygos vein lobe. The patient was surgically treated after chemotherapy, corresponding to NSCLC.
Fig. 76: Man 53 yrs. with pulmonary neoplasm in left superior lobe, prior to surgery. In MSCT, spiculated pulmonary nodule with central cavitation in the left superior lobe and partial anomalous pulmonary venous drainage of the same lobe (red arrows). The patient was surgically treated without complications.
Conclusion

Thoracic vascular disease in oncology patients is clinically important and may have an influence on the treatment and prognosis. Multislice CT is an excellent tool for the diagnosis and follow-up of these conditions.

Personal Information

References


