

- 7:30 – 8:00 Coffee and Pastries
- 7:30 – 7:45 Case of the Day
Diane C. Strollo, MD
- 7:45 – 8:00 Case of the Day
Shuji Adachi, MD

STR/NASCI
Cardiovascular Symposium

Moderator: Curtis E. Green, MD

- 8:00 – 8:20 Normal Cardiac Anatomy
William Stanford, MD
- 8:20 – 8:40 How Does a Noncardiac Radiologist Approach Cardiac MRI?*
- 8:40 – 9:00 Problem-Solving Techniques for Cardiac Cases
Stephen W. Miller, MD
- 9:00 – 9:20 Complications of Cardiothoracic Surgery
Jo-Anne O. Shepard, MD
- 9:20 – 9:40 Rheumatic Heart Disease
Curtis E. Green, MD
- 9:40 – 10:00 Questions
- 10:00 – 10:25 Break

Moderator: Robert M. Steiner, MD

- 10:25 – 10:45 Vascular Causes of an Abnormal Mediastinum
Rosita M. Shah, MD
- 10:45 – 11:05 Imaging of Acquired Aortic Disease
Gautham P. Reddy, MD, MPH
- 11:05 – 11:25 Traumatic Aortic Arch Injury: To Scan or To Squirt?
Joseph H. Tashjian, MD
- 11:25 - 11:45 The Multimodality Diagnosis of Pericardial Disease
Robert M. Steiner, MD
- 11:45 – 12:05 Current Therapy for Cardiomyopathy: VADs, Myocardial Resection and Cardiac Transplantation
Barbara L. Knisely, MD
- 12:05 – 12:15 Questions
- 12:15 – 1:15 Lunch

1:15–3:15 **Issues and Controversies in Lung Cancer Screening**

Moderator: Gordon Gamsu, MD

*Abstract not available at time of publication.

Tuesday



Normal Cardiac Anatomy

William Stanford, MD

University of Iowa Hospitals and Clinics, Iowa City, IA

Introduction

Computed tomographic (CT) images of the heart differ from the normal longitudinally oriented views one is accustomed to seeing in anatomy texts. In CT imaging the heart is often viewed in cross-sectional and oblique planes that are more familiar to echocardiographers than to diagnostic radiologists. However, since significant numbers of CT and magnetic resonance cardiac imaging examinations are being done, it becomes increasingly important to understand and become familiar with the axial, short and long axis projections used in cardiac imaging. It is also important to recognize the limitations inherent in these scanning projections.

The electron beam computed tomography (EBT) scanner is unique in that it can generate both static and dynamic images and, therefore, provide an additional dimension to the conventional CT imaging of the heart. In addition, the EBT scanner is capable of producing images of ventricular contraction and valvular motion in near realtime and can also provide information as to circulation pathways and arrival times of contrast within the cardiac chambers and great vessels.

Imaging Sequences and Contrast Administration

There are three imaging sequences that are used to define cardiac anatomy and function. If multiple target rings are sequentially imaged (flow mode), eight 8-mm slice thicknesses of the heart can be obtained in 224 msec. These slices are contiguous except for a 4-mm separation between images from adjacent target rings. Triggering is from the R wave of the ECG and all images are generated at about the same point in the cardiac cycle. Another important imaging sequence is the movie mode sequence. In this sequence same level, cross-sectional images of the heart can be obtained at a rate of 17 images per second. This sequence produces images from which it is possible to quantify cardiac function. The third sequence (volume mode) produces sequential tomographic slices of variable thickness as the patient moves through the scanner. This mode produces 100msec contiguous images of 1.5-, 3-, 6-, or 10mm slice thicknesses. Allowing for table movement each slice requires approximately 0.5 seconds and with imaging and moving the table, 40 slice thicknesses

can be obtained in 76 seconds. With the continuous volume scanning upgrade 40 slices of 6 or 10 mm thickness can be obtained in 17 seconds.

If contrast is administered it is given via a peripheral vein and, in order to eliminate the perturbations of heart rate, non-ionic contrast medium is generally recommended. In the flow mode imaging sequence 40 ml of contrast medium are administered at 10 ml/sec; in movie mode and volume mode sequences approximately 60-150 ml of contrast are administered at 1.4 to 2.0 ml/sec.

Positioning

In the evaluation of ventricular function, short-axis projections are the more appropriate. In these projections the ventricle is sliced in cross section similar to "slicing a cucumber". This projection provides visualization of the superior, inferior, medial, and lateral walls of the atria and ventricles; it does not visualize the left ventricular apex particularly well. Internal structures such as papillary muscles are prominently displayed; however, the mitral valve is less well visualized than on the long-axis images.

Because the heart is oriented with the apex displaced caudally and to the left, to obtain short-axis images the table is rotated 25° to the patient's right and tilted downwards 15°. This brings the longitudinal axis of the left ventricle into the scanning circle and orients the ventricle almost perpendicular to the scanner gantry. If necessary, an 18° wedge can be inserted under the patient's back and this would increase the tilt of the heart to 20-25° and produce a truer cross-sectional image of the ventricle. The latter maneuver is rarely done.

The long-axis or four-chamber view is the better projection for evaluating the cardiac apex, mitral valve, and aortic outflow tract. This projection is also used to evaluate medial and lateral left ventricular wall motion. To obtain this view the scanner table is kept level but moved 25° to the patient's left. This brings the heart more in line with the scanner gantry and, because the heart projects leftward and is tilted ventrally, the slices are oriented in a longitudinal configuration. The latter views are similar to the four-chamber views of echocardiography and the images are termed "horizontal long-axis images" in keeping with the standard imaging planes used in angiography and nuclear medicine.



Positional Correlations

Neutral Axis

The neutral axis is an excellent imaging projection to view the aorta, pericardium and cardiac chambers. In this configuration the table remains perpendicular to the scanner gantry. The pulmonary arteries and atria are cut in cross section as are the aortic root, aortic valve, both ventricles, pericardium, and adjacent mediastinal structures. Because the neutral axis closely approximates the short-axis and because the short-axis images are the more important from the functional viewpoint, only the short-axis projections will be illustrated.

Short Axis

The short axis is the projection used in calculating cardiac function. In this projection the orientation of the short-axis images changes slightly during cardiac contraction as the left ventricular outflow tract and aortic root move from a more nearly cross-sectional configuration to a more oblique configuration. This change in image configuration is caused by the base

of the heart descending and moving caudally during systole. Aortic valvular leaflet motion is well visualized in the short axis projection and usually all three leaflets of the aortic valve can be identified.

Long Axis

The long-axis better defines the mitral valve and left ventricular outflow tract. These images are also helpful in visualizing ventricular chamber size, apical and septal anatomy, and pericardial abnormalities. (Disclosure Statement: William Stanford, MD, is a member of the CT Advisory Board of Toshiba, Inc.)

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2. Stanford W. Normal MR Cardiac Anatomy in El Khoury G, Bergman RA, Montgomery WJ (eds). Sectional Anatomy by MRI New York, Churchill Livingstone. 1995.

How Does a Noncardiac Radiologist Approach Cardiac MRI?

Lawrence M. Buxt, MD

Abstract not available at time of printing



Problem-Solving Techniques for Cardiac Cases

Stephen W. Miller, MD
Massachusetts General Hospital

Objectives

Complex cardiac problems frequently require multi-modality imaging with chest radiographs, CT, MRI, and echocardiography. The objectives of this lecture are to (1) present an approach to the differential diagnosis of common cardiac problems, (2) describe the recent clinical and imaging advances in selected cardiac problems, and (3) briefly summarize the currently imaging approach to some cardiac problems that are yet to have sharply defined answers.

Mitral Valve Prolapse

First thought to be a leaflet abnormality, mitral prolapse actually may have abnormalities in all parts of the mitral apparatus. Primary mitral valve prolapse has mitral regurgitation when the leaflets are large and have multiple, redundant scallops, mainly on the posterior leaflet. The chordae typically are elongated and together with the scalloped valve allow regurgitation because the valve edges no longer coapt. With enlargement of the left ventricle from mitral regurgitation, the mitral annulus dilates and the heads of the papillary muscles separate, contributing to the regurgitation. Mitral prolapse can also occur with an otherwise normal mitral valve if the geometry of the left ventricle is distorted. A common example is in a patient with an atrial septal defect where the dilated right atrium and ventricle distort the shape of the left ventricle, allowing usually mild but occasionally severe prolapse.

Initial estimates of the prevalence of mitral valve prolapse ranged from 5 to 35 percent, based mainly on screening echocardiographic studies. But recent two- and three-dimensional analyses of the mitral valve have refined the geometric criteria for mitral valve prolapse. Prolapse now is defined if the leaflets have a left atrial displacement of more than 2 mm in systole from the line connecting the hinge points, and also have a thickness of at least 5 mm in diastole. With this new set of criteria, the prevalence of mitral valve prolapse ranges between 1 and 2 percent and the rate of heart failure, atrial fibrillation and cerebrovascular disease is no higher than among patients without prolapse.

Cardiac Tumors

A frequent referral to the cardiac radiologist is a mass seen on echocardiography. MRI is requested for tissue characterization. Other cardiac problems arise when a known mediastinal or lung tumor is adjacent to the heart, and there is concern that the pericardium contains tumor. Both CT and MRI are used to assess these types of problems, while angiography is usually reserved for interventional therapy. The first step is to be certain that the mass is actually a cardiac tumor. An aneurysm in the aortic root may deform the right atrium and compress the superior vena cava. Imaging with a blood flow technique as part of the imaging sequence resolves this issue. Tumors and cysts of the heart can be recognized with most imaging modalities, but frequently both CT and MRI are necessary for full evaluation.

Metastatic tumors of the heart and pericardium are 20 to 40 times more frequent than primary heart tumors. Melanoma, leukemia, and lymphoma frequently are found in the heart at autopsy, and the adjacent lung and breast tumors also are common metastatic deposits. Primary tumors of the heart are rare. In children, rhabdomyomas which constitute 40 percent of tumors in children are frequently seen in the ventricular septum in patients with tuberous sclerosis. In adults, myxomas constitute 25 percent of benign tumors. Angiosarcoma is the most common malignant primary heart tumor with rhabdomyosarcoma and mesothelioma nearly as common. The MRI appearance of myxomas is performed to (1) assess the size, location, and attachment of the tumor, and (2) characterize the tissue as having a signal intensity similar to that of the myocardium. Lipomatous hypertrophy of the interatrial septum is a rather common tumor that presents as a mass on echocardiography and needs to be distinguished from an atrial myxoma. On both CT and MRI, fatty tissue is identified in the interatrial septum with occasional extension of large lobules of fat into both right and left atria. These masses are benign and not associated with obesity, but can produce supraventricular arrhythmias. Arrhythmogenic right ventricular dysplasia can also have fatty replacement in the right ventricle adjacent to the tricuspid annulus, in the free wall, and near the pulmonary outflow tract, but this disease is usually associated with an abnormally thin wall.



Aortic Valve Stenosis and Sclerosis

Calcium is frequently seen in the heart, in the valves, coronary arteries, mitral annulus, and pericardium. Calcified cardiac structures usually signify a pathologic condition, but in older individuals may be a part of the aging process. In the aortic valve region, calcium may be either in the leaflets, the aortic annulus, or the adjacent aorta. The imaging problems that arise are: Where is this calcium and what is its clinical significance?

Aortic valve calcification is best seen on the lateral view of the chest because it projects this calcium free from the spine. Locate the calcium by following the greater curvature of the ascending aorta back to the aortic root. A line drawn from the sternodiaphragmatic angle to the sternum will pass through the aortic valve and distinguish it from a posterior mitral valve calcification.

Calcification occurring only in the aortic valve rarely occurs before the age of 35 unless there has been a previous episode of endocarditis. Isolated calcific aortic disease usually indicates a bicuspid aortic valve. The distinctive bicuspid valve calcification is a ring with a linear bar through it. The aortic valve, unlike the other three valves, has a moderate correlation between the amount of the leaflet calcium and the grading across the valve. The detection of aortic valve calcification of more than a minor degree on the chest film usually indicates the need for aortic valve replacement. Less correlation exists between the quantification of the stenosis when the calcium is seen on a CT scan because the CT is too sensitive to separate out a minor degree of calcification from severe disease.

Aortic valve sclerosis is calcification and thickening of a trileaflet aortic valve without left ventricular outflow obstruction. Aortic valve sclerosis may progress to stenosis. This distinction is best assessed with echocardiography where the thickened leaflets have reduced systolic opening and an increased velocity of greater than 2.5 meters per second across the aortic valve. Although the sequelae of valvular aortic stenosis are well known, including ventricular hypertrophy, occasional coronary artery disease, and rarely sudden death, the natural history of aortic sclerosis without valvular obstruction is also associated with an 50 percent increase in the risk of death from cardiovascular causes and myocardial infarction.

Coronary Atherosclerosis

Coronary atherosclerosis has been imaged for clinical diagnosis for 30 years and for angioplasty for 20 years. Newer imaging modalities are on the horizon to evaluate atherosclerotic plaques non-

invasively, namely, endovascular ultrasound, magnetic resonance angiography, and electron beam CT. Ambrose et al showed that coronary lesions with irregular borders, narrow necks, and clefts were more common in unstable angina compared with lesions that had concentric lumens. An important problem in cardiology is to identify patients that have plaques that are vulnerable to rupture. The key variables appear to be the size of the plaque and the consistency of its lipid core, the thickness of the fibrous cap covering the core, and the inflammatory repair within the cap that initiates the formation of an intimal thrombus. Since lipid-lowering therapy is designed to deplete the amount of lipid core in the plaque, the ability to image the location and size of the plaques is an important problem and currently limits the analysis of much of the therapy in coronary artery disease. Endoluminal ultrasound can image the thickness of the fibrous cap over the atherosclerotic core. Eccentric caps are often thinnest and probably are more prone to rupture, thereby releasing the atherogenic lipid contents and initiate a potentially occlusive thrombus. Examples will be shown of the vulnerable plaque imaged by standard coronary arteriography as compared with endoluminal ultrasound and MRI.

Pericardial Calcification

Constrictive pericarditis, if correctly identified, potentially is a curable disease, but the clinical assessment and intracardiac pressure measurements of pericardial constriction overlap considerably with other types of heart disease, particularly restrictive cardiomyopathy and ischemic heart disease with multiple myocardial infarcts. The pericardial calcification has two types of appearance. The most common type is a shaggy and nodular sheet of calcification around the heart, typically in the atrioventricular grooves. The uncommon appearance is a diffuse eggshell calcification involving most of the cardiac silhouette except for the left atrium which is not covered by pericardium. These calcific deposits represent the endstage of any nonspecific inflammatory process. In the past, infectious agents such as tuberculosis and several viruses, particularly Cocksackievirus, were ascribed to the calcified pericardium, but many other processes such as rheumatic fever, uremia and trauma may all cause local or diffuse calcification. Pericardial stripping may have a dramatic improvement in symptomatology, but pericardectomy may not result in a good long term result, particularly if the pericarditis was caused by radiation. CT imaging is the method of choice to look for the calcification.



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Complications of Cardiothoracic Surgery

Jo-Anne O. Shepard, MD

Introduction

The expected findings following cardiac surgery include atelectasis, pulmonary edema, mediastinal widening, extrapulmonary air collections, increased cardiac size, pericardial changes and chest wall findings. Unexpected complications include mediastinitis, osteomyelitis, post-pericardiotomy syndrome, constrictive pericarditis, aortic pseudoaneurysm and aortic dissection. (Goodman, 1992)

Acute Complications and Findings

Atelectasis

Left lower lobe atelectasis and right lower lobe atelectasis is seen in 90% and 60% respectively of patients following cardiopulmonary bypass surgery. It generally causes little clinical disability. The atelectasis generally progresses from day 1 to day 4 post operative, and then starts to resolve. It is seen as a homogeneous opacity behind the heart often with air bronchograms. (Carter, 1983)

The etiology of the atelectasis is obscure. Proposed mechanisms include adhesive atelectasis and intraoperative hypothermia leading to phrenic nerve paralysis or paresis. Lower lobe atelectasis is more severe when a larger number of grafts are performed, when the pleural space is entered, when cardiac insulating pads are not used and with lower body temperature.

Mediastinal Bleeding

Major postoperative bleeding following median sternotomy requiring re-exploration occurs in up to 5% of patients. Predisposing factors include coagulopathy, anticoagulation, repeat sternotomy, and internal mammary artery grafting. The indications for re-exploration include hemodynamic deterioration or rapid acute widening of the mediastinum. (Goodman, 1992)

Cardiac Tamponade

Cardiac tamponade occurs in 2-3% of patients due to mediastinal hemorrhage immediately following surgery. The site of bleeding is usually at the graft suture line, in the IMA bed or along the sternotomy. In 50% of patients with tamponade the CXR shows an increase in the cardiac silhouette. Free pericardial fluid is best diagnosed by echocardiography. CT is

useful in distinguishing mediastinal from pericardial fluid and identifying pericardial hematomas.

Pneumoperitoneum

Free peritoneal air usually results from inadvertent entry into the peritoneum during opening of the lower sternum. (Glanz, 1978)

Chest Wall Lesions

Acute fractures of the 1st and 2nd ribs are visible on 2-4% of CXRs. (Curtis 1975) Bone scans demonstrate a greater prevalence. (Greenwald, 1973) A lucent stripe up to 3mm can be seen in the sternal split under normal circumstances and is not considered to be a precursor to dehiscence. (Ziter, 1977) The occasional broken sternal wire is not associated with dehiscence without evidence for re-orientation or migration of the sternal wire.

Delayed Complications and Findings

Post Pericardiotomy Syndrome

Post pericardiotomy syndrome presents days to months following the surgery as a febrile illness with a combination of pericarditis, pleuritis, and/or pneumonitis and is associated with elevation of the SED rate, elevated WBC count, and EKG changes of pericarditis. (Ebert, 1990) The pathogenesis is obscure, but may represent an autoimmune reaction. Patients who have had a mediastinal or pericardial hematoma are more likely to develop this complication. CXR will demonstrate an enlarged cardiac silhouette, left pleural effusion and possibly patchy basilar opacities. (Kaminsky, 1982) Although the illness is generally self-limited, it may lead to cardiac tamponade or to constrictive pericarditis. Anti-inflammatory drugs are used for treatment.

Constrictive Pericarditis

Constrictive pericarditis complicates 0.2% of median sternotomies months to years following the surgery. Approximately half of these patients have had a history of prior pericardial bleeding, mediastinal bleeding or post pericardiotomy syndrome. Common symptoms include dyspnea, chest pain, leg swelling, pedal edema, acities, hepatomegaly and jugular



venous distension. In half of the patients with constrictive pericarditis the CXR will demonstrate cardiac enlargement. Other findings include a dilated SVC and rarely pericardial calcification. CT may demonstrate pericardial thickening in the range of 2-5 mm, ascities and pleural effusion in addition to dilatation of the IVC. (Killian, 1989, Kutcher, 1982)

Chest Wall Findings

Median sternotomy is the primary approach to the heart and great vessels. The incidence of complications is less than 5%. Major complications include dehiscence of the sternum, osteomyelitis and mediastinitis.

Normal findings following median sternotomy include small mediastinal air bubbles, indistinctness of the fat planes and small focal fluid or blood collections which tend to resolve by 3-4 weeks after surgery. Minimal pericardial fluid and air bubbles are also seen which tend to resolve within 1-2 weeks. (Goodman, 1983a)

Complications generally become apparent 1-2 weeks after surgery and assume 6 clinical presentations: 1)serosanguineous discharge with stable sternum; 2)unstable sternum with or without discharge; 3)sternal dehiscence alone; 4)superficial wound infection alone; 5)subcutaneous infection, retrosternal infection and unstable sternum; 6)mediastinitis with or without sternal separation. (Serry, 1980)

Dehiscence

Sternal dehiscence is an uncommon but very serious complication of median sternotomy. Isolated sternal wire fracture may be an incidental finding in patients without postoperative complications. However, "wandering wires" describes the characteristic alteration in the appearance of sternal wires on radiographs, ie sternal wires pull out or cut through the sternum rather than break. Displacement of sternal wires is a highly specific sign of dehiscence. (Boiselle, 1999)

Boiselle reported that the mean time between the date of surgery and date of sternal dehiscence was 9 days, (range 4-15d). The CXR demonstrated sternal wire abnormalities in 17/19 (89%) with dehiscence; displacement in 16/19 (85%), rotation in 10/19 (53%), disruption in 4/19 (21%). The mean number of displaced wires was 2.3 (1-5 range). The mean distance of maximal displacement was 20 mm (range 6-45 mm). Sternal wire abnormalities were present before the date of diagnosis in 13/19 (68%).

Sternal wire abnormalities on CXR preceded the clinical detection of dehiscence by a mean of 3 days in 70%. Sternal wire abnormalities were frequently not reported prospectively.

Sternal Wound Infection/ Osteomyelitis

Sternal wound infections occur in 1% of patients

following median sternotomy. The major risk factors include prolonged ventilation, combined coronary and valve surgery, re-exploration for bleeding, low cardiac output and infection elsewhere. Clinical manifestations of sternal wound infection include pain (40%), erythema and drainage (41%), sternal instability (75%), fever (70%), leukocytosis (48%) and positive blood culture (58%). (Grossir, 1985)

Osteomyelitis may occur as an isolated finding or in conjunction with mediastinitis. CT scan will show demineralization of the bone, frank destruction and adjacent inflammation and/or fluid collections. (Templeton, 1992) An early CT scan may not demonstrate convincing bony abnormalities, but followup scanning several days later will generally show bony changes as described when osteomyelitis is present. A Gallium scan will often show increased uptake in addition to the expected linear uptake corresponding to the sternotomy site. (Salit, 1983)

Mediastinitis

Deep postoperative mediastinal infection involving the anterior mediastinal compartment has a reported incidence of 0.4-0.5% with a mortality of 7-80%. It is diagnosed on the average by the 10th to 11th hospital day.

Persistence or progression in indistinct fat planes, mediastinal air and fluid collections suggest infection. In equivocal cases followup CT scans are helpful in identifying resolution or ongoing changes of infection. Carrol et al reported that superior mediastinal widening is present in 63%, pleural effusion in 54%, focal or diffuse mediastinal gas in 22% and focal mediastinal collection in 9%.

The specificity of CT findings is time dependent. Jolles et al reported that all patients with clinical mediastinitis had primary CT findings of mediastinal air, fluid or both. Through day 14, CT had a sensitivity of 100% for clinical mediastinitis and a specificity of 33%. After day 14, the sensitivity was 100% and specificity was 100%.

Vascular Complications

Pseudoaneurysm and Ascending Aortic Dissection

Ascending aortic complications following cardiopulmonary bypass occur in less than 1% of patients. (Goodman, 1983) Complications of the ascending aorta usually occur at the site of cardiopulmonary bypass, site of aortotomy for aortic valve replacement or at the site of aortic anastomosis for coronary artery bypass graft. Acute or chronic infection was found to weaken the suture lines in about half the patients studied by Sullivan. The mean duration from surgery to pseudoaneurysm presentation was 22 months (1-84 months range). Contrast enhanced CT or MRI is

indicated in evaluation of potential pseudoaneurysm or aortic dissection. (Moore, 1984, Thorsen, 1988)

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Objectives

To summarize the major postoperative complications following cardiothoracic surgery and describe the characteristic imaging findings.



Rheumatic Heart Disease

Curtis E. Green, MD

Acute rheumatic fever (ARF) is thought to be a result of an autoimmune type reaction to infection with group A streptococcus. This is fairly firmly established, but based on indirect evidence such as the direct relationship between outbreaks of strep throat and the occurrence of ARF and the fact that the attack rate is directly proportional to the antibody response. Although the incidence of ARF has decreased dramatically in North America and Europe over the past several decades, it remains a leading cause of death in the 5 to 24 year old age group in many parts of the world. The decrease has been attributed to a number of factors including widespread use of antibiotics, decreasing organism virulence and better living conditions. Interestingly, only about half of all patients with proven rheumatic valve disease give a definite history of ARF.

ARF occurs most frequently in children between 2 and 6 years of age, mirroring the incidence of strep throat (40% of all cases) in that age group. It causes a pancarditis with cardiac (CHF, heart block, pericarditis) and non-cardiac (subcutaneous nodules, erythema marginatum, chorea, fever, abdominal pain, epistaxis, pneumonia) manifestations. The primary physiologic valvular abnormality is acute mitral valve regurgitation.

Rheumatic valvular disease is the long-term result of the initial (and any additional) insults to the valve leaflets and support apparatus. The initial episode of inflammatory endocarditis causes formation of granulation tissue which eventually leads to scarring. Anatomically this results in fusion of the valve commissures, shortening and fusion of the chordae tendineae and fibrosis of the valve leaflets. Functionally this can cause stenosis, incompetence or both. It is not certain whether the majority of the damage is caused by the initial episode or whether ongoing inflammation and/or secondary trauma to the damaged valve are the major culprits. The end result is that functionally significant rheumatic valve disease is usually delayed in onset, but progressive. The mitral valve is most commonly affected, being clinically involved in about 90% of patients. Aortic valve involvement is next most common and can be present without clinical mitral disease. Primary tricuspid involvement is uncommon and pulmonary valve involvement is extremely rare. Lupus and rheumatoid arthritis have been reported to cause mitral valve stenosis and, very rarely, heavy calcification of the mitral valve annulus and cal-

cium from the aortic valve stenosis have been known to impede mitral valve opening. There is also a low incidence of congenital stenosis of the mitral valve. The incidence of all of these other causes is so low, however, that from a practical perspective one can equate mitral stenosis with rheumatic heart disease.

Mitral Valve Disease

The mitral valve is the most commonly affected valve in rheumatic heart disease. There is usually a combination of stenosis and regurgitation with the stenosis dominant. The onset of clinical disease is indolent, usually occurring many years after the episode of ARF. Onset of MS at an early age suggests multiple episodes of ARF and is rarely seen in this country.

The normal mitral valve is composed of anterior and posterior leaflets which are continuous across a fibrous annulus. They are supported by corresponding papillary muscles to which they are attached by chordae tendineae. Both leaflets receive chordae from each papillary muscle. Flow across the mitral valve is through both the primary (valve) orifice and through the secondary orifices between the chordae.

The pathologic changes described above result in significant anatomic changes in the heart. Left atrial enlargement is the most common manifestation and can result from pressure overload, volume overload or a combination of the two. The left ventricle is typically normal to small in size unless there is concomitant mitral or aortic regurgitation. Calcium may be present in the left atrial wall or appendage and the mitral. Chronic pulmonary arterial hypertension can lead to right ventricular hypertrophy and eventual right ventricular failure and tricuspid regurgitation which in turn cause right heart enlargement.

Clinically there are four stages through which a patient with mitral stenosis (MS) may pass. Patients with mild stenosis have normal left atrial pressures and are usually asymptomatic. With moderate degrees of stenosis increasing left atrial (LA) pressure causes pulmonary venous hypertension (PVH) and passive (normal pulmonary vascular resistance) pulmonary arterial hypertension (PAH). The primary symptom is dyspnea on exertion. With severe MS muscular hypertrophy causes active pulmonary arterial hypertension with the PA diastolic pressure exceeding the LA pressure. These patients typically have dyspnea at rest. In the final stage of MS, PA hypertension becomes fixed, the right heart fails, tricuspid regurgitation ensues and



cardiac output decreases. Ironically, these patients may actually experience a decrease in dyspnea as the lungs are unloaded. Operative mortality at this point is very high and pulmonary artery pressures may remain elevated after valve replacement. There are two factors which may increase symptoms at any stage of MS: volume overload and tachycardia. The former is one reason why patients may present initially during pregnancy. Tachycardia can occur with exercise or from atrial fibrillation (a common complication of MS) and causes increased LA pressure by decreasing the diastolic filling time of the ventricle.

The radiographic findings in MS are characteristic enough so that the diagnosis can be made with a high degree of confidence in most cases. There is almost always some degree of pulmonary venous hypertension and the left atrium is almost always obviously enlarged. Enlargement of the pulmonary trunk is common and in the presence of PVH strongly suggests MS since LV failure never and non-rheumatic chronic MR rarely cause it to be enlarged. The left atrial appendage may be prominent and if so is a relatively specific marker for rheumatic disease. Absence of an enlarged appendage is not necessarily evidence against MS, however. Calcium in the left atrial wall and mitral valve leaflets is rarely visible on chest radiographs. Visible LA calcium indicates severe, long-standing MS. Interestingly, the correlation between the PCWP and the radiographic appearance of the pulmonary vasculature is as not good as in patients with LV failure. This may be because patients with MS become very efficient at removing fluid from their interstitial space. Also keep in mind that a normal size LA in a patient with PVH and an enlarged pulmonary trunk should make the diagnosis of MS suspect as the prominent trunk may be due to PAH from another cause. In those few cases where the LA is huge and the pulmonary trunk is normal, one is likely dealing with either chronic non-rheumatic MR or the unusual case of rheumatic disease where MR is the dominant lesion. In this case an enlarged atrial appendage will help distinguish the two.

Aortic Valve Disease

The aortic valve is frequently affected, but usually in conjunction with the mitral valve. Combined aortic stenosis (AS) and regurgitation (AR) is the most common combination when there is significant involvement. Pure AS is rare, especially when compared to the most common cause of AS, calcific degeneration. Mild AR, however, is quite common in patients with MS.

Because the aortic valve does not tend to heavily calcify in rheumatic disease, one does not expect to see calcium on the chest radiograph. The ascending

aorta may be dilated, but that sign is both insensitive and nonspecific for aortic stenosis. Similarly there are not any diagnostic plain film findings in AR. Premature tortuosity of the descending aorta, the usual sign of chronic AR, is even less reliable in the setting of MS because the aorta may be displaced by an enlarged LA. Bottom line: rheumatic aortic valve disease is not a plain film diagnosis.

Tricuspid Valve Disease

Rheumatic endocarditis of the tricuspid valve occurs in only 5 to 10% of patients with rheumatic heart disease. Virtually all patients will have coexisting mitral disease. Secondary tricuspid regurgitation, on the other hand, is very common in patients with severe MS and results from RV failure caused by chronic pulmonary arterial hypertension.

The radiographic findings in tricuspid stenosis (TS) or regurgitation (TR) are right heart enlargement and signs of systemic venous hypertension (dilated azygous and venae cavae). Primary tricuspid involvement should be suspected in a patient with MS when the radiograph shows mild PVH, a normal pulmonary trunk and systemic venous hypertension and/or right heart enlargement. This is, however, a rare circumstance. Secondary TR will manifest as right heart enlargement in the setting of moderate to severe MS. If there is any indication of pulmonary arterial hypertension, secondary TR is almost certainly the answer.

Pulmonary Valve Disease

Clinical rheumatic involvement of the pulmonary valve essentially never occurs. A rare patient will develop pulmonary regurgitation secondary to severe chronic pulmonary arterial hypertension, but you will probably go your entire career without seeing a case.

Multivalvular Disease

The patient with rheumatic heart disease may present with any combination of aortic and mitral disease and secondary tricuspid regurgitation. MS plus TR is the easiest combination to recognize and will usually present with PVH, an enlarged pulmonary trunk, LA enlargement and right heart enlargement. Elliott has described this as the “chicken roosting sign” of multivalvular rheumatic disease. Concomitant aortic valve disease will almost always be radiographically silent. One will also not be able to recognize significant MR since the right heart enlargement will preclude evaluation of LV size and the patient might have AR to account for it. Multivalve replacement is almost always due to rheumatic heart disease with an occasional case of multivalve endocarditis. The combination of mitral valve replacement and tricuspid valve



anuloplasty is almost specific for rheumatic disease. One will occasionally see evidence of aortic valve replacement and a left thoracotomy or thoracotomy alone. These suggest open mitral commissurotomy. Although sometimes entertaining, it is rarely useful for the radiologist to attempt to sort out all the lesions: even the best cannot reliably do so!

Caveats

Although the diagnosis of rheumatic mitral valve disease is usually straightforward, there are several factors which may complicate recognition and assessment, namely other causes of an enlargement of the pulmonary trunk and left atrium.

There are a number of non-rheumatic causes of left atrial enlargement. Patients with LV failure can have secondary MR from papillary muscle dysfunction or displacement. Endocarditis and mitral prolapse can both cause MR. When LV compliance is very poor LA pressure may rise and the atrium dilate. A classic example is hypertrophic cardiomyopathy. Lastly, and rarely, there are other causes of obstruction to left ventricular inflow which can mimic MS, the classic example being left atrial myxoma.

An enlarged pulmonary trunk can result from other causes of pulmonary arterial hypertension such as chronic lung disease, idiopathic pulmonary hypertension, vasculitis and recurrent pulmonary embolism. With the exception of lung disease, these may be impossible to diagnose by chest radiograph. Other obstructions proximal to the mitral valve such as LA myxoma cor triatriatum and pulmonary vein stenosis can also cause chronic pulmonary hypertension.

Summary

In the patient with pulmonary venous hypertension, enlargement of the pulmonary trunk is presumptive evidence of mitral stenosis. This should be confirmed by the identification of left atrial enlargement with prominence of the left atrial appendage as further evidence. Absent a prominent pulmonary trunk, mitral valve disease (either stenosis or regurgitation) should be suspected when the left atrium is significantly enlarged, but may not be rheumatic. With a normal or minimally enlarged LA and a normal pulmonary trunk, LV failure is the most likely explanation for the PVH. Aortic valve involvement is in general not a radiographic diagnosis. Tricuspid regurgitation is very common in patients with moderate to severe MS and is usually recognizable because of right heart enlargement.

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Vascular Causes of an Abnormal Mediastinum

Rosita M. Shah, MD

Thomas Jefferson University Hospital

The radiographic diagnosis of vascular mediastinal pathology requires recognition of an abnormal contour or a change in the size of a given vessel. Many embryologic variants and acquired cardiovascular diseases produce characteristic alterations of the mediastinal contours, allowing for radiographic diagnosis.

Normal Mediastinal Reflections

Right mediastinal contours are primarily determined by venous anatomy, including the right brachiocephalic vein, SVC, azygous arch and right atrium.

Left mediastinal contours primarily reflect arterial anatomy, including the left subclavian artery, aortic knob and descending aorta, and the main pulmonary artery. The left superior intercostal vein, recognized as the aortic nipple, is border forming in 1.5% of normal frontal chest radiographs. The left atrial appendage constitutes the third, borderforming cardiac mogul, when enlarged.

Essential Physiology

Vessel size can be considered a function of 2 factors; intraluminal pressure causing dilation and elastic recoil causing constriction. Inherent elasticity of the aorta is greater than the pulmonary arteries, which is greater than that of the pulmonary veins. Increased vessel size can be expected with increased pressure or decreased elasticity. Decreased elasticity is characteristic of diseases producing structural mural changes, such as atherosclerosis or cystic medial necrosis.

Essential Arterial Embryology

Early arterial circulation consists of a ventral aorta and 2 dorsal aortas with 6 paired arches allowing communication between the anterior and posterior circulation. The ventral aorta becomes the truncus arteriosus which divides as the ascending aorta and MPA. The dorsal aortas fuse to become the descending aorta. Portions of the 4th, 5th and 6th arches persist.

Essential Venous Embryology

Early venous circulation consists of paired anterior and posterior cardinal veins, with the right anterior and posterior cardinal veins fusing to form the common cardinal vein, which subsequently contributes to the SVC.

Congenital Arterial Variants

Left Aortic Arch with Aberrant Right Subclavian Artery

The aberrant vessel, always located on the side opposite of the aortic arch, is recognized as an oblique edge or tubular opacity seen through the trachea on frontal chest radiographs, and as a retrotracheal opacity with anterior tracheal displacement on lateral radiographs.

Estimated incidence is 0.5-2.3%. Embryologic basis reflects involution of R 4th arch segment, normally forming the RSCA.

Aneurysmal dilation of the diverticular origin is termed the Ductus of Kommerell.

Right Aortic Arch with Aberrant Left Subclavian Artery

The right aortic arch produces a soft tissue convexity at the right tracheobronchial angle with mass effect on the adjacent trachea. The aberrant vessel on the lateral view produces anterior tracheal displacement.

Estimated incidence is 0.05%. Embryologic origin reflects persistence of the L dorsal aortic root, or ductus segment, with interruption of the L 4th arch proximal to the LSCA.

This variant, with coexistent patent ductus, contributes to a vascular ring.

Right Aortic Arch with Mirror Image Branching

On the lateral view, retrotracheal opacity is NOT associated with tracheal displacement.

Embryologic origin reflects involution of the L 4th arch distal to LSCA.

Congenital heart disease, most commonly tetralogy of Fallot and truncus arteriosus, occurs with a 98% incidence.

Aortic Coarctation

The aortic arch contour is usually ill-defined. The '3' sign reflects dilation of the LSCA, proximal to the stenosis, and the proximal descending aorta, distal to the stenosis. Rib notching and mediastinal widening due to arterial collaterals further support the diagnosis.

Embryologic origin reflects segmental aortic atresia. Adult, or juxtaductal coarctation, is characterized



by a short atretic segment at the ductus. Infantile, or preductal coarctation, is characterized by diffuse atresia.

Associations include bicuspid aortic valve, Turner's syndrome and berry cerebral aneurysms. Preductal coarctation is associated with a 30% incidence of CHD, most commonly, patent ductus arteriosus.

Hypoplastic Pulmonary Artery

The radiographic findings are dominated by an ipsilateral reduction in lung volume, diminutive pulmonary artery contour, and an irregular vascular pattern representing bronchial collaterals.

Embryologic origin reflects involution of the R or L 6th arch.

Associations include partial anomalous venous return, including Scimitar syndrome, and truncus arteriosus III.

Congenital Venous Variants

Azygous Continuation of IVC

An enlarged azygous arch will be evident, without associated tracheal mass effect.

Embryologic origin reflects failure of R subcardinal vein to anastomose with hepatic veins to form the intrahepatic IVC.

Cardiosplenic syndromes and other IVC anomalies may occur.

Persistent Left SVC

A faint straight edge lateral to LSCA and aortic arch may be present.

Embryologic origin reflects persistent L common cardinal vein, allowing venous drainage from the LBCV to coronary sinus.

Associations include cardiosplenic syndromes and ostium secundum atrial septal defects.

Partial Anomalous Pulmonary Venous Return 'LUL'

Radiographic findings mimic a persistent LSVC

Results from failure of normal anastomoses between pulmonary vein and heart, with remnant of left anterior cardinal vein forming the left vertical vein.

Sinus venosus atrial septal defects and polysplenia syndromes may occur.

Acquired Cardiovascular Disease

The diagnosis of acquired cardiovascular disease is facilitated by an understanding of the aorto-pulmonary artery relationship, the vascular pedicle and the effects of aging.

Aorta:Pulmonary Artery Relation

The main pulmonary artery normally projects medial to a line drawn from the aortic knob to the left ventricle. Increases in pressure, volume or turbulent flow can increase the size of the MPA.

Vascular Pedicle

The vascular pedicle, referring to the vascular components of the mediastinum between the thoracic inlet and the heart, varies in size with body habitus, normally measuring between 4.3 and 5.3cm in the upright position. Within a given patient, changes in pedicle width are a strong indicator of changes in circulatory volume.

Effects of Aging

Age related structural changes in the elastic fibers of vessel walls results in dilation and lengthening of the effected vessels. Uncoiling of the ascending aorta causes convexity of the right mediastinum and SVC contour. Uncoiling of the descending aorta produces greater convexity of the left mediastinum and frequently displaces the left paraspinal line laterally. Tortuosity of the innominate and subclavian arteries can produce convexity of the paratracheal soft tissues.

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Imaging of Acquired Aortic Disease

Gautham P. Reddy, MD, MPH

Introduction

Aortography is the conventional imaging technique for assessment of the thoracic aorta. However, helical computed tomography (CT) and magnetic resonance imaging (MRI) have developed into the primary techniques for aortic evaluation. Their advantages over conventional angiography include their non-invasive nature. In addition, tomographic imaging modalities can depict structures such as an intimal flap more readily than a projectional imaging technique such as aortography.

Helical CT angiography has been an important clinical tool for almost ten years. With the advent of multi detector-row helical CT in the past two years, this technique holds great promise for non-invasive vascular imaging.

The utility of MRI for evaluation of the thoracic aorta was recognized early in the development of cardiovascular MRI. In recent years gadolinium-enhanced three-dimensional (3D) magnetic resonance angiography (MRA) has also proved useful for depiction of aortic abnormalities.

Clinical applications of these techniques in the thoracic aorta include the evaluation of aneurysm, dissection, and pseudoaneurysm.

CT Techniques

When evaluating a patient with suspected aortic dissection, it is helpful to obtain a sequence before administration of contrast agent, because this scan is more sensitive for intramural hematoma. All patients should have a scan after contrast administration. Volumetric reconstruction of CT angiography can be important, especially for evaluation of aortic aneurysm or pseudoaneurysm. These reconstructions are less important for the evaluation of dissection.

Sample Protocol for Helical CT

Unenhanced

Pitch 2

5 mm — reconstruct at 5 mm

Enhanced

Injection 150 cc at 2.5 cc/sec

Scan Delay 35 sec

Pitch 2

5 mm —reconstruct at 2.5 mm

Sample Protocol for Multi Detector-row

Helical CT

Unenhanced

Pitch 3

5 mm — reconstruct at 5mm

Enhanced

Injection 150 cc at 3.5 cc/sec

Scan Delay 30 sec

Pitch 6

2.5 mm — reconstruct at 2.5 mm

MRI Techniques

The thoracic aorta is optimally evaluated with several MRI techniques, including spin-echo (SE) MRI, gadolinium-enhanced 3D MRA, and cine MRI.

Spin-Echo MRI

SE MRI of the aorta requires electrocardiographic (ECG) gating to overcome artifact of cardiac motion and arterial pulsation. Respiratory compensation reduces artifact due to movement of the chest during breathing.

The multislice ECG-gated SE MRI technique is ideal for depiction of anatomy, intramural hematoma, and periaortic pathology. TR is equal to the length of one heartbeat (typically 700 to 1100 msec) and TE is 20 msec, providing approximate T1 weighting. A sagittal sequence is generally acquired first. Transverse images from the aortic arch to the cardiac apex usually provide the maximum diagnostic information.

Gadolinium-enhanced 3D MRA

Gadolinium-enhanced 3D SGPR MRA yields excellent vascular contrast that does not depend on blood flow. Instead, 3D MRA relies on the T1-shortening effect of gadolinium, which increases the signal intensity of the blood. This technique is performed with bolus injection of gadolinium chelate contrast agent. For simplicity, a standard dose of contrast media may be used in adults. Typically, 40 cc (20 mmol) of gadolinium is administered intravenously at a rate of 2 cc / second.

Scan delay after contrast material injection varies from 5 to 7 seconds (for pulmonary arteries) to 25 seconds or more (for abdominal vessels). For the thoracic aorta, the delay is approximately 10 seconds when using sequential K space acquisition, or 20 to 25 seconds for centric K space acquisition. Scan timing can be optimized with bolus-detection software or by the use of a test bolus. Two or three sequential scans are acquired in the sagittal plane for the thoracic aorta, each during a 20 to 30 second breath hold (TR 4-7, TE 1-2, flip angle 45). Each acquisition



consists of 28 to 40 slices with a slice thickness of 2 to 3 mm.

After the examination, the MRA data can be viewed on a 3D computer workstation. Maximum intensity projection (MIP) reconstructions in multiple planes can be invaluable for interpretation of the study.

Cine MRI

In most cases, 3D MRA should be supplemented by an axial sequence for optimal assessment of the vessels. Patients with suspected aortic dissection should undergo SE T1-weighted and / or GRE cine imaging for evaluation of intramural hematoma and periaortic abnormalities.

Cine MRI is composed of multiple GRE images obtained at different phases of the cardiac cycle. Cine MRI can be used to evaluate ventricular contraction. Breath hold (“segmented K space”) cine MRI (TR 33, TE 5 to 6, flip angle 20) is an excellent technique for visualization of vessels. Slice thickness is typically 10 mm. Because it is flow-sensitive, cine MRI does not require the use of contrast media. However, when this technique is used in conjunction with 3D MRA, it should be performed after the angiographic sequence to take advantage of improved contrast resolution after gadolinium administration.

Sample MRI Protocol

- ECG gating, Respiratory compensation
- Phased-array surface coil
- 1. Sagittal SE T1-weighted (TR = gated every beat, TE = 20); 256 x 192
8 mm skip 2; 2 excitations
- 2. Axial SE T1-weighted (TR = gated every beat, TE = 20); 256 x 192
5 mm skip 1; 2 excitations
- 3. Sagittal or Oblique Sagittal 3D SPGR MRA (TR = 4 to 7, TE = min (1 to 2), flip angle = 45); 256 x 160 or 512 x 192
2 to 3 mm, 28 to 40 slices (“partitions”); 0.5 to 1 excitation
40 cc (20 mmol) IV gadolinium chelate contrast agent at 2 cc / second
Scan delay 10 seconds
- 4. Axial breath hold GRE cine (TR = 33, TE = minimum (5 to 6), flip angle = 20); 256 x 128; Flow comp
10 mm skip 0; 1 excitation

Clinical Applications

Aneurysm

An aneurysm of the thoracic aorta is at increased risk for rupture if its diameter is 6 cm or greater. Thoracic aortic aneurysms may be secondary to atherosclerosis, infection, or cystic medial necrosis (annuloaortic ectasia).

For evaluation of the thoracic aorta, CT and MRI can be more accurate than x-ray angiography. X-ray angiography depicts only the opacified lumen of the vessel, whereas CT and MRI demonstrate the entire diameter of the aorta, including the mural plaque. In addition, the relationships of branch vessels are more readily defined on cross-sectional imaging, due to overlap of structures on a projectional technique such as x-ray angiography.

Helical CT angiography is useful for identifying the size of the aorta and for assessing the extent of an aneurysm. The CT angiography can be reconstructed using one of several techniques, such as multiplanar reformations, maximum intensity projections, shaded surface displays, or volume rendering. Although these reformations are not usually required for diagnosis, they can be invaluable when communicating with the referring physician.

Conventional MRI has been shown to be accurate for evaluation of thoracic aortic aneurysm; the aneurysm location and extent can be depicted readily. The addition of contrast-enhanced MRA to SE MRI can improve the demonstration of the arch vessel morphology and the relationship of the great vessels to the aneurysm. When the aneurysmal aorta is tortuous, gadolinium-enhanced MRA may provide essential information because the true dimensions may be difficult to ascertain by SE MRI. Cine MRI and 3D MRA can be used to differentiate slow blood flow from thrombus. With use of 3D MRA, both atherosclerotic aneurysms and annuloaortic ectasia can be defined with precision. In patients who have annuloaortic ectasia, aortic regurgitation can be evaluated with cine MRI.

Pseudoaneurysm

Causes of pseudoaneurysm in the thoracic aorta include trauma, penetrating atherosclerotic ulcer, and infection such as endocarditis, which can result in a pseudoaneurysm of the aortic root. Pseudoaneurysm can also occur after aortic surgery, as mentioned previously. The characteristic appearance of a pseudoaneurysm is a focal dilatation of the aorta with a connection to the aortic lumen via a narrow neck.

Both helical CT and MRA can be used to image patients with pseudoaneurysm. Although SE MRI can identify a pseudoaneurysm, 3D MRA may provide higher resolution and improved accuracy.

Dissection

When imaging a patient with suspected dissection, it is crucial to identify the intimal flap and to determine whether the dissection involves the ascending aorta (Stanford type A) or if it is limited to the descending aorta (Stanford type B). Most patients with a type A dissection must undergo surgical repair,



whereas most patients with a type B dissection can be managed medically.

Helical CT and MRI both have sensitivities and specificities of 95% or greater for evaluation of thoracic aortic dissection. Choice of imaging modality depends on the availability of the scanner, the radiologist's familiarity with the techniques, and the preference of the referring physician. In our institution, CT is usually performed when acute dissection is suspected. MRI is reserved for patients who have a contraindication to iodinated contrast medium, patients in whom the CT scan does not provide definitive assessment of arch vessel involvement, and for patients with chronic dissection.

It should be noted that transesophageal echocardiography (TEE) has high sensitivity for dissection, but variable specificity, reported as low as 67-77%. For this reason, it may be prudent to perform CT or MRI instead of TEE unless the patient is unable to be moved out of the emergency room or intensive care unit.

Helical CT is well established as a means of evaluating aortic dissection. However, intramural hematoma can be overlooked if a non-contrast scan is not performed. Intramural hematoma can be recognized as thickened aortic wall of relatively high attenuation on unenhanced CT.

The accuracy of MRI for evaluation of thoracic aortic dissection is well established. However, at least one paper has suggested that SE MRI may be limited in assessment of the arch vessels. More recently, some studies have shown the efficacy of contrast-enhanced 3D MRA for depiction of aortic dissection. The use of 3D MRA appears to improve the detection of involvement of the arch vessels. An important limitation of gadolinium-enhanced MRA is its relatively low sensitivity for intramural hematoma. Thus, a SE and / or cine acquisition should be obtained in conjunction with 3D MRA when evaluating a patient with suspected aortic dissection.

MRI and MRA can be used in the routine long-term follow-up of patients who have undergone repair of a type A aortic dissection. Complications in these patients, such as aortic aneurysm or pseudoaneurysm, can be detected and surgically treated if necessary.

Conclusion

Helical CT and MRI have proved their utility for a number of applications in the thoracic aorta, including evaluation of aneurysm, aortic dissection, and pseudoaneurysm. Advantages of CT and MRI are their noninvasiveness, and their tomographic nature, which allows delineation of structures that can overlap on projectional techniques such as x-ray angiography.

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Traumatic Aortic Arch Injury: To Scan or to Squirt?

Joseph H. Tashjian, MD

Objectives

- 1) To describe a technique that will allow for the direct diagnosis of traumatic aortic injury in the vast proportion of cases.
- 2) To demonstrate the ability of CT to evaluate for traumatic aortic injury, both with direct and indirect signs, allowing CT to become the first and possibly last method of diagnosis in this disease.

Background

Acute injury to the thoracic aorta has been, and continues to be a diagnostic dilemma. It accounts for 10–20% of fatalities in high-speed deceleration accidents, with death at the scene of the accident in 80–90%. The patients that arrive at the hospital have a high mortality, with death occurring in 50% within 24 hours, and 90% within 4 days. Posttraumatic chronic aneurysms have been found to occur in 2–5%.

Prompt diagnosis and surgical repair are necessary to properly treat these surviving individuals. Successful surgical repair is dependent on diagnosis, and varies with the degree of instability, associated injuries, and degree of laceration. Postoperative paraplegia occurs in up to one quarter of patients, related to spinal cord ischemia.

Pathologically, the injury is manifest by transverse laceration of the wall of the aorta, which may be as small as one millimeter, or as large as to be completely circumferential. It may involve only the intima, or may extend through the media and adventitia; however, the adventitia is intact in 60% of cases. In a few cases, it will only involve the brachiocephalic vessels.

There have been several mechanisms proposed to cause the injury. The most common one has been rapid deceleration, causing a shearing action at the aortic isthmus, where the aorta changes from a relatively fixed portion, to a more mobile portion. An analogy for this mechanism would be similar to a cherry (the chest) being squeezed, and the pit (heart) being displaced. Because the aorta is fixed at the isthmus, the tear occurs in that location.

A second hypothesis relates to hydrostatic forces and a “water-hammer” effect from direct compression of the aorta. Intraluminal pressure may exceed 2500 mm Hg, which when coupled with the shearing

and stretching forces may explain the transverse tear, as well as the location at the aortic isthmus.

The third proposed mechanism is the “osseous pinch”, described by Cohen and Crass, refers to pinching of the aorta between the manubrium and thoracic spine. Cadaver work demonstrated that during compression, the manubrium frequently is directly in contact with the thoracic spine, resulting in the aorta being lacerated as it is compressed by these bony structures.

Screening

As the injury almost invariably leads to death, the purpose of screening is to identify those patients that need repair, while not subjecting those patients who do not have injury to further diagnostic work-up or surgery. The four most common methods of screening are history, portable chest radiography, conventional aortography, and computed tomography (CT).

History, and mechanism of injury, are important in identifying those patients that may be at risk. Mechanisms include fall from a height of 10 feet, high-speed deceleration >30 mph, unbelted driver or passenger, or struck pedestrian. Clinical signs include paresis or paralysis, hypotension, chest pain, and decreased or absent extremity pulses.

The portable chest has been, and will continue to be, the first method for radiographic evaluation. Unfortunately, it is a relatively poor method, both due to the technique of the exam, and a significant lack of specificity. Typical signs include widening of the mediastinum, indistinct aortic knob, deviated trachea or nasogastric tube, fractured first or second rib, and/or apical pleural cap.

Aortography remains the standard to which all other modalities are compared. It is both sensitive and specific, with both approaching 98–100%. Although cases of a false negative angiogram are rare, they do exist, even in retrospect. In addition, in older individuals with plaque formation, aortography may have difficulty distinguishing plaque from intimal injury. Aortography is invasive; however, its main drawback is that it takes 30–60 minutes to assemble a team to perform the procedure, and time is critical in the management of these cases.

CT was initially used as an indirect method to evaluate for mediastinal hemorrhage, as a means to



determine which patients would need aortography. Although mediastinal hemorrhage is non-specific, and may be due to venous injury, sternal or vertebral fracture, or injury to the thymus gland, the lack of a mediastinal hematoma essentially excludes the diagnosis of aortic injury. As more experience has been gained, and with improvement in technique and scanner capabilities, the direct diagnosis of aortic injury can frequently be made from the CT scan, obviating the need for aortography.

Technique of CT Aortography

The technique of CT aortography is completely dependent on the ability of the scanner available, and the radiologist. Helical CT has greatly aided in evaluation; however, many if not most, authors fail to take full advantage of the helical nature of the examination. Knowledge of contrast injection rates, pitch, slice thickness and slice overlap are essential if one is to make a direct diagnosis. 2-D and 3-D reformations are occasionally useful, especially to the surgeon, but only if they can be rapidly performed. Otherwise they just make pretty pictures for presentations or publications.

Contrast Injection

In any type of angiography with CT, it is important to inject at a rapid rate, and time the scan with the peak of the contrast injection. Injection rates of 1-2 cc's/second are inadequate if one wishes to obtain a CT aortogram and make a direct diagnosis of aortic injury. Injection rates need to be at least 3 cc's/second, and preferably 4 cc's/second. Peripheral injection sites in the hand or foot is inadequate as well. One needs a minimum of an 18 gauge IV either in the antecubital fossa, or groin. Central lines are preferred if they are of adequate size. In these patients, this rarely is a problem, as at least two or three 18 to 16 gauge lines have already been placed in the Emergency Department in the vast majority of cases.

The injection delay is also important. Fortunately, timing in the ascending and descending aorta in these patients is relatively constant. Most of them are tachycardic, and have a high cardiac output. When injecting at 4 cc's/second, a 20 second delay is appropriate, as 80 cc's will have already been injected, and will allow for continuous contrast injection while scanning.

The amount of contrast is important to allow for continuous scanning while the contrast is being injected. Typically, these patients will also need examination of the abdomen and pelvis. We inject 150 cc's of contrast that has been diluted with 50 cc's of saline. The saline dilution is to help prevent artifact from straight contrast from the superior vena cava, and allow for a little longer injection

time (a total of 50 seconds), especially since we are injecting at 4 cc's/second.

Pitch, Slice Thickness, and Slice Overlap

One of the most misunderstood concepts in helical CT is pitch, and indirectly, slice thickness. Pitch is defined as the number of slice thicknesses the table advances per tube rotation. A 1:1 pitch means the table advances one slice thickness per tube rotation. A 2:1 pitch means the table advances two slice thicknesses per tube rotation. For example, in a 1:1 pitch, with a slice thickness of 10 mm, the table advances 10 mm per tube rotation. For a 2:1 pitch, with a slice thickness of 10 mm, the table advances 20 mm per tube rotation. The advantage of increasing pitch is that you can cover a larger area for the same number of tube rotations. The disadvantage is that as you increase pitch, there is some decrease in resolution, due to broadening of the slice.

It however, is almost always better to increase pitch and decrease slice thickness. For example, one can use a 1:1 pitch, 5 mm slice thickness and cover an area of 20 mm with four tube rotations. The resulting four slices are 5 mm thick. One can also use a 1.67:1 pitch, 3 mm slice thickness and cover an area of 20 mm with four tube rotations. The time for each scan is the same. The resulting slice thickness, even including the slice broadening, is about 3.5 mm, and there are almost 7 slices. This results in an improvement of almost 50 % in slice thickness resolution, with more slices, for the same amount of tube rotations and time.

Slice overlap is how you want your images to overlap. Since the data is continuous, the amount of overlap is merely a function of computing power. It does not cause any increase in radiation to the patient, nor does it take any longer in terms of scan time. It does take longer in terms of reconstruction time, depending on how many images you want to overlap.

When looking for direct signs of aortic injury, the thinner the slice, the more likely you are going to be able to see intimal tears. In addition, the more slices you reconstruct, the more likely you are to be "centered" on the tear, and more likely you are to visualize it. Currently we are using a 3 mm slice thickness, reconstructed every 1.5 mm, with a 1.6:1 or 1.8:1 pitch, depending on the patient's size. We begin at the top of the lung apices, to include the great vessels, and continue to the aortic root. We then switch to 7mm images to the diaphragm. Usually we need to program in a delay, to allow the contrast to properly enhance the liver and spleen, before proceeding through the abdomen and pelvis to look for major organ injury.

To Scan or to Squirt?

The diagnostic dilemma regarding CT for evaluation of aortic injury has focused on whether CT is 100% sensitive for detecting aortic injury, as a missed diagnosis usually results in death. In addition, delay in diagnosis may also lead to death. These two factors have lead some to believe that CT is not appropriate for evaluation.

An initial study by McLean et al. demonstrated two false-negative examinations; however, these studies were performed at outside institutions with no mention of technique or criteria for interpretation. In another study by Miller et. al., there were five false-negative examinations. In this study the amount of contrast was only 75 cc's, and the slice thickness was 10 mm. In addition, one study was performed without contrast, and another was significantly degraded by motion, and a third was misinterpreted. These studies were not performed by radiologists.

A more recent study has shown CT to be quite effective in evaluation of this injury. Dyer et al. have demonstrated that over a 4½ year period of time, involving 802 patients examined with CT, there were 10 true-positive cases, and no false-negative cases. This resulted in a 100% sensitivity, and 100% negative predictive value. Their specificity was 62%, and positive predictive value of 7%. In this study they looked for direct signs of aortic injury, such as intimal disruption, rather than solely relying on periaortic or mediastinal hematoma. After the completion of the study, they did have one case that did not have a demonstrable intimal tear on CT, but did have a periaortic hematoma. Interestingly, on aortogram, a small pseudoaneurysm was present without an intimal tear. A sagittal oblique reconstruction of the CT scan probably would have demonstrated the same finding.

Areas of difficulty in interpretation include older patients with atherosclerosis, and evaluation of

branch vessel injuries. Atherosclerosis in the region of the ductus arteriosus presents difficulty for both aortography and CT. The ability to evaluate for a periaortic hematoma can be helpful in distinction from intimal tear. Branch vessel injuries have not been formally studied; however, we have seen tears of the subclavian arteries with the technique described.

Conclusions:

- 1) CT is able to exclude aortic injury if no mediastinal hematoma surrounding the aorta is present, and no intimal tear is identified.
- 2) If a periaortic hematoma is identified, or if there are equivocal signs of intimal tear, aortography is indicated.
- 3) If an intimal tear is identified, aortography or surgery should be performed, depending on the institution, depiction of the injury, and confidence in the diagnosis. The ability to make a direct diagnosis and proceed to surgery is an important advance.

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The Multimodality Diagnosis of Pericardial Disease

Robert M. Steiner, MD

Objectives of the Lecture

At the completion of this lecture those in attendance will be able to describe the normal and pathologic anatomy of the pericardium utilizing multimodality imaging. The value of echocardiology, plain film radiology, CT and MRI for the diagnosis of pericardial disease, will be discussed. The limited manifestations of disease affecting the pericardium including pericardial effusion, pericardial thickening, hemopericardium and pneumopericardium will be characterized.

Introduction

The importance and frequency of pericardial disease closely parallels the frequency of cardiac and non-cardiac thoracic surgery, irradiation and the use of a wide variety of therapeutic agents which effect the pericardium. The diagnosis of acute and chronic pericardial disease has increased significantly in tandem with the increasing sophistication of diagnostic imaging technology.

Physiology

The pericardium aids in maintaining ventricular compliance when end-diastolic ventricular pressures are elevated and it prevents sudden chamber dilatation due to hypervolemia. The pericardium also assists atrial filling during ventricular systole as negative pressure develops within the pericardium during ventricular ejection.^{1,2} Twenty-25 ml of lymphoid fluid is normally found in the pericardial space. The presence of the fluid helps to diminish friction between the heart and adjoining structures.

Anatomy

The pericardium is a double layered fibroserosal envelope which surrounds the heart, great vessels and the pulmonary veins. The pericardium consists of an outer fibrous layer, consisting of a dense collagen network and an inner serous layer. The inner serous layer is referred to as the epicardium and is a glistening membrane attached to the surface of the heart. The adventitia of the great vessels blend seamlessly with the serous pericardium to become the parietal pericardium. The parietal pericardium attaches anteriorly to the pericardial sternal ligament, inferiorly to the central tendon of the diaphragm and posteriorly to the esophagus and descending aorta.^{1,3} The inferior

vena cava enters the pericardium through the central tendon of the diaphragm and is not covered by a fibrous pericardial layer.² The visceral pericardium is fused to the heart, and forms a complex network of sinuses and recesses. Familiarity with this complex anatomy is important when interpreting CT or MRI studies,^{2,4-8} since the fluid within the pericardial can stimulate soft tissue masses such as the the thymus, aortic aneurysm or lymphadenopathy. It appears that the pericardial sac that covers the heart and vessels is organized in the form of two (2) tubes. The aorta and main pulmonary artery are enclosed within an anterior-superior pericardial tube and the vena cava and the pulmonary veins are enclosed within an inferior-posterior tube. The communicating passageway between these tubal structures is the transverse sinus.^{2,3,9} Diverticula extending outward from the transverse sinus lie between great vessels and the atria.^{6,9} These include the right and left pulmonary recesses and superior-inferior aortic recesses. The oblique sinus lies posterior to the left atrium and medial to the superior vena cava. It is separated from the transverse sinus by a double layer of serous pericardium containing the left and right superior pulmonary veins. Other recesses include the posterior caval recess and the pulmonary venous recess which lie between the superior and inferior pulmonary veins.⁹

Imaging the Normal Pericardium

Plain Chest Radiograph

In the frontal projection, the normal pericardium is only occasionally seen as a distinct structure because it lies close to the heart and is of similar attenuation value to the heart. Sometimes a normal pericardial stripe is seen along the left heart border because of abundant fat surrounding the pericardium.¹⁰ In the lateral view, fat in front of the pericardium [anterior-mediastinal fat] and fat behind the pericardium [subepicardial fat] allows one to see the normal pericardial stripe as a curvilinear opacity 1-2mm in thickness.^{2,10} The thickness of the pericardial stripe consists of the width of the fibrous sac together with the 25-50 ml of fluid in the normal pericardial space.

Computed Tomography

The normal pericardium is invariably identified on CT imaging because of the superior contrast resolution



of CT in the cross-sectional imaging format. The pre-ventricular pericardium is most often visualized because of the abundant retrosternal epicardial fat at that level.^{11,12} Laterally the pericardium may not be visible on CT because of motion blurring and pixel misregistration.¹ On cross-sectional imaging recesses of the pericardium may be normally seen containing small amounts of fluid. The transverse sinus is invariably identified as a half moon-shaped structure behind the ascending aorta.

CT can detect small amounts of pericardial effusion because of the differential X-ray absorption coefficients of pericardial fluid and the pericardium itself. In fact, CT has a lower false positive rate than ECHO because of the better resolution of boundaries between the heart, pleura, pericardium and other mediastinal structures.

It is possible that CT will identify and differentiate exudate, chylous and transudative fluid because of differences in attenuation coefficients of these three densities. Conversely, hemopericardium is often difficult to characterize because of its similar density with the myocardium.

Echocardiography

The diagnosis of pericardial disease is most commonly made by echocardiography, the imaging modality of choice for detecting pericardial effusion with high sensitivity and specificity. One of the major advantages of ECHO over CT and MRI and plain film radiography is its ability to be performed at the bedside in critically ill patients. On M-mode echocardiography an echo-free space between the parietal and visceral pericardium is identified throughout the cardiac cycle is the most valuable diagnostic feature. Transesophageal echocardiography has the advantage of showing the distribution of the effusion and has the ability to distinguish other causes of echo-free space such as pleural effusion, the coronary sinus and the descending thoracic aorta.¹³ An echo-free space located posteriorly less than 1 cm in greatest width is considered a small effusion while pleural effusion in both the anterior and posterior echo-free space but less than 1 cm in width is considered moderate. A large pericardial effusion is a echo-free space that surrounds the heart for a distance of at least 1 cm.

Loculated pericardial effusions occurring after surgery or pericarditis or diffuse neoplasm may not be easily appreciated with conventional echocardiography and transesophageal echocardiography may be necessary to adequately image the effusion. CT and MRI are also helpful in such circumstances.¹⁴

Magnetic Resonance Imaging

The pericardium is clearly seen as a low signal in-

tensity structure surrounding the pericardium on T₁-weighted images. The difference between the high intensity fat and the lower intensity pericardium is the source for the high contrast resolution between the two structures.^{15,16}

With MRI, pericardial effusion can be detected with high sensitivity and fluid collections as small as 30 mm can be visualized. Loculated effusion, pericardial thickening and separation of myocardium from pericardial tissue because of pericardial fat is particularly well-seen with MRI. MRI has the capability of characterizing the nature of pericardial effusion and has been known to distinguish hemorrhagic from exudative effusions as well as exudative from transudative effusions in at least one experimental canine model.¹⁷

Specific Pericardial Disorders

Pericardial Effusion (Table I)

Fluid in the pericardium space is regulated by the relative differences in osmotic and hydrostatic pressure between the pericardial space and the pericardial capillaries related to the degree of permeability of the serous pericardium and the clearance of fluid by lymphatics and venous drainage. The ability to absorb pericardial fluid may be compromised by epicardial lymphatic occlusion and blockage of the venous drainage pathways of inflammation, fibrosis or neoplasm.¹

Plain Film Findings

The radiographic appearance of the cardiac silhouette in a patient with pericardial effusion may be normal with small effusions but with larger effusions the

TABLE 1
Causes of Acute and Chronic Pericarditis Resulting in Pericardial Effusion

1. Myocardial infarction with left ventricular failure.
2. Right-sided heart failure
3. Pulmonary hypertension
4. Aortic Dissection
5. Post-pericardotomy syndrome
6. Viral infection (including Coxsackie virus, echo virus)
7. Tuberculous pericarditis, especially in patients with acquired immune deficiency syndrome (AIDS)
8. Bacterial infection (including staphylococcal infection, hemophilus influenza)
9. Infective endocarditis with extension to the myocardium
10. Tumor of the Pericardium: Primary and metastatic
11. Pericardial-esophageal or bronchial fistula from carcinoma of the lung and esophagus
12. Collagen Vascular Disease (including Lupus erythromatosus and scleroderma)
13. Uremia
14. Blunt Thoracic Trauma
15. Surgery



cardiac silhouette is triangular, globular or flask shaped in configuration with effacement of the normal indentations of the heart border.¹ There is encroachment the retrosternal clear space on the lateral projection and overlay of the hila in the frontal projection by the enlarged pericardial sac. Cardiomyopathy may also cause diffuse dilatation but the hilar shadows are displaced away from the midline and there is no hilum overlay.

With pericardial effusion a wide pericardial stripe or “positive pericardial fat pad sign” may be seen on the lateral projection if the pericardium thickened beyond 2 mm between the epicardial and anterior mediastinal fat. Although this sign is reliable it is present in less than half of patients with significant pericardial effusion.¹⁰ A retrosternal extrapericardial hematoma or other sub-sternal collection may mimic pericardial effusion leading to a false positive diagnosis.¹⁷

Echocardiography is the preferred screening study for the presence or absence of pericardial effusion. A small effusion may appear as an echo-free space between the left lung and the posterior left ventricular free wall. A larger effusion may be found between the anterior wall of the right ventricle and the chest wall. Sometimes, a false positive diagnosis of pericardial effusion is due to hypoechoic mediastinal fat which may simulate a loculated effusion.¹⁴ Pleural effusion, atelectasis and pneumonia may also be mistaken for a posterior pericardial effusion. If a loculated or complex pericardial effusion is inaccessible to the ultrasound beam it may not be identified at all.¹⁸

CT will resolve many of the false positive or negative diagnoses made with ECHO because of its superior contrast resolution and cross-sectional format. For example, anterior mediastinal fat is easily distinguished from pericardial fluid often not possible with echocardiography.^{14,18} Pericardial calcifications are also clearly visible with CT but not with echocardiography.

MRI, particularly with T₁ weighted images, will show a low signal intensity around the cardiac chambers. Pericardial fluid will appear as a bright signal intensity zone on T₂ weighted spin ECHO and gradient refocused echo pulse sequences. The multiplanar ability of MRI also makes it possible to see small loculated and other obscure pericardial effusions. The high signal found in pericardial fluid will help to distinguish edema fluid from pericardial thickening.¹⁹

Cardiac tamponade is defined as hemodynamically significant compression of the heart causing equalization of intrapericardial and ventricular diastolic pressures resulting in impaired diastolic ventricular filling and increased atrial pressure. Enlargement of the cardiac silhouette as seen with chest radiography in patients with cardiac tamponade is usually not apparent until about 250 cc fluid is present in the pericardial

space. If the fluid accumulates slowly several liters of fluid may be present without tamponade occurring clinically.

Cardiac tamponade does not only occur with fluid accumulation. Air within the pericardial space can also result in tamponade due to tension pneumopericardium.²⁰ This phenomenon may occur in children through assisted ventilation or in patients who have undergone penetrating chest trauma or fistulas from an adjacent bronchus, esophagus or stomach.^{20,21}

If the accumulation in the pericardial cavity is rapid, cardiac tamponade may occur in face of a normal heart size. In patients with pericardial tamponade the lungs are generally normal without pulmonary edema. Clues to the diagnosis of tamponade by cross-sectional imaging include dilation of the azygous vein, reflux of contrast into the coronary sinus because of high right-sided pressures, pronounced changes of the size and shape of the superior vena cava in systole in diastole and straightened or uneven concavity of the right heart border. The right ventricle will be depressed and concave and there may also be tapered narrowing of the left ventricle.^{1,22-25}

Constrictive Pericarditis

Due to viral and tuberculous pericarditis, uremic pericarditis, hemopericardium and following surgery or trauma. The pericardial space is obliterated by dense fibrous tissue. Calcification of the pericardium is a sequella of pericardial fibrosis. Pericardial effusion and pericardial thickening may coexist. Calcification when present is most commonly found in the region of the atrioventricular groove where abundant normal fat is usually found. This fat provides the additional contrast allowing for the calcium to be clearly seen.^{1,2,16} On occasion the focal pericardial fibrosis will obstruct the atrioventricular valves, the superior and inferior vena cava or even the right ventricular outflow tract. Ascites, hepatomegaly, with evidence of “cardiac cirrhosis” may be found.

On plain film radiographs, the silhouette is usually normally or slightly enlarged when coexistent pericardial effusion is present. Pericardial calcification may be thin or thick, patchy or continuous and are most often found in the atrioventricular groove and along the diaphragmatic surface of the right ventricle. It is present in 30-50% of patient with constriction and is best seen on lateral radiographs. If constriction is primarily right sided, azygous vein dilatation and widening of the cavae may be observed. If constriction predominantly effects the left heart, left atria enlargement and pulmonary venous hypertension may be present.

Echocardiography

With echocardiography pericardial thickening may be difficult to separate from adjacent pericardium.



However, associated pericardial effusion and the size and function of the surrounding ventricles can be established. Mitral stenosis can be differentiated from constriction when the plain film findings are consistent with pulmonary venous hypertension.

Computed Tomography

The normal thickness of the pericardium is 1-2 mm. Patchy areas of thickening also may normally be found in 1/3 of patient(s). In constrictive pericarditis the thickness of the pericardium may be as great as 20 mm. With CT, abnormal left ventricular filling, pericardial calcification, loculated diffusion, deviation of the interventricular septum and inferior vena cava dilation are common findings.

Magnetic Resonance Imaging

There is superior delineation of intercardiac and pericardial structures with MRI. Thickening of the pericardium greater than 4 mm has been found in most patients with confirmed constrictive pericarditis.¹⁴ A small tubular shaped ventricle is an additional sign of pericarditis.

Comparison with Restrictive Cardiomyopathy (Table 2)

Constrictive pericarditis may be difficult to distinguish from restrictive cardiomyopathy based simply on hemodynamics. Calcification of the pericardium is present in many patients with constrictive pericarditis but does not occur in restrictive cardiomyopathy. With constriction coronary artery motion is decreased in constrictive pericarditis but not in restrictive cardiomyopathy and the location of the coronary artery in relation to the mediastinal silhouette shows a degree of separation with constriction but not with restriction.

Pericardial Surgery

Pericardial surgical transection most often occurs in conjunction with a coronary artery bypass graft or valve surgery. Partial resection or fenestration is usually performed with these procedures. Fenestrations are usually small retrosternal incisions that heal quickly following

TABLE 2
Constrictive Pericarditis and Restrictive Cardiomyopathy*

Radiologic Finding	Constrictive Pericarditis	Restrictive Cardiomyopathy
Pericardial calcification	Present (30-50%)	Absent
Right atrial wall motion	Normal	Restricted
Right atrial wall border	Straight or Concave	Convex
Coronary artery motion	Decreased	Normal

*Modified from reference 1

surgery. When sternal separation occurs the pericardial communication may remain open or even reopen. Primary pericardial procedures include pericardiocentesis, pericardiotomy for abscess drainage and relief of cardiac tamponade. Pericardial pneumonectomy is infrequently performed but is the procedure of choice for radical treatment for diffuse mesothelioma.

Complications of cardiac surgery include **hemo-pericardium**. Occuring within the first 48 hours after surgery anticipated by the cardiac silhouette enlargement. Pericardial postoperative effusion usually obtains maximum size by the tenth day but some effusion is present between days 1-5.

Postpericardotomy Syndrome is usually related to excision of the pericardium but may also occur following trauma or minor surgical procedures. It occurs in 10-40% of patients undergoing bypass or valve surgery. Symptoms begin approximately one to six months following the procedure and include fever, chest pains, friction rub, pleural and pericardial effusions and parenchymal consolidations. This syndrome is thought to be related to an immune reaction involving necrotic visceral pericardium.

Pericardial tamponade occurs in less than 5% of patients who undergo bypass surgery. The diagnosis is expected when excessive blood loss through the drainage tubes is accompanied by increase in central venous pressure, absence of pulmonary interstitial edema, decrease in systemic arterial pressure, tachycardia and decreased urinary output. Delayed tamponade may result from long-term anti-coagulant therapy or from perforation of the pericardium by a transvenous catheter. Chylopericardium following bypass surgery has been described.

Acute cardiac herniation may follow surgery particularly when a defect has been created as part of a pericardial pneumonectomy. Herniation of the heart may also occur through the heart with increased negative pleural pressure following thoracoscentesis. When herniation occurs following development of a right-sided pericardial defect, the cardiac apex usually rotates to the right. The cardiac apex remains left-sided in a left-sided hernia and the defect may be difficult to appreciate. Separation of the cardiac apex from the diaphragm by air in the pleural space is a clue to cardiac herniation in the presence of a pneumothorax. Impending herniation through a small pericardial defect may result in "a snowcone" appearance characterized by a mound of soft tissue extending away from the heart border.

Pericardial Trauma

Blunt abdominal thoracic trauma is a well recognized cause of pericardial disease. A intrapericardial hernia of abdominal contents may occur when a defect is present in the transverse septum of the





diaphragm providing a passageway between the peritoneum and the pericardium. Those patients who present with acute traumatic herniation of abdominal contents into the pericardial space may present with hypovolemic shock, tamponade and respiratory and abdominal signs and symptoms. Conversely, when the hernia develops slowly the patient may be asymptomatic. CT, barium studies and plain film radiographs may be suggestive of this diagnosis.

Pneumopericardium may be a complication of blunt or penetrating trauma, surgery or infection. Pneumopericardium can occasionally lead to tamponade or to tension pneumopericardium. Air reaches the pericardium by dissection of interstitial planes into the mediastinum and subsequently into the pericardial space. Pneumopericardium may be difficult to distinguish from pneumomediastinum. Air that outlines the aortic knob or more than 1-2 cm of superior vena cava lies outside confines of the pericardium. Conversely, pericardial air may demonstrate movement to the nondependent position with decubitus positioning unlike pneumomediastinum which remains fixed in position. "Small heart" sign due to tension pneumopericardium has also been described.

Congenital Absence of the Pericardium

Congenital absence may involve all or part of the parietal pericardium. Most defects are partial involving a defect over the left atrial appendage and the adjacent pulmonary artery.¹ About 20% of patients with pericardial defects have associated heart and mediastinal abnormalities including atrial septal defect, tetralogy of Fallot, bronchogenic cysts and pulmonary sequestration.¹ Those patients with a partial defect are at risk for herniation of a portion of the heart through the defect.

Most defects can be identified on plain film radiographs. Defects on the left side mediastinum rotate the heart in that direction producing pronounced levocardia. Other signs include 1) a prominent notch between the aorta and pulmonary artery filled with aerated lung or fat, 2) lung between the heart and the diaphragm, 3) continuity of the pericardial space with the pleural cavity on CT and 4) lung between the right atrium and right ventricular outflow tract.¹

Pericardial Masses

Pericardial tumor may originate from the heart, pericardium or adjacent structures. Pericardial metastases are found in half of those patients dying from lung or breast carcinoma. In addition, metastatic tumor to the pericardium may occur from melanoma or lymphoma.

Cysts of the pericardium are common and most occur at the right cardiophrenic angle. Bronchogenic cysts may also develop within the pericardium. Lipomas, fibromas and other benign masses of the pericardium are uncommon.

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Current Therapy for Cardiomyopathy: VADs, Myocardial Resection, and Cardiac Transplantation



Barbara L. Knisely, MD

Objectives

The number of people surviving myocardial infarctions and other heart problems has steadily increased with advances in medicine. As a result, an increasing number of patients are left with a damaged heart requiring treatment with medications, surgical procedures, pumps to assist the heart, and heart transplantation. In this presentation, the normal radiographic and CT findings of cardiac transplantation and two commonly implanted ventricular assist devices will be presented. Radiographic and CT findings of complications related to these surgical procedures will also be presented.

Introduction

Cardiac transplantation is the currently accepted treatment for end-stage cardiomyopathy and has 1- and 5-year survival rates of 79% and 63%, respectively. In light of the severe shortage of organ donors, the implantable left ventricular assist device (LVAD) is being considered as a permanent device for heart failure. Currently, LVADs perform the work of the left ventricle as a bridge to cardiac transplantation. A controversial operation, left ventricular partial ventriculectomy, has been proposed for treatment of refractory heart failure due to idiopathic dilated cardiomyopathy. The partial ventriculectomy reduces the size of the left ventricle thereby improving cardiac function.

Cardiac Transplantation

Orthotopic Cardiac Transplantation

In orthotopic cardiac transplantation, the donor heart is joined to the recipient's atria, aorta, and pulmonary artery. The donor inferior vena cava is sewn into the right atrial anastomosis and the donor superior vena cava shunt is tied off just superior to the right atrium.

The normal post-operative chest radiograph typically reveals an enlarged cardiac silhouette related to the discrepant sizes of the transplanted heart and the native pericardium. Occasionally a pericardial effusion is present as a result of immunotherapy with cyclosporine or after placing a small donor heart into a larger pericardial sac. The enlarged cardiac silhouette

decreases in size over several months. On post-operative chest radiographs, the overlap of donor and recipient right atria may cause a double right atria contour. Mediastinal widening is seen in the first and second post-operative weeks in all cases, related to bleeding. Delayed mediastinal widening may be seen in cases of steroid-induced mediastinal lipomatosis. In the first 20 days following median sternotomy, mediastinal fluid collections and substernal air pockets are normally seen. Subcutaneous emphysema, pneumothorax, pneumomediastinum, and pneumopericardium are frequently present immediately following sternotomy and usually resolve in several days.

On chest CT, a high redundant main pulmonary artery may be present as a normal post-operative appearance. A large space may be present between the donor ascending aorta and main pulmonary artery and between the recipient superior vena cava and donor ascending aorta. The recipient and donor ascending aortas may differ in size. A radiosynthetic patch may surround either the pulmonic or aortic anastomosis. The remnant donor superior vena cava may be positioned medial to the recipient superior vena cava and posterior to the donor ascending aorta. The native inferior vena cava may be larger than the donor inferior vena cava. An indentation, or waist, in the atria may be caused by the atrial anastomosis.

Heterotopic Cardiac Transplantation

In heterotopic cardiac transplantation, the donor heart is put into the right thorax and joined to the recipient heart. The donor left ventricle provides the majority of left side cardiac output and the native right ventricle supplies the bulk of right side cardiac output. Patients who have potentially reversible or acute myocardial dysfunction, who have high pulmonary vascular resistance or who receive a small donor heart are candidates for a heterotopic cardiac transplantation. Arrhythmias, angina, and thromboembolism risk from the recipient's native heart are disadvantages of this operation. Long-term anticoagulation therapy is necessary to inhibit potential systemic embolization.

On chest radiographs, the heterotopic donor heart is positioned in the right chest, lateral to the native



heart. Significant right lower lobe atelectasis may occur as a result of the heterotopic heart positioned in the right hemithorax. If the heterotopic nature of the transplanted heart is unknown, the patient will appear to have a significantly enlarged heart.

Cardiac Transplantation Complications

Common complications of cardiac transplantation are rejection, infection, malignancy, and accelerated graft atherosclerosis. Within the first year post transplantation, infection and rejection are common causes of death. Accelerated graft atherosclerosis is the most common cause of death after the first year post transplantation. Non-lymphoid malignancies are the second most common causes of death between the fourth and fifth years post transplantation and they approach the incidence of accelerated graft atherosclerosis (18.6% versus 25%). Lymphoma, including lymphoproliferative disease, causes 3-4% of deaths in the first year following transplantation.

Chest radiography and CT play a prominent role in diagnosis of infection and malignancy. Specific diagnoses that can be suggested or made are bronchogenic carcinoma, lymphoproliferative disorder, *Aspergillus* pneumonia, mediastinitis, pneumomediastinum, pneumothorax, hemothorax, aortic dissection, aortic pseudoaneurysm, and pulmonary embolism. Standard modalities for diagnosing acute rejection and accelerated atherosclerosis are endomyocardial biopsy and coronary angiography. Potential alternative tests for diagnosing rejection and accelerated atherosclerosis are ultrafast CT and Ga-67 cardiac scintigraphy, currently being evaluated as less invasive examinations.

Ventricular Assist Devices (VAD)

By the year 2010, 70,000 patients a year may be eligible for VAD placement. Currently, left ventricular assist devices are surgically implanted mechanical devices costing \$17,000 to \$50,000 a unit. The LVAD is being considered as an alternative to cardiac transplantation, in light of the shortage of donor hearts. Complications of VADs are thromboembolism, infection, mechanical failure, pneumothorax, hemothorax, and bowel obstruction.

TCI Heartmate LVAD (Thermocardiostems)

The Heartmate LVAD is implanted in the left upper quadrant of the abdomen. The inflow cannula (directed at the mitral valve) inserts into the left ventricular apex and draws blood into the pump. The 12-15 cm in length Dacron graft (Meadox Medicals, Oakland, NJ) carries the blood from the pump to the ascending aorta. Porcine biosynthetic valves are in the inflow and outflow conduits, located outside the pump. A drive line travels through a fascial tunnel

and connects the device to an external portable console. Either pneumatic or electric power from the portable console drives the device. On CT, the pump may be accurately localized either within a preperitoneal pocket or an intraabdominal location. The preperitoneal pocket provides improved control of pocket infection and bleeding and prevents the development of intraabdominal adhesions. The intraabdominal position is the choice procedure for patients with small body size.

Pierce-Donachy Thoratec VAD (Thoratec Medical)

Unlike the Heartmate LVAD, the Thoratec device may be used as either a right or a left LVAD. The pump and parts of the outflow and inflow cannulas of the Thoratec VAD are external to the patient. The outlet and inlet valves are mechanical Bjork-Shiley valves, allowing unidirectional flow. Biventricular support is needed when signs of acute right ventricular failure develop intraoperatively following insertion of a LVAD. The inflow cannula of a RVAD connects with the right atrium or ventricle, and the Dacron graft outflow cannula connects with the pulmonary artery.

Myocardial Resection

Left ventricular partial ventriculectomy is an unconventional operation designed to reverse some aspects of cardiac remodeling, as a treatment of congestive heart failure. The concept of ventriculectomy is that reduction of left ventricular chamber diameter may improve cardiac function in heart failure. The operation was developed two years ago in Brazil by Dr. Batista. The operation involves a wide resection of the lateral wall of the left ventricle, starting from the apex, extending between the papillary muscles, and ending proximal to the mitral annulus. The partial ventriculectomy may be isolated or may be associated with valve annuloplasty. The left ventricular partial ventriculectomy improved left and right ventricular function, cardiac output, and functional class in early studies. Initial results were associated with a high prevalence of sustained ventricular tachycardia following the procedure. American surgeons have lost their enthusiasm for the operation as the majority of their patients relapsed into heart failure and were returned to the transplant list.

The Future

In the United States, approximately 40,000 patients die from congestive heart failure each year. Cardiac transplantation would benefit about 25,000 of these patients, however there were only approximately 3,000 heart transplants performed in 1998. New alternative innovations to transplantation are in progress.

Researchers are currently working on implantable pumps that do not need wires passing through the skin, reducing the risk of infection. A device to replace the entire heart and a process to transform marrow cells into healthy myocardial cells are other developments that are currently being studied. Hopefully, research will yield alternative therapies to treat the growing number of patients with cardiomyopathies.

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