

Extrinsic compression of coronary and pulmonary vasculature

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Abstract: Coronary artery disease from atherosclerosis induced stenosis remains the leading cause of acute coronary syndrome (ACS) and death worldwide, however extrinsic compression of coronary arteries from adjacent anatomical and pathological structures is an infrequent but important diagnosis to be aware of, especially given the nonspecific symptoms of chest pain that mimic angina in patients with pulmonary hypertension (PHT) and congenital heart disease. Non-invasive CT angiography is an invaluable diagnostic tool for detection of coronary artery compression, pulmonary artery dilatation and pulmonary vascular compression. Although established guidelines are not available for management of left main coronary artery (LMCA) compression syndrome, percutaneous coronary intervention and stent implantation remain a feasible option for the treatment, specifically for patients with a high surgical risk. Treatment of pulmonary vein or artery compression is more varied and determined by etiology. This review article is focused on detailed discussion of extrinsic compression of coronary arteries, mainly the LMCA and brief discussion on pulmonary vasculature compression by surrounding anatomical and pathological entities, with focus on pathophysiology, clinical features, complications and role of imaging in its diagnosis and management.

Keywords: Coronary; compression; pulmonary hypertension (PHT); extrinsic; aneurysm; pulmonary venous; fibrosing mediastinitis (FM)

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Introduction

Extrinsic compression of coronary vessels from surrounding structures is an unusual etiology of coronary insufficiency and has been alluded to mostly in case reports and case series. Compression of coronary arteries from an enlarged pulmonary artery (PA) (1), unruptured left sinus of Valsalva aneurysm (SVA) (2,3), aortic root abscess (4), prosthetic pulmonary valve placement (5), stent placement in a PA conduit (6), metastatic cardiac tumor (7), parasitic infections (8), and ventricular pseudoaneurysm (9) (amongst others) have been described as etiologies of this potentially life-threatening condition that may lead to refractory angina, persistent myocardial dysfunction and sudden cardiac death. Cases of compression of the right coronary artery (RCA) and left anterior descending coronary artery (LAD) have been described as a result of aortic root pseudoaneurysms and SVAs (10-12). Extrinsic compression of pulmonary vasculature is a rare but important etiology of chest pain and dyspnea that may be encountered in various clinical scenarios including chest wall deformities such as pectus excavatum and malignant infiltration and/or compressive narrowing of PA and veins. In this article, we will focus on extrinsic compression of the coronary arteries and pulmonary vessels with discussion of relevant etiopathogenesis, anatomy, clinical manifestations, potential complications, cross-sectional imaging features, and management.

Coronary compression syndrome

Coronary compression syndrome is often indistinguishable from acute coronary syndrome (ACS)—a constellation of clinical symptoms resulting from a myocardial ischemic event. Approximately 7–29% of patients with pulmonary hypertension (PHT) may report typical characteristics of angina pain, which may be attributed to coronary insufficiency from compression by a distended PA rather than atherosclerotic stenosis of the coronary artery (1).

Relevant anatomy

Typically, the left main coronary artery (LMCA) arises from the left sinus of Valsalva (LSV) and courses 2–4 mm though the aortic wall, running at about 90° from the aortic ostium. It travels posterior to the pulmonary trunk, and then in the left atrioventricular groove, leftward, posterior and superior as it courses to its bifurcation or trifurcation into the left anterior descending, circumflex coronary arteries and ramus intermedius respectively (13-15). The RCA typically arises from the right sinus of Valsalva (RSV), courses anteriorly and to the right between the right atrial appendage and the PA and then descends vertically in the right atrioventricular sulcus. The left circumflex (LCX) is directed towards the left, travels in the atrio-ventricular groove and onto the diaphragmatic cardiac surface, usually terminating before reaching the posterior interventricular sulcus (15,16).

The main PA arises from the right ventricular outflow tract and courses superior, posterior and to the left of the aorta before it bifurcates into right and left main PA inferior to the arch, at the level of the carina. The right and left pulmonary arteries further divide into 2 lobar branches each, and thereafter into segmental and sub-segmental branches.

Coronary compression from enlarged PA

PA dilatation is a frequent finding with an incidence of 76.6% in a cohort of severe PHT patients (17). The prevalence of greater than 50% LMCA stenosis as a result of extrinsic compression from an enlarged PA is at least 6% in the overall PHT population and around 40% in the population with angina-like symptoms who had undergone a diagnostic procedure (18). Corday et al. (1957) first suggested that compression of the LMCA by a dilated PA might account for symptoms of coronary insufficiency in patients with PHT (19). Subsequently, several case reports and case series have surfaced describing LMCA compression as a complication of PHT (20-24). A 26-patient series identified a prevalence rate of 26.9% (1). An aberrant origin of the LMCA from the RSV or a rightward-positioned left coronary sinus predicts a higher risk of compression (25). Potential complications include myocardial ischemia, myocardial infarction, left ventricular dysfunction and ventricular arrhythmia (26-29). Sudden death has been reported in more than 25% of patients with PHT, possibly underscoring the importance of this diagnosis (30).

A massively enlarged main PA can occur with PHT, or in the presence of a pulmonary artery aneurysm (PAA). An enlarged PA from PHT may occur in the setting of chronic untreated left to right intracardiac shunts and chronic pulmonary thromboembolic disorder (31,32). Less frequent etiologies include connective tissue disorders, portal hypertension, HIV, heritable and drug-induced or idiopathic causes (32). Small stature has also been suggested as a risk factor of coronary compression from enlarged PA (26). Incidentally, minimal or no atherosclerotic disease in the LMCA or other coronary arteries is identified in this patient group as compared to other registries, likely due to a younger age group and underlying cardiac shunts (18). The average time for diagnosis of the LMCA compression is around 5 years after the diagnosis of PHT.

A rare cause of PA enlargement is a PAA, defined as focal dilatation of a blood vessel wall encompassing all three vascular layers beyond 40 mm or its maximal normal caliber based on various criteria. A pseudoaneurysm on the other hand is a focal contained rupture of the artery. Both entities can be complicated by thrombosis, dissection, rupture, and/or compression of adjacent structures (33). Coronary insufficiency may occur in the absence of PHT in the setting of a low pressure PAA, and secondary to its anatomical relation with PA (34).

A rare cause of symptomatic extrinsic coronary



Figure 1 Measurement of main pulmonary artery diameter on CT. The diameter of the pulmonary artery is measured at the level of the bifurcation and orthogonal to the long axis. LMCA, left main coronary artery; LVOT, left ventricular outflow tract.

compression is post-surgicalrelated to surgical implantation of a sewing ring and/or pulmonary or aortic prosthetic valve strut that, based on proximity to the LMCA, can impinge on this vessel. This is usually evident in the immediate perioperative period (5).

Coronary compression from unruptured SVA

SVAs are rare cardiac anomalies that occur in 0.14% to 0.96% of the population and manifest as dilatation in the aortic wall just superior to the three cusps of the aortic valve, between the aortic valve annulus and the sinotubular ridge (35-37). Congenital aneurysms are more frequent with an incidence of 0.1–3.5% of all congenital heart defects and often caused by weakness at the junction of the aortic media and the annulus fibrosus (38,39). Acquired aneurysms can occur in conditions affecting the aortic wall, such as infections (bacterial endocarditis, syphilis, or tuberculosis), trauma, or degenerative disease (atherosclerosis, connective tissue disorders, or cystic medial necrosis).

SVAs most frequently originate from the right coronary sinus (94%), followed by the noncoronary sinus (5%) and left coronary sinus (1%) (40). Unruptured SVAs are typically symptom free and clinical presentation is usually due to rupture or fistulization, however a number of case reports have been published in recent years describing compression of coronary arteries, particularly the LMCA, usually involving the left coronary sinus. This typically results in angina, myocardial infarction or even sudden cardiac death (41,42), thus prompt diagnosis and management are critical.

Imaging findings

Multidetector cardiac CT is a comprehensive tool for evaluating the degree of LMCA compression, angulation of the LMCA relative to the sinus of Valsalva, and evaluation of biventricular functions. On CT, the main PA artery diameter is measured at the PA bifurcation level, orthogonal to its long axis and 3 cm from the root (28) (*Figure 1*). The sex-specific cutoff value for main PA for men is 29 mm and for women, 27 mm using the 90th percentile. The ratio of the main PA to the ascending aorta diameter is 0.9 for both genders (43). The main PA diameter greater than 40 mm is identified as a strong predictor of the presence of extrinsic compression of LMCA with a sensitivity of 83% and a specificity of 70% in patients with PHT and angina (1,18).

Additionally, a PA/aortic ratio of more than 1.5 and a PA diameter index of 24 mm/m² portend as risk predictors for LMCA stenosis greater than 50% with >70% sensitivity and 68–70% specificity (18). However, not all symptomatic patients with compression of the LMCA greater than 50% have a main PA diameter of greater than or equal to 4 cm and vice versa, suggesting additional anatomic variables that affect the relationship between the LMCA and the PA.

As the PA enlarges, it causes inferior displacement of LMCA and decrease in the take off-angle of the artery from the aorta. Kajita *et al.* evaluated angiographic determinants of LMCA extrinsic compression and found that a mean LMCA take off angle of 23° (compared with an angle of 70° in the control group) resulted in inferior displacement of the LMCA adjacent to a dilated PA (25). Another report found that a LMCA angle of <30° relative to the left sinus of Valsalva increased the likelihood of significant myocardial ischemia (29) (*Figure 2*). A strict relationship between mean PA pressure and LMCA compression has not been demonstrated.

Four anatomic categories of patients have been described, based on the relationship between the main PA and LMCA (*Table 1*). The normal distance between the main PA and LMCA is greater than 1 mm. The LMCA is considered contiguous with the main PA if the distancePA is less than 1 mm but the artery is not indented or compressed. The LMCA is considered dislocated if the take-off angle of LMCA from the aorta is <60° irrespective of lumen stenosis. Stenosis is considered significant if luminal diameter decreases >50% (*Figures 3,4*). Selective coronary angiography is indicated when dislocation or significant stenosis pattern is identified on coronary computed tomography angiogram (CTA). Coronary angiography may also be considered in the presence of symptoms, even when LMCA is contiguous with main PA (*Figure 5*).

Chest radiograph may depict abnormal cardiomediastinal silhouette depending on the location, size, and presence or absence of rupture of SVA. SVAs vary in size from subtle dilatation of an aortic sinus to saccular outpouchings from the body or summit of the sinus. On 3D CT reconstructions, the sinus of Valsalva is measured in double short axis at the aortic root from the coronary sinus to its opposite trigone for accurate measurement (*Figure 6*). The end diastolic measurement of sinus of Valsalva is 3.2 ± 0.6 cm for men and 2.9 ± 0.5 cm for women (44).



Figure 2 The oblique coronal multi planar reformat CT image adjusted for the best visualization of the proximal left main coronary artery demonstrates the measurement of its take-off (TO) angle. The angle between two planes (1-orthogonal to the aortic annulus and 2-along the proximal left main coronary artery) is measured.

Management

The appropriate management of symptomatic patients with extrinsic compression of the LMCA secondary to PHT is not well established. Invasive approaches aimed at establishing adequate blood flow to the undersupplied myocardial tissue territories include percutaneous coronary intervention (PCI) and coronary artery bypass graft (CABG). PCI is minimally invasive and may include cardiac catheterization, balloon angioplasty and stent placement for vessel recanalization. The procedure has good long-term efficacy and safety and is preferred over CABG because of risks related to general anesthesia and cardiopulmonary bypass in patients with severe pre-capillary PHT (24). Stent selection and size is based on the vessel diameter, atherosclerotic changes in the coronary artery, and risk of bleeding complications. Drug eluting stent is preferred over bare metal stent, and the latter is favored if there is a prolonged risk of bleeding with dual oral anticoagulant and antiplatelet therapy (45). Rate of restenosis is around 11% and occurs from mechanical recoil, recompression, and instent neo-intimal hyperplasia but can be treated successfully with repeat PCI (18). CABG is a more invasive operation requiring blood vessels reimplantation from another part of the body to bypass the stenosed or occluded coronary segment. This surgery is preferred if concomitant operative intent to treat the underlying cause of PHT is being already considered (32).

In patients with a congenital defect and potentially reversible PHT, the correction of the congenital defect has been shown to decrease the progression of LMCA compression (46). Surgical treatments with symptomatic and prognostic benefits include atrial septostomy, reduction plasty of the PA, and lung transplantation.

There are currently no established guidelines for the management of PAAs. However, surgery remains the

| Table 1 Four ar | natomic categ | ories, based or | the relationship | between the main | n PA and LMCA |
|-----------------|---------------|-----------------|------------------|------------------|---------------|
|-----------------|---------------|-----------------|------------------|------------------|---------------|

| Findings | Normal | Contiguous | Dislocation | Significant stenosis |
|---|-------------------|----------------|----------------|----------------------|
| Minimal distance between the MPA and the LMCA | Greater than 1 mm | Less than 1 mm | Less than 1 mm | Less than 1 mm |
| Indentation/compression of LMCA | _ | _ | + | + |
| Take-off angle of the LMCA, <60 degree | _ | _ | + | + |
| Stenosis of greater than 50% | _ | _ | +/- | + |

MPA, main pulmonary artery; LMCA, left main coronary artery.



Figure 3 The illustration of the relationship between LMCA and main PA detected on cardiac CTA as categorized in four different groups. (A) Normal distance; (B) contigous; (C) dislocation; (D) significant stenosis. LMCA, left main coronary artery; CTA, computed tomography angiogram.

cornerstone of therapy for main PA lesions. Surgical intervention is considered if the aneurysm is 55 mm or greater, if the rate of dilatation is greater than 5 mm in 6 months, or if complications such as compression of adjacent structures, thrombosis, dissection, or rupture occur (47). Coronary revascularization may not be necessary if LMCA compression resolves after aneurysm surgery.

Aneurysms of the left sinus of Valsalva are extremely rare, and compression of the coronary artery resulting in coronary insufficiency is an uncommon complication (*Figure 7*). Surgical repair is required in cases of unruptured but symptomatic aneurysms causing ventricular outflow tract obstruction, coronary ostia obstruction, or malignant arrhythmias (42). The timing for intervention for unruptured aneurysms is still debatable as patients with unruptured, stable, or asymptomatic SVAs are usually followed clinically and with serial imaging. However, current guidelines recommend surgical correction if sinuses exceed 5.5 cm, or 4.5 cm in the setting of connective tissue disease. Repair should also be considered when the growth rate exceeds 0.5 cm/year (48). When surgically treated, unruptured SVA repair includes aneurysm exclusion and, in some cases, replacement of the aortic valve, saphenous vein bypass grafting or a combination there of (49).

Pulmonary venous compression

In the majority of patients, there are four pulmonary veins. The right superior vein drains the right upper lobe and right middle lobe, right inferior vein drains the right lower lobe, the left superior vein drains the left upper lobe and lingula, and the left inferior vein drains the left lower



Figure 4 Coronal and axial oblique multi planar reformat image (A) and (B) show significant left main coronary artery stenosis due to compression by an enlarged main pulmonary artery (C) in a 27-year-old man with pulmonary hypertension. LVOT, left ventricular outflow tract.



Figure 5 Oblique coronal multiplanar reformat CTA depicts dislocation of LMCA due to enlarged main PA. The main PA measures 5 cm and the take-off angle is less than 30 degree. No luminal narrowing of the left main coronary artery is noted. LMCA, left main coronary artery; CTA, computed tomography angiogram.

lobe. Variant anatomy includes a common pulmonary vein draining the left lung and accessory veins draining the right middle or right upper lobe (50). Near the insertion site into the left atrium, the pulmonary veins are intrapericardial and are surrounded by a sleeve of pericardium and myocardium.

Pulmonary vein compression has been described in a variety of clinical scenarios. Malignant lesions include bronchogenic carcinoma, esophageal tumors, metastases and lymphoma (51). Inflammatory lesions such as fibrosing mediastinitis, sarcoidosis, tuberculosis have been described (51-54). Bronchogenic cyst, aneurysmal dilatation of the aorta and PA, and post-surgical compression following implantation of a left atrial appendage occlude, as well as chest wall deformities such as pectus excavatum (55-59).

Malignant tumors can infiltrate as well as extrinsically compress the pulmonary veins. Infiltration of the intrapericardial portion of the pulmonary veins upstages a bronchogenic carcinoma to a T4 lesion, alters surgical planning, and decreases survival (60) (*Figure 8*). In



Figure 6 The measurement of sinus of Valsalva. The cross-cursors should be aligned orthogonal to aortic annulus on coronal (A) and sagittal (B) views in order to obtain double oblique short axis image of sinus of Valsalva (C), three linear distances for each sinus of Valsalva are measured (D).

patients with adequate epicardial and mediastinal fat, the pericardium may be visible and can be used to distinguish the intrapericardial from extrapericardial portion of the pulmonary vein. If the pericardium is not visible, infiltration of the epicardial fat or obliteration of the pulmonary vein ostium is suspicious for intrapericardial invasion. Choe et al. found that obliteration of the ostia by bronchogenic carcinoma is a reliable sign of intrapericardial extension for the superior pulmonary veins but a less reliable sign for inferior pulmonary veins (61). In patients with equivocal findings of pulmonary vein compression on chest CT, pulmonary vein evaluation can be performed with MRI using white-blood cine imaging, contrast-enhanced venography and phase-contrast velocity mapping (62). Indirect evidence of pulmonary vein compression includes asymmetric pulmonary edema due to venous congestion. Pulmonary

edema confined to the territory drained by the occluded pulmonary vein manifests as progressive local interstitial thickening and airspace opacities (63). On CT, disruption of pulmonary venous drainage results in increased pulmonary venous pressure, leading to decreased pulmonary perfusion which can be mistaken for pulmonary thromboembolism. It can progress to pulmonary infarction which manifests as airspace opacities with internal lucencies (64).

Sternal depression in patients with pectus excavatum distorts and displaces the heart (*Figure 9*). Although the right ventricle is the most frequently compressed chamber, there have been reports of right and left atrial, left ventricular, and pulmonary vein compression. Pulmonary vein angle, as determined by measuring the angle between the respective pulmonary veins and patient's midline on chest CT, has been correlated with the severity of sternal depression (65).



Figure 7 Reprinted with permission from reference. (A) Pre-operative imaging using computed tomographic angiography revealing giant aneurysm of the left sinus of Valsalva; (B) selective left main coronary artery angiogram revealing extrinsic compression of the left main coronary artery by the giant aneurysm of left sinus of Valsalva.



Figure 8 Axial Steady state free precession (SSFP) image in a patient with right hilar mass (small cell lung cancer) shows total occlusion of the right lower lobe pulmonary vein (arrow, A), coronal cardiac SSFP MRI image of the same patient demonstrates encasement and narrowing of the right lobar pulmonary arteries (arrows, B).

Treatment aims at resolving the cause of compression, often comprising antineoplastic therapy including radiation and chemotherapy, though percutaneous intervention, including endovascular stenting, has been described (63).

PA compression

Acquired PA compression is a rarely reported entity in adults. Compression of the right or left branch of the PA, without involvement of the main PA, is more common. Extrinsic PA compression, while rare, can result from a mediastinal tumor, including lymphoma, germ cell tumors, thymoma, carcinoid tumor, lung cancer, and mediastinal metastatic disease (66-68). More rare causes of compression have been reported secondary to mediastinal sarcoma (69), mesothelioma of the pericardium (70), and sternal tumors. Non-neoplastic etiologies include infection/inflammatory causes such as FM, mediastinal cyst, thoracic aortic aneurysm, calcified pericardial ring encircling the PA above pulmonary valve level, and compression by short saphenous venous CABG (71-74). Amongst mediastinal neoplasms, teratomas and Hodgkin's disease are the most Cardiovascular Diagnosis and Therapy, Vol 11, No 5 October 2021



Figure 9 Axial EKG-gated cardiac CT for the evaluation of pulmonary vein stenosis in a patient with history of prior pulmonary vein ablation demonstrates the compression of the left common pulmonary vein between descending aorta and coronary sinus. The proximal portion of the pulmonary vein (A red arrow) is mildly narrowed compared to distal part (yellow arrow); (B) the vertebral column is located more anteriorly resulting in decreased anteroposterior diameter of the chest and increased Haller index (a/b).



Figure 10 Axial contrast enhanced chest CT revealed (A) extrinsic compression of main and proximal right pulmonary artery by hematoma in left pulmonic recess of transverse sinus (blue arrow) with HU (Hounsefield unit) of 53, note of Type A aortic dissection and pericardial hematoma (HU 50) in (B).

common cause of extrinsic pulmonic compression. It is rare for mediastinal neoplasms to compress the PA enough to produce hemodynamically significant obstruction and is likely due to tendency of mediastinal tumors to enlarge laterally. This is more likely to result in superior vena cava obstruction (74). Extrinsic compression of PA or its central branches manifests on cross sectional imaging as compression, displacement, distortion, extrinsic indentation, luminal narrowing and in severe cases as slit-like lumen or complete occlusion (*Figure 10*). Chronic compression can secondarily result in right ventricular hypertrophy, as well as pulmonary and tricuspid valvular regurgitation (70).

Chest pain and dyspnea are the most common clinical manifestations of PA compression. Systolic ejection murmur is the most frequent physical examination finding (73). Clinical manifestations can overlap with acute pulmonary embolism (69).

Extrinsic compression of the main and branch PAs due to neoplastic etiology is a rare cause of right ventricular hypertension which can result in right ventricular failure. Right heart catheterization with pulmonary angiography can confirm this diagnosis and assess severity of the obstruction. CT and MRI are less invasive but nevertheless accurate methods of diagnosis (*Figure 11*). 2D and 3D transthoracic

Figure 11 Post-contrast coronal chest CT reveals non-small lung cancer related large conglomerate soft tissue mass centered in the right superior mediastinal region causing complete obstruction of the right upper lobe pulmonary artery, irregular compression of the distal right pulmonary artery, near complete obstruction of the right upper lobe bronchus and invasion of SVC.

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echocardiogram (TTE) play pivotal roles as non-invasive tools for detection of hemodynamic compromise resulting from the pulmonary flow obstruction (68).

FM is an uncommon, progressive, benign inflammatory process related to proliferation of fibrotic tissue in the mediastinum. The localized and infiltrative subtype is more common and is frequently associated with granulomatous infection such as Histoplasma capsulatum. The diffuse form can be idiopathic or associated with autoimmune entities (75,76). Two distinct radiological subtypes are seen. On CT or MRI, granulomatous FM typically manifests as localized and infiltrative heterogeneously enhancing middle mediastinal soft tissue, usually with stippled or dense calcifications. The non-granulomatous subtype presents as diffuse and infiltrative mediastinal soft tissue involving multiple compartments (*Figure 12*). Calcifications are typically absent in this subtype (75,77).

FM characteristically presents on imaging with compression or obstruction of mediastinal vasculature and/ or the tracheobronchial tree and esophagus. FM-related obstruction of the central pulmonary arteries is typically



Figure 12 (A,B) Axial chest CT angiography, (C) coronal maximum intensity projection (MIP) image and (D) volume rendered 3 dimensional image show complete occlusion of right main pulmonary artery (white arrow) and right superior pulmonary vein (yellow arrow) due to right hilar and subcarinal ill defined soft tissue in a 40-year-old man with fibrosing mediastinitis. Note patent left sided pulmonary veins and tiny bronchial collaterals (thin arrow). LSPV, left superior pulmonary vein; RSPV, right superior pulmonary vein; LIPV, left inferior pulmonary vein.

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unilateral. It is characterized by hypoattenuating narrowing or obliteration of pulmonary vasculature due to surrounding soft tissue. This can result in hypoperfusion and mosaic attenuation on CT secondary to reduced vascularity of the affected lung parenchyma (77). Chronic PA compression can result in compensatory bronchial artery dilatation (78). MR angiography may demonstrate an attenuated PA, suggesting a chronic occlusive thrombus or periarterial fibrosis (76). Chronic PA occlusion due to FM may result in pulmonary infarction. Chronic pulmonary arterial hypertension and right ventricular failure can occur as a terminal complication of FM. Note that FM should be distinguished from chronic PA narrowing related to chronic thromboembolism, as these conditions can have a similar clinical presentation, but distinctly different management (79).

Clinical management

The clinical management of extrinsic PA compression is directed towards treatment of the underlying disease. Management of PA compression secondary to an advanced malignancy aims to provide symptomatic relief. Intervention such as stenting or angioplasty is considered in cases of right ventricular dysfunction, severe pulmonary valvular regurgitation, or worsening hemodynamics (80). A neoplastic etiology may benefit from surgery, radiation, or chemotherapy, though endovascular stenting of the PA can provide rapid palliation of malignant external compression.

Conclusions

Extrinsic vascular compression of coronary and pulmonary vasculature from neighboring anatomic and pathologic structures is a rare but an important etiology to consider in patients with angina and dyspnea. Patients with congenital heart disease are at particular risk for these conditions. PA dilatation is common among patients with PHT but adjacent LMCA compression is rare and may be dynamic with sporadic clinical manifestations. Pulmonary vein and artery compression can result from a wide range of neoplastic and non-neoplastic causes, with a similarly broad range of therapeutic options depending on etiology.

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