World Journal of Radiology

REVIEW

Radiological features of uncommon aneurysms of the cardiovascular system

Kevin Kalisz, Prabhakar Rajiah

Kevin Kalisz, Department of Radiology, University Hospital Cleveland Case Medical Center, Case Western Reserve University School of Medicine, Cleveland, OH 44106, United States

Prabhakar Rajiah, Department of Radiology, Cardiothoracic Imaging, UT Southwestern Medical Center, Dallas, TX 75390, United States

Correspondence to: Prabhakar Rajiah, MD, Associate Professor of Radiology, Department of Radiology, Cardiothoracic Imaging, UT Southwestern Medical Center, 5323 Harry Hines Boulevard, Dallas, TX 75390,
United States. radprabhakar@gmail.com
Telephone: +1-214-6483121
Fax: +1-216-8445205

Abstract

Although aortic aneurysms are the most common type encountered clinically, they do not span the entire spectrum of possible aneurysms of the cardiovascular system. As cross sectional imaging techniques with cardiac computed tomography and cardiac magnetic resonance imaging continue to improve and becomes more commonplace, once rare cardiovascular aneurysms are being encountered at higher rates. In this review, a series of uncommon, yet clinically important, cardio-vascular aneurysms will be presented with review of epidemiology, clinical presentation and complications, imaging features and relevant differential diagnoses, and aneurysm management.

Key words: Cardiovascular; Computed tomography; Magnetic resonance; Aneurysm; Vascular

Core tip: This article will review the epidemiology, clinical presentation, complications, imaging features, differential diagnoses, and management of a series of uncommon cardiovascular aneurysms.

INTRODUCTION

Compared to true aneurysms of the thoracic aorta, aneurysms of other cardiovascular structures are rela-

Table 1 Summary of findings, complications, and treatment in uncommon aneurysms

Aneurysm	Findings	Complications	Treatment
Giant aneurysm	Thoracic aortic aneurysm > 10 cm	Rupture; mediastinal compression	Surgical repair
Pseudoaneurysm	Saccular, thin-neck aneurysm at aortic arch	Rupture; infection	Surgical repair
Ductus aneurysm	Saccular, thin-neck aneurysm at aortic isthmus	Rupture; infection	Surgical repair
Mycotic/inflammatory aneurysm	Mycotic: Saccular outpouching with surrounding inflammatory stranding, fluid collection, gas; inflammatory: Broader based aneurysm with surrounding inflammatory stranding	Rupture	Mycotic: Antibiotics, surgical repair; inflammatory: Anti-inflammatory medications, surgical repair
Aneurysm with fistula	Aneurysm with connection to adjacent bowel or vessel	Rupture; GI bleed (aortoesophageal, aortoenteric); sepsis (aortoesophageal, aortoenteric); cardiac failure (aortocaval)	Surgical repair
Ventricular aneurysm	Ventricular aneurysm containing myocardium (true) or absent myocardium (false)	Rupture with tamponade; thromboembolism	True: Medical/interventional therapy; false: Surgical repair
Atrial/ventricular septal aneurysm	Atrial: Focal outpouching at fossa ovalis > 1.5 cm; Ventricular: Focal outpouching at membranous septum	Thromboembolism (atrial); arrhythmias (atrial); outflow tract obstruction (ventricular); shunt (ventricular); rupture (ventricular)	Atrial: Medical/stroke prevention; ventricular: Unknown[1]
Coronary artery aneurysm	Focal (aneurysm) or diffuse (ectasia) dilatation greater 1.5 times normal diameter	Myocardial infarction; rupture; fistula formation	Medical therapy; surgical repair
Sinus of Valsalva aneurysm	Outpouching of sinus without aortic aneurysm; "windsock" deformity	Rupture; thromboembolism; infection	Surgical repair
Aberrant right subclavian aneurysm	Aneurysm at origin of aberrant right subclavian artery	Esophageal, airway compression; rupture; thromboembolism	Surgical repair
Brachiocephalic artery aneurysm	Aneurysm of brachiocephalic artery ± aortic arch	Mediastinal compression; rupture; infection Thromboembolism	Surgical repair
Pulmonary artery aneurysm and pseudoaneurysm	Pulmonary artery dilatation greater 1.5 times normal diameter	Right heart failure; rupture	Unknown[1]
SVC aneurysm	Aneurysmal dilatation of SVC	Thromboembolism; rupture	Unknown[1]

[1]Unknown: No clear consensus on treatment exits or not enough experience. SVC: Superior vena cava; GI: Gastrointestinal.

tively uncommon. As computed tomography (CT) and magnetic resonance imaging (MRI) technology continues to improve and are more commonly used, these once rare entities are now being encountered more frequently by radiologists. Many of these aneurysms produce non-specific symptoms often overlapping with more common cardiovascular disease processes. Even if asymptomatic, many of these aneurysms are associated with a high risk of potential complications that may still require prompt treatment.

Although these aneurysms may initially be encountered at screening echocardiography, sonographic evaluation is often incomplete both in evaluating the extent of the aneurysms as well as for the presence of complications. Echocardiography has limited field-of-view and is operator dependent and challenging in patients with poor acoustic windows. CT is the most commonly used cross-sectional imaging modality in the evaluation of these aneurysms due to its wide availability and rapid acquisition time. CT has good spatial and temporal resolution, with wide field-of-view and multi-planar reconstruction capabilities. Electrocardiographic (ECG) gating is performed, either prospectively or retrospectively, to minimize motion artifacts which are common in the heart and ascending aorta. Radiation dose is a factor when using CT, but can be minimized by optimizing tube current and voltage, using prospective instead of retrospective ECG gating and using iterative reconstruction algorithms.

MRI is also a valuable imaging modality, which is used for imaging aneurysms without the need for ionizing radiation. It has good spatial resolution, good temporal resolution, wide field-of-view and multi-planar imaging capabilities. MR angiography can be obtained in any desired plane. MRI can also provide functional information, including data on ventricular and valvular function. Late gadolinium enhancement (LGE) is used to evaluate ventricular aneurysms and myocardial scarring. The use of gadolinium contrast media, however, is restricted in patients with severe renal dysfunction due to the risk of nephrogenic systemic fibrosis. Recent technologic developments allow acquisition of MR angiographic images without the use of intravenous contrast, using techniques such as ECG gated three-dimensional balanced steady state free precession (SSFP), time of flight, phase contrast, and ECG-gated fast spin echo.

In this review, a series of uncommon cardiovascular aneurysms will be presented with review of epidemiology, clinical presentation and complications, imaging features and relevant differential diagnoses, and aneurysm management.

A summary of key findings, complications, and treatment in uncommon aneurysms, discussed in greater detail in the following sections, are in included in Table 1.
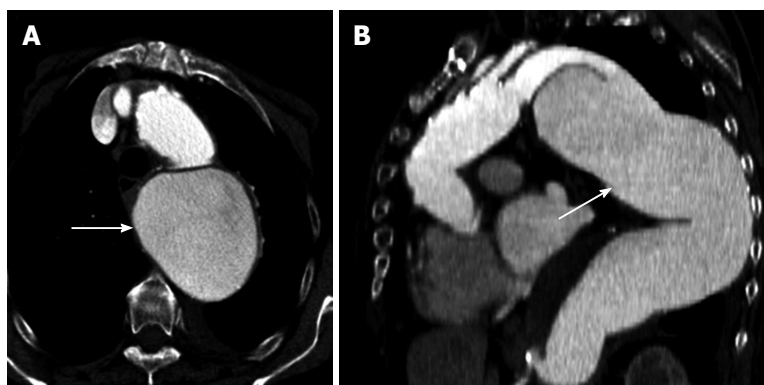
Figure 1 Giant aortic aneurysm. A: Axial contrast enhanced computed tomography (CT) shows markedly dilated 12 cm aneurysm (arrow) in the proximal descending thoracic aorta; B: Sagittal contrast enhanced CT shows aneurysm (arrow) origin beyond an elephant trunk graft.
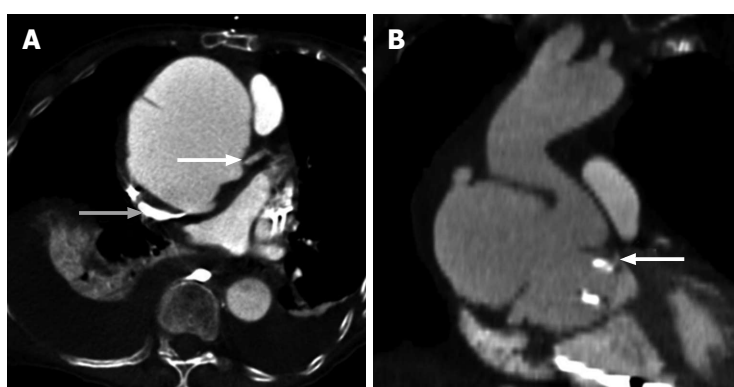
Figure 2 Giant aortic aneurysm with vascular compression. A: Axial contrast enhanced computed tomography (CT) shows giant aortic aneurysm with dissection resulting in compression of the left main coronary artery (LMCA) ostium (white arrow) and superior vena cava (gray arrow); B: Coronal contrast enhanced CT shows giant aneurysm with dissection compressing the LMCA ostium (white arrow).

GIANT ANEURYSM

While thoracic aortic aneurysm is a relatively common entity, giant aneurysms are thought to be extremely rare. Giant aortic aneurysms are defined as aortic aneurysms measuring greater than 10 cm in diameter[1]. Risk factors are thought to be similar to those as smaller thoracic aortic aneurysms, which include atherosclerotic disease, connective tissue disorders (Marfan's syndrome, Ehlers-Danlos syndrome), vasculitis (giant cell arteritis, Takayasu's arteritis), infections (syphilis, tuberculosis, human immunodeficiency virus), cystic medial necrosis, and post-surgical. However, it is unclear which, if any, of these risk factors are more prone to develop giant aneurysms as larger studies are lacking. The presentation of giant aneurysms is variable and largely depends on the presence of complications. Symptoms can vary from chest pain and dyspnea in uncomplicated cases to hemodynamic compromise and death in cases of dissection or rupture. Incidentally found, asymptomatic cases have also been reported[1,2].

Imaging findings of uncomplicated giant aneurysms are analogous to those of smaller thoracic aneurysms (Figure 1). However, given the large size of these aneurysms, there may be compression and mass effect on surrounding vascular and soft tissue mediastinal and thoracic structures (Figure 2). Complications include rupture into the mediastinum or pericardium and dissection. Furthermore, compression of surrounding structures has been reported leading to complications such as lung collapse[3], superior vena cava (SVC) syndrome[4,5], and airway compression[6]. Treatment of giant aneurysms involves surgical resection per Task Force guidelines[7], although operations in these cases may be more technically challenging and associated with higher morbidity[1].

AORTIC PSEUDOANEURYSM

A pseudoaneurysm is a focal dilatation of an artery that does not contain all three of the normal arterial wall layers. Pseudoaneurysms are contained by an outer adventitial layer or thrombus and fibrous tissue. Non-traumatic pseudoaneurysms are usually seen in the setting of inflammatory or infectious etiologies[8]. Additionally, they may be seen as an uncommon complication of cardiovascular surgeries. Post-operative pseudoaneurysms preferentially develop at aortotomy sites, cannulations sites in the setting of cardiopulmonary bypass, needle puncture sites, cross clamping sites, and surgical anastomoses[9]. The main and most feared complication is progressive enlargement and rupture. Other potential complications include fistula formation or
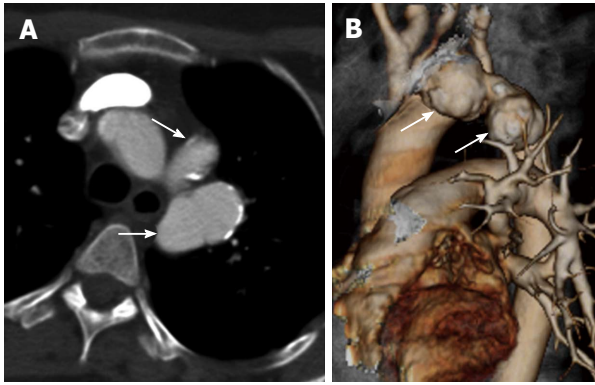
Figure 3 Traumatic aortic pseudoaneurysm. A: Axial contrast enhanced computed tomography (CT) shows a pair of peripherally calcified pseudo-aneurysms at the aortic isthmus (arrows) from remote trauma; B: Three-dimensional volume rendered CT shows a pair of pseudoaneurysms at the aortic isthmus (arrows).
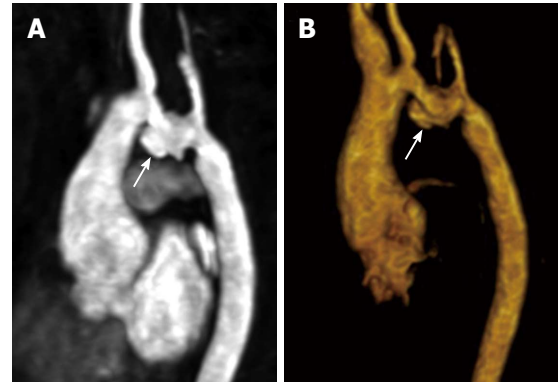
Figure 4 Coarctation repair pseudoaneurysm. A: Magnetic resonance angiography (MRA) maximum intensity projection reconstruction shows post-ductal aortic pseudoaneurysm (arrow) at the surgical site after coarctation resection and end-to-end anastomosis; B: Three-dimensional volume rendered MRA shows post-ductal aortic pseudoaneurysm (arrow) post coarctation repair.
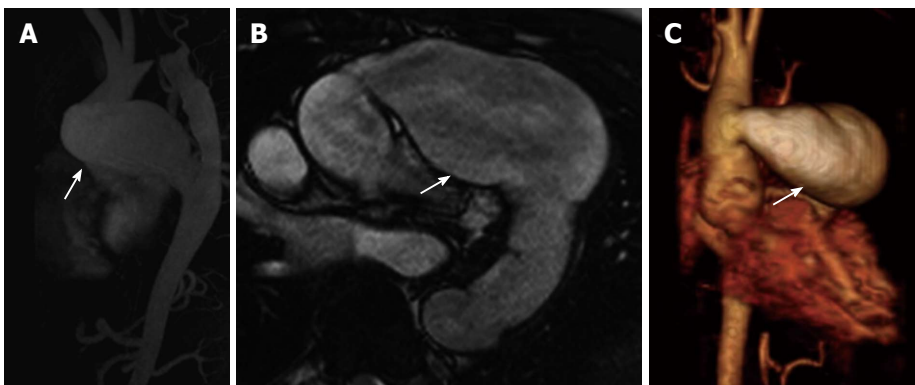
Figure 5 Aortic pseudoaneurysm after bypass graft. A: Sagittal contrast enhanced magnetic resonance angiography (MRA) maximum intensity projection reconstruction shows proximal anastomotic pseudoaneurysm (arrow) of the aorta post ascending to descending aortic bypass graft after prior type B aortic dissection; B: Axial contrast enhanced MRA shows proximal anastomotic pseudoaneurysm (arrow) compressing the main pulmonary artery posteriorly; C: Three-dimensional volume rendered MRA shows proximal aortic anastomotic pseudoaneurysm (arrow).

superinfection[10].

On CT and MRI, pseudoaneurysms appear as sac-cular dilatations arising from the aorta with a narrow neck forming an acute angle with the aorta (Figure 3). The underlying etiology of the pseudoaneurysm may assist in determining the likely location. For example, locations of post-surgical pseudoaneurysms (Figures 4-7) will depend on the preceding surgical technique (location of cannulation sites, *etc*.). These aneurysms may also contain mural calcifications or thrombus. When detected, these aneurysms are treated with surgical repair given the risk of rupture, either soon after the initial traumatic event or later.

DUCTUS ANEURYSM

A subset of aortic pseudoaneurysms includes ductus aneurysms, which are located at the aortic isthmus. Ductus aneurysms typically develop secondary to aortic trauma. In the setting of blunt thoracic trauma, particularly involving significant shear or deceleration forces as in motor vehicle collisions, approximately 2.5%

of patients who survive the initial aortic injury go on to develop chronic aortic pseudoaneurysms[11]. Thus, while ductus aneurysms are seen in the acute traumatic setting, they can also be encountered long after an initial trauma, particularly in patients who did not initially receive cross sectional imaging of the chest. Ductus aneurysms are also seen as a complication of aortic coarctation repair[12]. CT and MRI features mirror those of pseudoaneurysms discussed previously occurring at the aortic isthmus (Figure 8). Chronic ductus aneurysms, typically have similar morphology as those in the acute setting, but often demonstrate a peripheral rim of calcification and may contain thrombus[13]. Additionally, one should also inspect for features of prior thoracic trauma or coarctation repair.

An important differential diagnosis of these aneurysms, particularly ductus aneurysms, is a ductus diverticulum. A ductus aneurysm is a focal outpouching formed as a remnant of a closed ductus arteriosus or possibly a remnant of the right dorsal aortic root[14]. A ductus diver-ticulum is typically seen at the anteromedial aspect of the aorta at the site of the ductus arteriosus insertion.
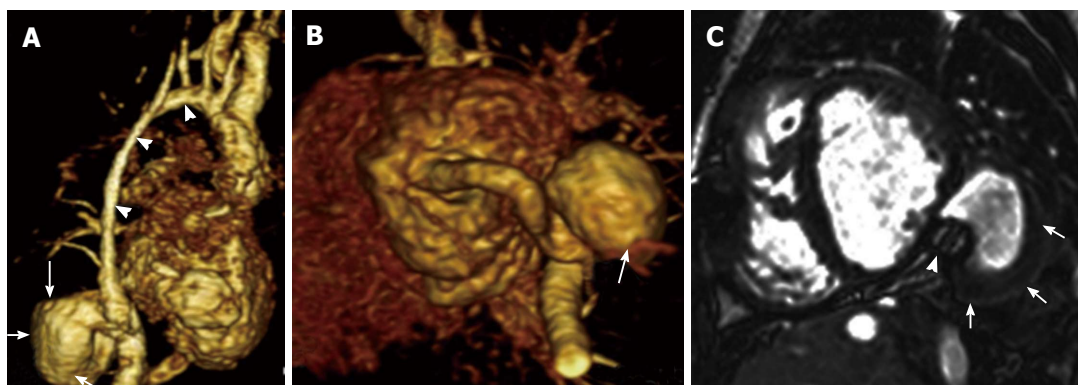
Figure 6 Aortic pseudoaneurysm with aortic conduit. A: Three-dimensional (3D) volume rendered magnetic resonance angiography (MRA) in a patient with a history of trisomy 21, atrioventricular canal defect, and aortic hypoplasia status post left ventricular apex-descending aorta aortic conduit (arrowheads) shows aortic pseudoaneurysm (arrows) distal to the anastomotic site; B: 3D volume rendered MRA shows distal anastomotic site aortic pseudoaneurysm (arrow) post left ventricular apex-descending aorta aortic conduit; C: Short axis contrast enhanced magnetic resonance image shows pseudoaneurysm (arrows) adjacent to the tilted valve of the conduit (arrowhead).
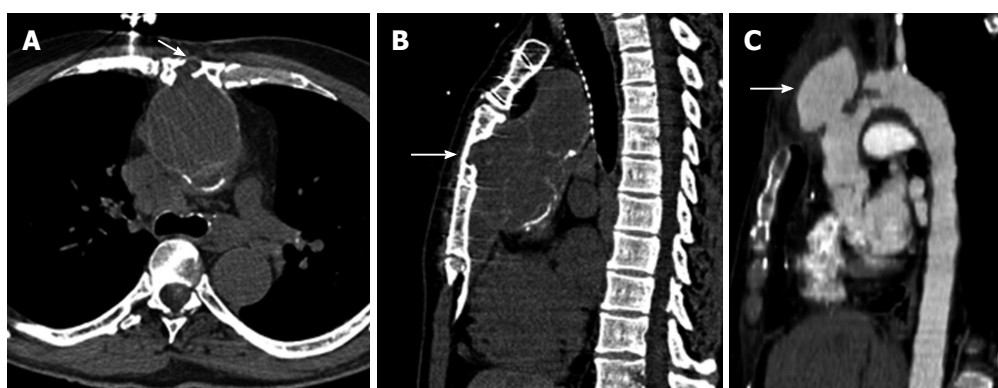
Figure 7 Aortic pseudoaneurysm with sternal erosion. A: Axial non-enhanced computed tomography (CT) shows pseudoaneurysm of the ascending aorta in a patient post aortic graft repair extending to and eroding into posterior aspect of the sternum (arrow); B: Sagittal non-enhanced CT shows large pseudoaneurysm of the ascending aorta occupying a majority of the anterior mediastinum with extension into the adjacent sternum (arrow); C: Sagittal contrast enhanced CT shows filling pseudoaneurysm eroding into the sternum (arrow).
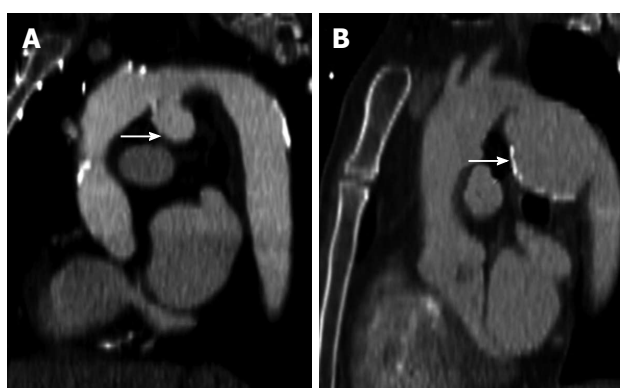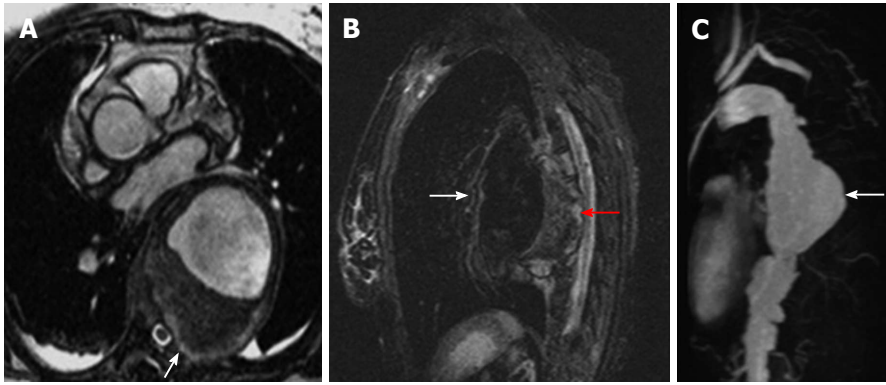

Figure 8 Ductus aneurysm. A: Sagittal contrast enhanced computed tomography (CT) shows pseudoaneurysm (arrow) arising from the aortic isthmus and projecting inferiorly in a patient with a history of remote thoracic trauma; B: Sagittal contrast enhanced CT shows large, peripherally calcified ductus aneurysm (arrow).

As opposed to ductus aneurysms, ductus diverticuli form smooth margins and obtuse angles, rather than acute, with the aorta. Simple ductus diverticuli do not require regular follow-up or treatment.

MYCOTIC ANEURYSMS

A mycotic aneurysm is an aneurysm arising from an infection involving the arterial wall. In the setting of infection, structural components of the arterial wall are destroyed, leading to contained rupture and pseudoaneurysm formation. Mycotic aneurysms are thought to account for 0.7%-1% of all surgically treated aortic aneurysms. Although mycotic aneurysms can arise from any artery, the aorta is the most common site. Underlying infectious etiology is usually bacterial with staphylococcus and streptococcus species comprising the most common offending agents. Risk factors for aneurysm development include bacterial endocarditis, intravenous drug abuse, and immunocompromised states[15]. Presenting symptoms may be primarily due to underlying infection, but may also include chest pain and pulsatile mass, depending on aneurysm size. In the setting of active infection, mycotic aneurysms expand more rapidly than other types of aneurysms and are associated with high mortality, either from rupture or underlying sepsis.

On CT and MRI, mycotic aneurysms usually appear

Figure 9 Mycotic aneurysm. A: Axial contrast enhanced magnetic resonance angiography (MRA) shows large proximal descending aortic aneurysm with eccentric thrombus and invasion of adjacent thoracic vertebral body (arrow); B: Sagittal short tau inversion recovery shows large descending aortic aneurysm (white arrow) with high signal within the wall posteriorly (red arrow) indicating acute inflammation; C: Sagittal contrast enhanced MRA maximum intensity projection reconstruction shows proximal descending aortic mycotic aneurysm (arrow) extending posteriorly.
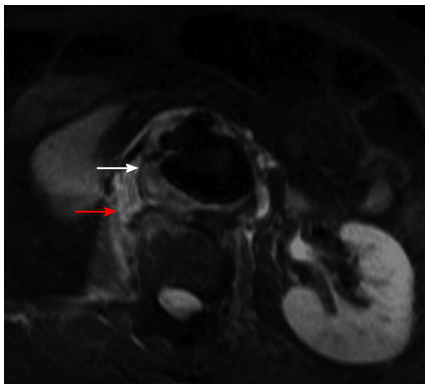
Figure 10 Aortitis aneurysm. Axial short tau inversion recovery in a patient with Takayasu arteritis shows abdominal aortic aneurysm (white arrow) with wall thickening and high signal (red arrow) indicating acute inflammation.

as solitary, focal saccular outpouching arising from the aortic wall. These aneurysms demonstrate associated infectious signs including periaortic soft tissue stranding, edema, fluid and possibly air foci. On MRI, high signal on T2 weighted images is seen indicating active in-flammation (Figure 9). Thrombus formation within the aneurysm may be seen, although, unlike typical athero-sclerotic aneurysms, calcifications are rare. In addition to antibiotics to treat the underlying infection, emergent surgical repair is undertaken given the high risk of rupture[16].

## ANEURYSM ASSOCIATED WITH AORTITIS

A related, but pathologically distinct form of aneurysm is that associated with inflammatory aortitis. Aneurysms resulting from aortitis are generally seen in patients with giant cell arteritis, Takayasu's arteritis, Bechet's disease, rheumatic disease, and ankylosing spondylitis. These conditions may directly lead aneurysm formation as a consequence of active inflammation of the arterial wall. The incidence of aneurysm development depends on the underlying inflammatory disease. For example,

aortic aneurysms can be seen up to 45% of patients with Takayasu's arteritis[17]. Also, aortic aneurysm can be the first presenting sign of the underlying vasculitis or inflammatory disease[18].

Although there is a similar underlying inflammatory etiology, imaging findings are usually easily differentiated from those of mycotic aneurysms. Specifically, non-infectious aortitis aneurysms are typically more fusiform and involve a longer segment of the aorta. CT and MRI demonstrate arterial wall thickening with surrounding inflammatory changes. Surrounding signs of infection such as fluid collections or air foci are typically not present in non-infectious aneurysms. Again, at MRI high T2 weighted signal within the arterial walls indicates the presence of mural edema (Figure 10). Contrast enhancement is also seen in the arterial wall. Treatment of inflammatory aortitis involves anti-inflammatory and immunosuppressive therapies. However, aortitis aneurysms should undergo repair, although typically pursued less urgently than with infectious mycotic aneurysms.

ANEURYSM WITH FISTULA

As aneurysms enlarge, vessels can form fistulous con-nections with surrounding structures, both vascular and non-vascular. A number of factors predispose to fistula formation including trauma, infection, or other inflammatory conditions. Given the proximity of the thoracic and abdominal aorta to a variety of different structures within the chest and particularly the upper abdomen, a number of potential fistulous connections can form with a variety of clinical consequences. For example, thoracic aortic aneurysms may erode into the adjacent esophagus. Although aortoesophageal fistulas are relatively rare in general, erosions from thoracic aorta aneurysms are the most common etiology[19]. Continued enlargement of these aneurysms may pro-duce ultimate rupture with often fatal gastrointestinal bleeding. Similarly, within the abdomen, abdominal aortic aneurysms may form fistulas with adjacent loops
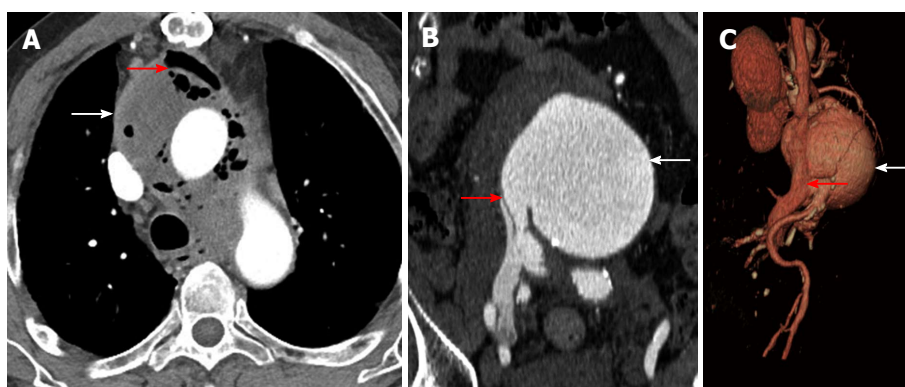
Figure 11 Fistula. A: Aortoesophageal fistula-axial computed tomography (CT) scan shows multiple gas locules in the ascending aorta (arrows) consistent with aortoesophageal fistula; B: Coronal contrast enhanced CT shows a large abdominal aortic aneurysm (white arrow) near the level of the bifurcation communicating with the inferior vena cava (red arrow), which is isoattenuating to the aorta; C: Three-dimensional volume rendered CT shows abdominal aortic aneurysm (white arrow) with fistulous connection to the inferior vena cava (red arrow).

of bowel, with the duodenum being the most common site of connection[20]. Rupture with lethal gastrointestinal bleeding is the most feared complication. Additionally, any fistula formation with bowel predisposes patient to sepsis from enteric-born pathogens. Lastly, aortic aneurysms may forms fistulas with the adjacent inferior vena cava (IVC), resulting in an aortocaval fistula. Aortocaval fistula is a similarly rare phenomenon occurring in only 1% of abdominal aortic aneurysms and 4% of ruptured aneurysms at the time of operation[21]. Aortocaval fistulas most typically form between the posterolateral aorta and adjacent IVC and may present with symptoms of a pulsatile abdominal mass, abdominal and back pain, and high output cardiac failure[22].

Common imaging findings among the various types of aneurysmal fistulas include distortion of vessel and adjacent structure contours with loss of the normal distinct fat plane between the structures. In aortoesophageal of aortoenteric fistulas, direct signs of fistula formation include the presence of gas foci with the vessel (Figure 11A) or active contrast extravasation into the esophageal of bowel lumen. Other direct signs include increased attenuation of luminal contents or presence of discrete hematoma. Findings of aortocaval fistulas include early opacification of the IVC, eventually becoming isoattenuating to the aorta (Figure 11B and C). A direct fistulous tract may or may not be seen. Treatment of aneurysms with fistula formation is urgent surgical repair.

VENTRICULAR ANEURYSM, PSEUDOANEURYSM, AND DIVERTICULUM

Although more commonly affecting the vasculature, aneurysms can also occur within the heart chambers. Within the ventricles, both true and false (pseudoaneurysms) can form. True ventricular aneurysms contain a thinner component of normal myocardium as well as fibrous tissue and necrotic myocardium, while pseudoaneurysm walls are composed of organized hematoma and pericardium without normal functional myocardium[23]. Ventricular aneurysms most commonly occur after myocardial infarction where anterior location predominates overall. However, aneurysms can also occur in the setting of non-ischemic conditions such as hypertrophic cardiomyopathy and Chagas disease where there is apical predominance. Following myocardial infarction, aneurysms occur in approximately 3.5%-5% of cases, although this rate is thought to be declining with more prompt and effective treatment of myocardial infarctions[24]. Right ventricular aneurysms have been reported, but are quite rare[25]. When symptomatic, ventricular aneurysms may present with heart failure symptoms due to systolic bulging with resulting "steal" of stroke volume as well as ventricular arrhythmias. The most feared complication of ventricular aneurysms is rupture resulting in tamponade. Aneurysm rupture is much more common in pseudoaneurysms than true aneurysms after the acute setting[26]. Thrombus formation is a potential complication of both true and false aneurysms.

Noninvasive differentiation of true and false ventricular aneurysms by imaging is critically important given the different risk of complications and subsequent treatment strategies for each type. True aneurysms (Figures 12 and 13) generally have a wider mouth that is greater in size than that of the actual aneurysm, while pseudoaneurysms (Figure 14) typically have a narrow mouth typical of pseudoaneurysms in other locations. Although contrast enhanced CT can show overall aneurysm morphology and possible myocardial thinning and scarring, cardiac MRI is particularly useful in evaluating ventricular aneurysms and ventricle morphology. SSFP images are useful for evaluating bulging and morphological changes during the cardiac cycle. Contrast enhanced MRI using LGE for myocardial scar detection can potentially be a useful tool in differentiating true from false aneurysms as pseudoaneurysms have been shown to have intense delayed enhancement[23]. Furthermore, MRI can detect areas of viable myocardium in cases of potential revascularization. Imaging can also detect thrombus formation within the aneurysm. Treatment of
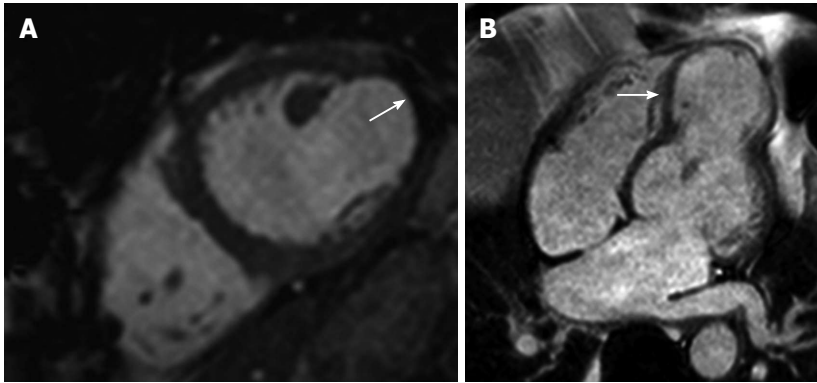
Figure 12 Left ventricular aneurysm. A: Short axis magnetic resonance (MR) steady state free precession shows broad outpouching of the lateral wall of the left ventricle representing true aneurysm with associated dark thrombus formation (arrow); B: Four chamber MR delayed contrast enhanced image demonstrates large true aneurysm of the left ventricular apex (arrow).
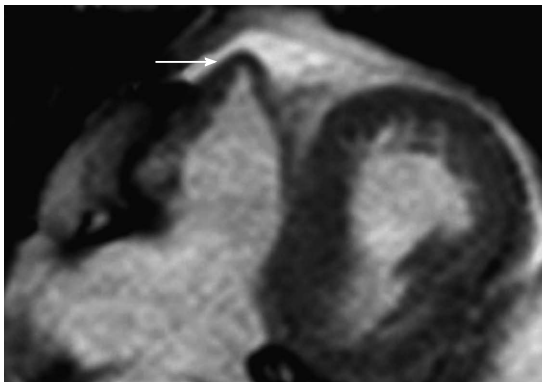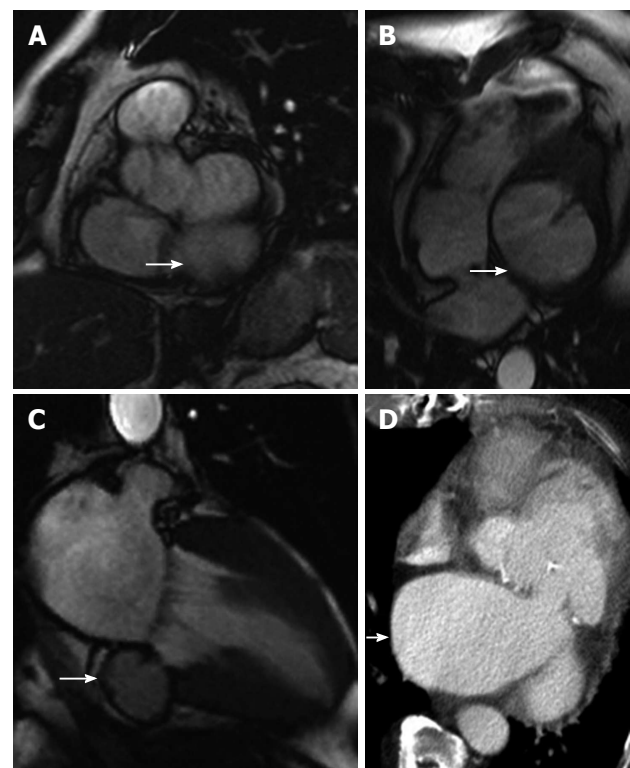

Figure 13 Right ventricular aneurysm. Four chamber magnetic resonance steady state free precession image shows small true aneurysm at the right ventricular apex (arrow).

ventricular aneurysms depends on aneurysm type and presence of symptoms. Asymptomatic true ventricular aneurysms are typically treated medically, while large or symptomatic true aneurysms are treated surgically[27]. Alternatively, ventricular pseudoaneurysms all usually undergo surgical repair given the higher rupture risk.

An uncommon, but important, differential of left ventricular aneurysms is a left ventricular diverticulum. A ventricular diverticulum is an outpouching of the myocardial wall containing normal endocardium, myocardium, and pericardium with normal ventricular contraction in synchrony with the surrounding ventricle. Ventricular diverticuli are congenital with a prevalence of 0.4% and may be associated with other anomalies including midline thoracoabdominal defects[28]. MRI is most useful for differentiating diverticuli from ventricular aneurysms as diverticuli demonstrate normal appearance of all ventricular components and contract synchronously with the surrounding myocardium on cine imaging (Figures 15 and 16). Diverticuli usually do not warrant treatment.

VENTRICULAR SEPTAL ANEURYSM

Ventricular septal aneurysms are uncommon focal outpouchings usually at the membranous portion of


Figure 14 Left ventricular pseudoaneurysm. A: Short axis magnetic resonance (MR) steady state free precession (SSFP) shows pseudoaneurysm at the basal inferior wall of the left ventricle (arrow); B: Four chamber MR SSFP image shows pseudoaneurysm at the basal inferior wall of the left ventricle (arrow); C: Two chamber MR SSFP shows pseudoaneurysm at the basal inferior wall of the left ventricle (arrow); D: Axial contrast enhanced computed tomography image shows large basal inferolateral wall pseudoaneurysm (arrow).

interventricular septum. These aneurysms are usually caused by spontaneous closure of a membranous ventricular septal defect by apposition of the septal tricuspid leaflet. Like atrial septal aneurysms, these are most often discovered incidentally and are asymptomatic. However, unlike atrial aneurysms, ventricular septal aneurysms can result in right ventricular outflow tract (RVOT) obstruction, left to right shunting, rupture, systemic emboli and endocarditis[29]. CT and MRI demonstrate
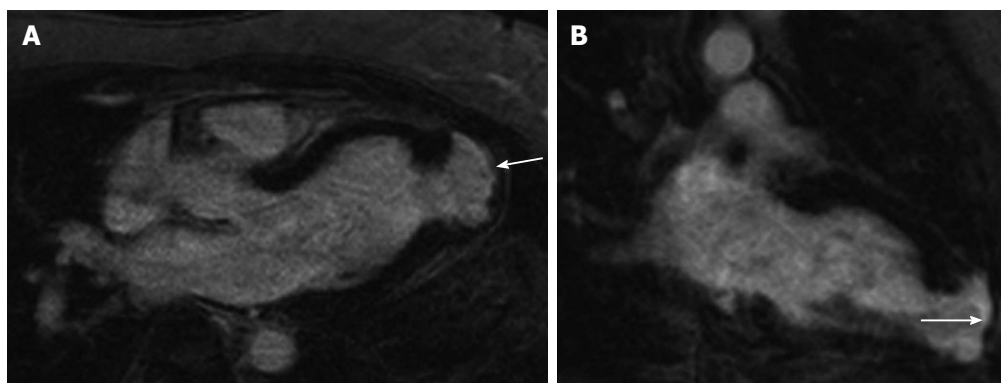
Figure 15 Left ventricular diverticulum. A: Three chamber delayed contrast enhanced (DCE) magnetic resonance (MR) shows focal outpouching at the left ventricular apex (arrow) in the absence of myocardial scarring. Cine images demonstrated synchronous movement during systole consistent with diverticulum (not shown here); B: Two chamber DCE MR image of the same patient shows left ventricle apical diverticulum (arrow).
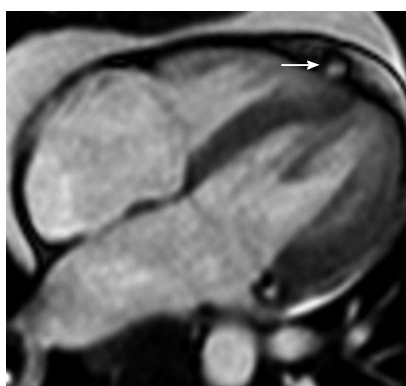
Figure 16 Right ventricular diverticulum. Four chamber magnetic resonance steady state free precession shows small focal outpouching at the right ventricular apex (arrow). Cine images demonstrated synchronous movement during systole consistent with diverticulum (not shown here).

a focal, saccular outpouching from the membranous septum into the right ventricle (Figure 17), which should be distinguished from sinus of Valsalva aneurysm. Although both aneurysm types may protrude into the RVOT, a sinus of Valsalva aneurysm originates above the level of aortic annulus, while a membranous septal aneurysm originates below the level of aortic annulus. A similar appearance can also be seen in a periaortic pseudoaneurysm, but this is typically associated with an abnormal aortic valve in a severely ill patient in the setting of endocarditis. Lastly, patch material from prior ventricular septal defect repair may bulge into RVOT, but the surgical patch material is usually calcified. MRI allows quantification with any functional complications including shunt quantification and assessment of outflow tract obstruction. If discovered in infancy, no treatment of ventricular septal aneurysms is typically pursued as these have shown to resolve spontaneously[30]. However, for aneurysms in adults, no clear treatment guidelines are in place. Surgical treatment can be considered if there are associated complications or hemodynamic abnormalities.
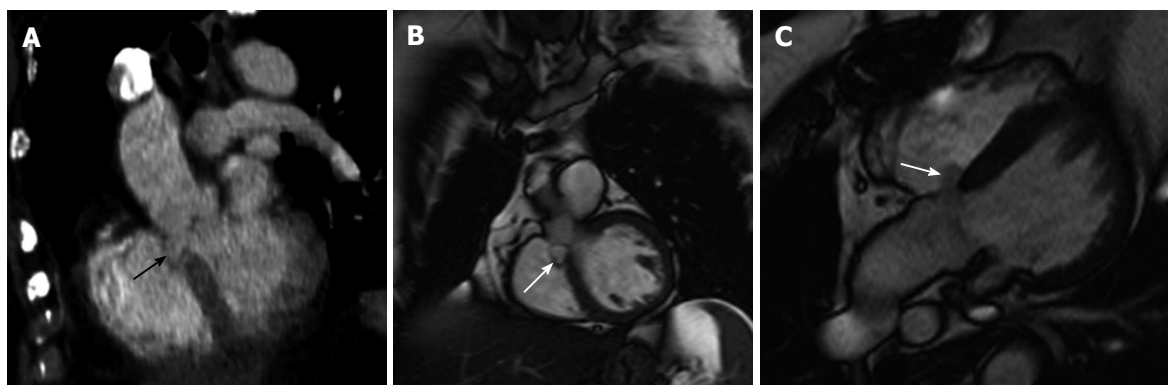
ATRIAL SEPTAL ANEURYSM

Atrial septal aneurysms, found in 2% of healthy patients and 4% of patients undergoing transesophageal echocardiography, are focal, saccular outpouchings typically occurring at the level of the fossa ovalis[31]. These aneurysms are typically congenital and can be associated with other cardiac abnormalities including patent foramen ovale, atrial septal defect, and mitral valve prolapse[32]. Atrial aneurysms can bow into one atrium (left or right) only or they may be bidirectional. These aneurysms in themselves are asymptomatic but may lead to atrial arrhythmias or thrombus formation and are associated with intracardiac shunting in up to 78% of cases[31]. Various studies have shown that the combination of atrial septal aneurysm and patent foramen ovale are associated with a higher risk of stroke[33]. The diagnosis is made by imaging if there is protrusion of the aneurysm sac by at least 1.5 cm into either atrium or if the total excursion exceeds 1.5 cm in cases of bidirectional aneurysms (Figure 18)[34]. Treatment of atrial septal aneurysms is unclear although medical stroke prevention therapy is pursued in cases complicated by embolic stroke.

CORONARY ARTERY ANEURYSM

A coronary artery aneurysm is characterized as a focal dilation, either saccular or fusiform, of a coronary artery greater than 1.5 times its normal diameter. A "giant" coronary aneurysm is defined as aneurysm greater than 20 mm. A related abnormality, coronary artery ectasia, is a diffuse dilatation of the coronary artery. Coronary artery aneurysms are uncommon, occurring between 0.15% and 4.9% at coronary angiography. Coronary artery aneurysms most commonly occur secondary to atherosclerosis with the right coronary artery being the most commonly affected artery[35]. Coronary artery aneurysms can also occur in up to 25% of patients with untreated Kawasaki disease[36]. Other risk factors include Takayasu's arteritis, connective tissue disorders, and trauma. Aneurysms may rarely develop as a complication of coronary bypass surgery[37]. Often times, these aneurysms are asymptomatic, although patients may present with acute coronary syndrome or failure when

Figure 17 Membranous ventricular septal aneurysm. A: Coronal contrast enhanced computed tomography demonstrates saccular aneurysm of the membranous portion of the ventricular septum (arrow), protruding into the right ventricle; B: Short axis magnetic resonance (MR) steady state free precession (SSFP) shows saccular aneurysm of the membranous portion of the ventricular septum (arrow); C: Four chamber MR SSFP shows saccular aneurysm of the membranous portion of the ventricular septum (arrow), protruding into the right ventricle.
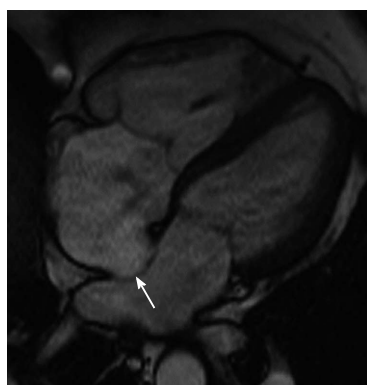
Figure 18 Interatrial septal aneurysm. Four chamber magnetic resonance steady state free precession shows broad-based aneurysm of the interatrial septum extending into the left atrium (arrow).

complicated by thrombosis and distal embolization, while rupture is a rare complication. Congenital aneurysms and ectasia can be associated with fistulous connections to a cardiac chamber or great vessel[35].

ECG-gated coronary CT is especially well suited to detect these aneurysms in relatively small coronary vessels. Coronary CT demonstrates the spectrum of coronary aneurysm findings including focal, short segment (saccular), broader based (fusiform), and diffuse (ectasia) aneurysms (Figures 19 and 20). Coexisting thrombus and coronary stenosis estimates can also be made with CT. Medical treatment is usually dependent on the degree of coexisting atherosclerotic disease. Although no accepted guidelines exist, surgical treatment is generally considered in the setting of aneurysm enlargement, obstruction, or embolization[35].

SINUS OF VALSALVA ANEURYSM

Aneurysms of Sinus of Valsalva (ASVs) are thin-walled outpouchings from a sinus of Valsalva, usually protruding into an adjacent cardiac chamber[38]. These aneurysms are rare with a general prevalence estimate of 0.9%[38]. ASVs most commonly arise from the right coronary

cusp (70%) with approximately 25% arising from the non-coronary cusp and 5% from the left coronary cusp. Aneurysms involving multiple cusps are exceedingly rare[39]. ASVs can be congenital, likely due to failure of fusion between annulus fibrosus of aortic valve and media of aortic wall. They can also be acquired from prior infection or trauma. Approximately 0.15%-1.5% of these aneurysms occur in patients undergoing cardiopulmonary bypass[40]. Most unruptured ASVs are asymptomatic. However, patients present with chest pain, dyspnea, and volume overload symptoms as the aneurysms enlarge and compress adjacent structures such as the RVOT or coronary arteries. The main complication of ASVs are rupture with presentation and symptoms depending in part by which cardiac chamber the rupture extends into. The most common sites of rupture are into the right ventricle and right atrium[40]. Other complications include thrombus formation within aneurysms with subsequent embolic events and aneurysm infection[38].

Imaging criteria for the diagnosis of ASVs include origin above the aortic annulus and a saccular shape without corresponding aortic root or ascending aortic aneurysm. As the aneurysm elongates, a "windsock" deformity extends from the aneurysm to the adjacent cardiac chamber (Figure 21). Although most commonly detected at echocardiography, ECG-gated CT and MRI offer superior, and often definitive, evaluation of ASVs. Specifically, CT allows for superior spatial resolution and anatomic evaluation of involved structures, and MRI can provide functional assessments of left ventricular function, valvular regurgitation, and aortocardiac shunting[41]. Treatment of ASVs involves surgical repair in the setting of ruptured ASV or the presence of malignant arrhythmias, coronary or RVOT obstruction, or superinfection[38].

ABERRANT RIGHT SUBCLAVIAN ARTERY ANEURYSM

An aberrant right subclavian artery (ARSA) is congenital vascular anomaly with estimated incidence between 0.5%-2.0% in which the right subclavian artery, instead
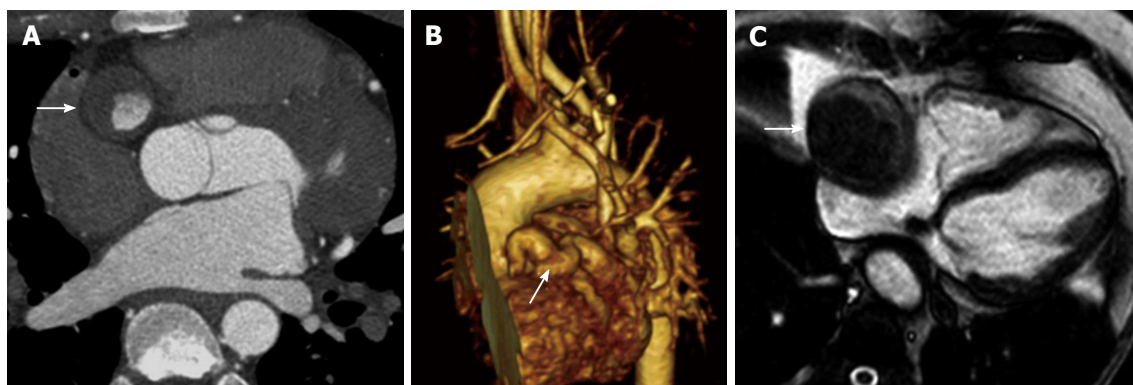
Figure 19 Coronary artery aneurysm. A: Axial contrast enhanced computed tomography (CT) in a patient with a history of Kawasaki disease shows aneurysm of the right coronary artery (arrow) with significant mural thrombus; B: Three-dimensional volume rendered CT shows aneurysm of the right coronary artery (arrow); C: Four chamber magnetic resonance steady state free precession image in patient with saphenous vein graft to the posterior descending artery shows aneurysm of the graft (arrow) with mural thrombus and mass effect posteriorly onto the adjacent right atrium.
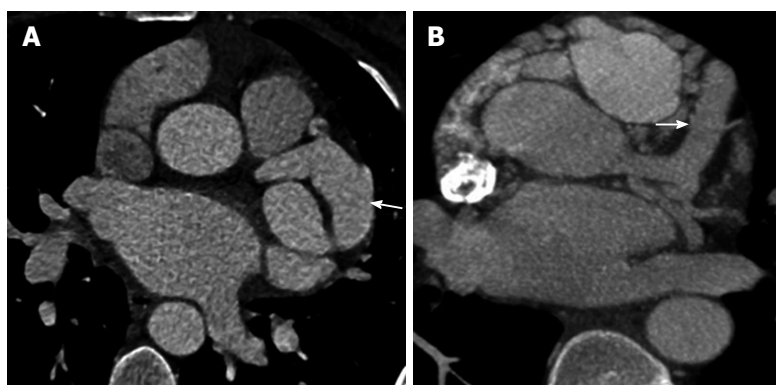
Figure 20 Diffuse coronary ectasia. A: Axial contrast enhanced computed tomography (CT) shows diffuse fusiform enlargement of the left circumflex artery (arrow); B: Axial contrast enhanced CT shows multiple dilated coronary vessels included a significantly dilated left main coronary artery (arrow). This patient's right coronary artery originated from the pulmonary artery.
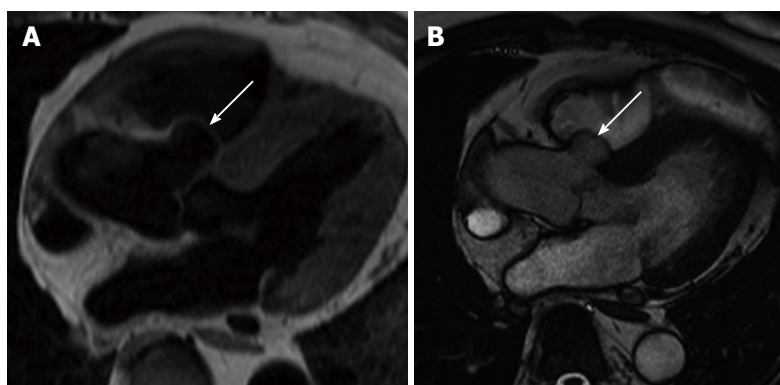

Figure 21 Sinus of Valsalva aneurysm. A: Four chamber black blood magnetic resonance (MR) shows right sinus of Valsalva aneurysm (arrow) protruding into the right ventricular outflow tract (RVOT); B: Four chamber steady state free precession MR shows right sinus of Valsalva aneurysm (arrow) protruding into the RVOT.

of arising as the first branch from the brachiocephalic artery, arises as a fourth branch directly off the aortic arch. The ARSA may take a variable course, but typically traverses posterior to the esophagus before extending to the right side of the thorax. In approximately 60% of cases, patients may have a focal dilatation at right subclavian origin (Kommerell's diverticulum)[42]. However, aneurysmal dilatation of an aberrant right subclavian

is a rare entity found in only approximately 3%-8% of these patients and most commonly occurs secondary to atherosclerotic disease[14,43]. ARSAs may present with symptoms of dysphagia from esophageal compression or dyspnea and cough from airway compression. Potential complications include thrombus formation and rupture with up to 50% mortality with rupture[44].

On imaging, ARSAs are seen as a right subclavian
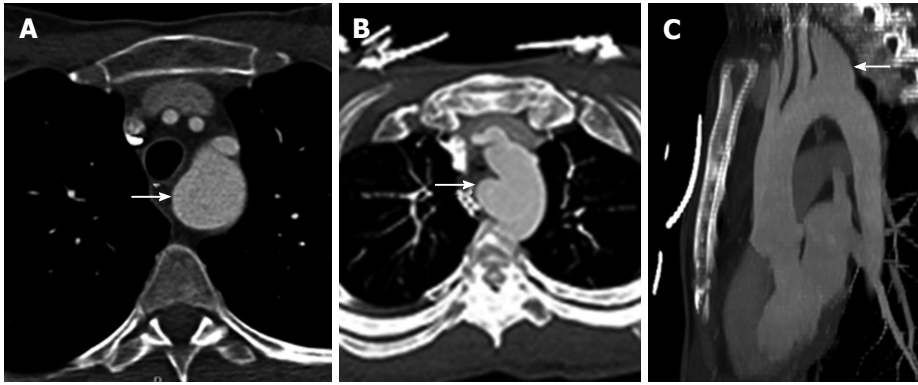
Figure 22 Aberrant right subclavian artery aneurysm. A: Axial contrast enhanced computed tomography (CT) shows aberrant right subclavian aneurysm (ARSA) arising as the last branch of the aortic arch (arrow); B: Axial CT maximum intensity projection reconstruction shows ARSA (arrow) arising from the aorta with posterior course in the mediastinum; C: Sagittal CT maximum intensity projection reconstruction shows ARSA arising as the last branch of the aortic arch (arrow).
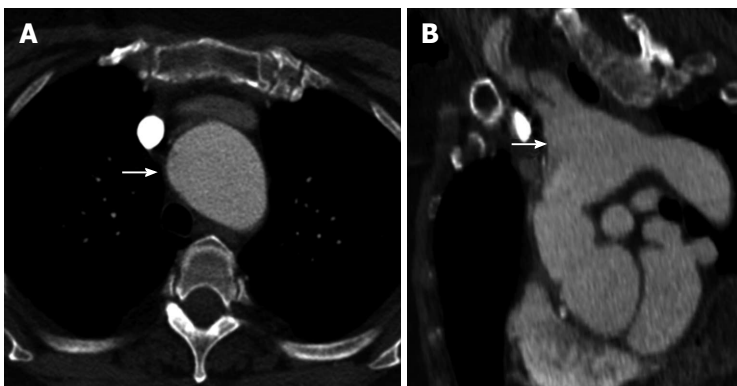
Figure 23 Brachiocephalic artery aneurysm. A: Axial contrast enhanced computed tomography (CT) above the level of the aortic arch shows aneurysm of the right brachiocephalic artery (arrow); B: Coronal contrast enhanced CT shows aneurysmal involvement of the origin of the right brachiocephalic artery (arrow) without aortic involvement.

artery arising directly from the aortic arch distal to the left subclavian origin and coursing posteriorly to the trachea and esophagus (Figure 22). An ARSA aneurysm appears as a focal dilatation at the artery origin, often with associated atherosclerotic disease and some degree of thrombus formation. Mass effects on adjacent mediastinal structures, namely the esophagus and trachea, can also be seen. While asymptomatic aberrant right subclavian arteries do not require any treatment, aberrant right subclavian artery aneurysms are treated surgically given the risk of rupture[44].

BRACHIOCEPHALIC ARTERY ANEURYSM

Brachiocephalic artery aneurysms account for approximately 3% of supra-aortic aneurysms with atherosclerosis accounting for a majority of aneurysms[45,46]. Aneurysms have also been seen in the setting of Takayasu's arteritis, syphilis, post-traumatic states, Marfan's syndrome, and post-operative states[47]. At diagnosis, patients may be asymptomatic, although presenting symptoms usually occur secondary to compressive effects on adjacent structures and include dyspnea, dysphonia, hoarseness, and SVC syndrome[45,47]. Complications include thrombus formation with resulting embolic stroke, aneurysm infection,

and aneurysm rupture. Aneurysm rupture occurs in approximately 11% of patients with trauma thought to be the main risk factor for rupture, although larger studies are lacking[47].

Imaging shows aneurysmal dilatation of the brachiocephalic artery (Figure 23), although different patterns of brachiocephalic involvement have been described. Kieffer *et al*[47] describe three categories of involvement: No involvement of the brachiocephalic origin, involvement of the brachiocephalic origin, but not the aorta (most common), and involvement of both the brachiocephalic and aortic arch. Furthermore, one must be sure to evaluate other thoracic vessels as aneurysms of other supra-aortic vessels can be seen concurrently. Mass effects on adjacent structures including the airways, pharynx, and SVC are also well evaluated on cross sectional imaging. Surgical repair is generally pursued in most cases, particularly in the setting of symptomatic disease, associated aortic aneurysms, or aneurysms greater than 3 cm in diameter[47].

PULMONARY ARTERY ANEURYSM AND PSEUDOANEURYSM

Pulmonary artery aneurysm, defined as focal pulmonary
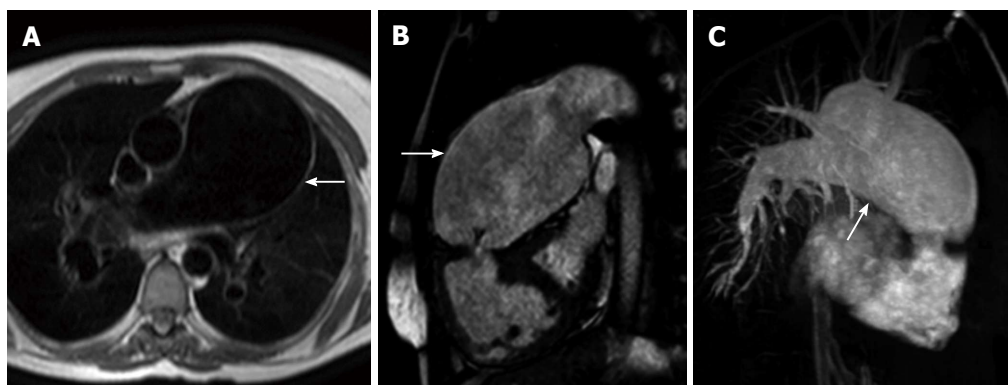
Figure 24 Giant pulmonary artery aneurysm. A: Axial black blood magnetic resonance (MR) image shows massive fusiform enlargement of the main pulmonary artery (arrow) in a patient with atrial septal defect complicated by pulmonary hypertension and Eisenmenger syndrome; B: Right ventricular outflow tract steady state free precession MR shows massive fusiform enlargement of the main pulmonary artery (arrow); C: MR angiography maximum intensity projection reconstruction shows massive fusiform enlargement of the main pulmonary artery (arrow).
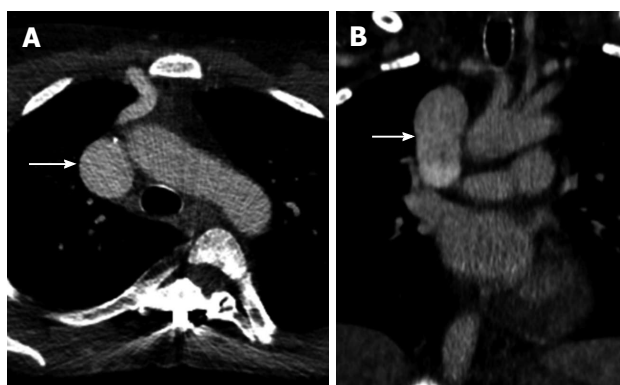
Figure 25 Superior vena cava aneurysm. A: Axial contrast enhanced computed tomography (CT) image shows fusiform enlargement of the superior vena cava (arrow); B: Coronal contrast enhanced CT image shows fusiform enlargement of the superior vena cava (arrow).

artery dilatation greater than 1.5 times the normal diameter, is a rare entity found in one of 14000 patients at autopsy[48]. Pulmonary artery aneurysms may be congenital or acquired. Risk factors include infection, trauma/iatrogenic injury, vasculitis, degenerative, and any underlying cause of pulmonary hypertension[49]. Congenital causes are defective vascular wall, pulmonic stenosis and left-to-right shunts. Idiopathic isolated pulmonary arterial dilation is a disease of exclusion. Pseudoaneurysms are caused by iatrogenic injuries such as catheter misplacement or infection. Mycotic aneurysms are seen in septic emboli, tuberculosis or necrotizing pneumonia. Symptoms are usually non-specific and include dyspnea, cough, and chest pain. If the pulmonic valve is involved, pulmonic insufficiency may develop resulting in right heart failure. Rupture is a rare complication[50].

CT and MRI show focal saccular or fusiform dilation of the pulmonary artery. Thrombus and wall calcification can be seen. CT and MRI are useful for not only measuring the maximal size of the aneurysm, but also the extent, including whether or not the pulmonic valve is involved (Figure 24). Additionally, associated causative factors, if present, such pulmonary emboli and vasculitis or infectious may also be evaluated. Given the rarity of these aneurysms, treatment guidelines have not been established, although surgical repair has been performed in symptomatic cases or in cases of rupture.

SVC ANEURYSM

Among the most uncommon aneurysms within the thorax are those of the systemic venous system (Figure 25). A majority of the reports are aneurysms of the SVC, although only several dozen cases have been reported[51]. A majority of these published cases are fusiform aneurysms with saccular aneurysms being even less common. They can be primary, due congenital weakness in the wall of SVC due to deficiency in longitudinal adventitial muscular layer or may be associated with cystic hygroma. Complications of pulmonary emboli and rupture have been reported. Cross sectional imaging can reliably assess aneurysms morphology and presence of thrombus formation. Although experience is limited, treatment of fusiform aneurysms is generally conservative while saccular aneurysms are more likely to undergo surgical repair[51].

CONCLUSION

There is a wide spectrum of uncommon aneurysms of the cardiovascular system. CT and MRI are important imaging modalities in the evaluation of the aneurysms, the early diagnosis of which is important for early management and prevention of complications.

REFERENCES

1   **Agarwal V**, Yaliwal C, Ofo E, Kolvekar S. Giant ascending aortic aneurysm--a case report and review. *Heart Lung Circ* 2007; **16**: 385-388 [PMID: 17314066 DOI: 10.1016/j.hlc.2006.09.009]

2   **Shah P**, Gupta N, Goldfarb I, Shamoon F. Giant dissecting aortic aneurysm in an asymptomatic young male. *Case Rep Vasc Med* 2015; **2015**: 958464 [PMID: 25802796 DOI: 10.1155/2015/958464]

3   **Yalçın M**, Eroglu M, Isilak Z, Aparci M, Cebeci BS. Acute respiratory failure due to giant aortic aneurysm mimicking massive

pulmonary embolism in a patient with Marfan's syndrome. *Kardiol Pol* 2013; **71**: 1212 [PMID: 24297729 DOI: 10.5603/KP.2013.0312]

4   **Boulia SP**, Metaxas E, Augoulea M, Krassas AI, Balis E, Tatsis G. Superior vena cava syndrome due to giant aortic aneurysm. *Asian Cardiovasc Thorac Ann* 2010; **18**: 396-397 [PMID: 20719796 DOI: 10.1177/0218492310375622]

5   **Dayan V**, Michelis V, Lorenzo A. Giant aortic aneurysm as a rare cause of superior vena cava syndrome. *Ann Thorac Surg* 2008; **86**: 1383 [PMID: 18805209 DOI: 10.1016/j.athoracsur.2007.11.004]

6   **Lorusso R**, Coletti G, Totaro P, Maroldi R, Zogno M. Treatment of giant aortic aneurysm with tracheal compression and sternal erosion without circulatory arrest. *Ann Thorac Surg* 2000; **69**: 275-278 [PMID: 10654535]

7   **Hiratzka LF**, Bakris GL, Beckman JA, Bersin RM, Carr VF, Casey DE, Eagle KA, Hermann LK, Isselbacher EM, Kazerooni EA, Kouchoukos NT, Lytle BW, Milewicz DM, Reich DL, Sen S, Shinn JA, Svensson LG, Williams DM. 2010 ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM Guidelines for the diagnosis and management of patients with thoracic aortic disease. A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, American Association for Thoracic Surgery, American College of Radiology,American Stroke Association, Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, Society of Interventional Radiology, Society of Thoracic Surgeons,and Society for Vascular Medicine. *J Am Coll Cardiol* 2010; **55**: e27-e129 [PMID: 20359588 DOI: 10.1016/j.jacc.2010.02.015]

8   **Quevedo HC**, Santiago-Trinidad R, Castellanos J, Atianzar K, Anwar A, Abi Rafeh N. Systematic review of interventions to repair ascending aortic pseudoaneurysms. *Ochsner J* 2014; **14**: 576-585 [PMID: 25598723]

9   **Sullivan KL**, Steiner RM, Smullens SN, Griska L, Meister SG. Pseudoaneurysm of the ascending aorta following cardiac surgery. *Chest* 1988; **93**: 138-143 [PMID: 3257182]

10   **Finkelmeier BA**, Mentzer RM, Kaiser DL, Tegtmeyer CJ, Nolan SP. Chronic traumatic thoracic aneurysm. Influence of operative treatment on natural history: an analysis of reported cases, 1950-1980. *J Thorac Cardiovasc Surg* 1982; **84**: 257-266 [PMID: 7098511]

11   **Heystraten FM**, Rosenbusch G, Kingma LM, Lacquet LK. Chronic posttraumatic aneurysm of the thoracic aorta: surgically correctable occult threat. *AJR Am J Roentgenol* 1986; **146**: 303-308 [PMID: 3484580 DOI: 10.2214/ajr.146.2.303]

12   **von Kodolitsch Y**, Aydin MA, Koschyk DH, Loose R, Schalwat I, Karck M, Cremer J, Haverich A, Berger J, Meinertz T, Nienaber CA. Predictors of aneurysmal formation after surgical correction of aortic coarctation. *J Am Coll Cardiol* 2002; **39**: 617-624 [PMID: 11849860 DOI: 10.1016/S0735-1097(01)01784-3]

13   **Cullen EL**, Lantz EJ, Johnson CM, Young PM. Traumatic aortic injury: CT findings, mimics, and therapeutic options. *Cardiovasc Diagn Ther* 2014; **4**: 238-244 [PMID: 25009793 DOI: 10.3978/j.issn.2223-3652.2014.06.02]

14   **Agarwal PP**, Chughtai A, Matzinger FR, Kazerooni EA. Multidetector CT of thoracic aortic aneurysms. *Radiographics* 2009; **29**: 537-552 [PMID: 19325064 DOI: 10.1148/rg.292075080]

15   **Lee WK**, Mossop PJ, Little AF, Fitt GJ, Vrazas JI, Hoang JK, Hennessy OF. Infected (mycotic) aneurysms: spectrum of imaging appearances and management. *Radiographics* 2008; **28**: 1853-1868 [PMID: 19001644 DOI: 10.1148/rg.287085054]

16   **Malouf JF**, Chandrasekaran K, Orszulak TA. Mycotic aneurysms of the thoracic aorta: a diagnostic challenge. *Am J Med* 2003; **115**: 489-496 [PMID: 14563506]

17   **Sueyoshi E**, Sakamoto I, Hayashi K. Aortic aneurysms in patients with Takayasu's arteritis: CT evaluation. *AJR Am J Roentgenol* 2000; **175**: 1727-1733 [PMID: 11090411 DOI: 10.2214/ajr.175.6.1751727]

18   **Gornik HL**, Creager MA. Aortitis. *Circulation* 2008; **117**: 3039-3051 [PMID: 18541754 DOI: 10.1161/CIRCULATIONAHA.107.760686]

19   **Hollander JE**, Quick G. Aortoesophageal fistula: a comprehensive review of the literature. *Am J Med* 1991; **91**: 279-287 [PMID: 1892150]

20   **Dossa CD**, Pipinos II, Shepard AD, Ernst CB. Primary aortoenteric fistula: Part I. *Ann Vasc Surg* 1994; **8**: 113-120 [PMID: 8192994 DOI: 10.1007/BF02133413]

21   **Baker WH**, Sharzer LA, Ehrenhaft JL. Aortocaval fistula as a complication of abdominal aortic aneurysms. *Surgery* 1972; **72**: 933-938 [PMID: 5087277]

22   **Tonolini M**, Ippolito S, Rigiroli F. Images in medicine: Spontaneous aortocaval fistula complicating abdominal aortic aneurysm. *J Emerg Trauma Shock* 2014; **7**: 129-130 [PMID: 24812461 DOI: 10.4103/0974-2700.130888]

23   **Konen E**, Merchant N, Gutierrez C, Provost Y, Mickleborough L, Paul NS, Butany J. True versus false left ventricular aneurysm: differentiation with MR imaging--initial experience. *Radiology* 2005; **236**: 65-70 [PMID: 15955851 DOI: 10.1148/radiol.2361031699]

24   **Ba'albaki HA**, Clements SD. Left ventricular aneurysm: a review. *Clin Cardiol* 1989; **12**: 5-13 [PMID: 2643491]

25   **Lyons CJ**, Scheiss WA, Johnson LW, Parker FB. Surgical treatment of right ventricular aneurysm: an uncommon procedure. *Ann Thorac Surg* 1977; **23**: 221-224 [PMID: 849026]

26   **Vlodaver Z**, Coe JI, Edwards JE. True and false left ventricular aneurysms. Propensity for the altter to rupture. *Circulation* 1975; **51**: 567-572 [PMID: 1132088]

27   **Antman EM**, Anbe DT, Armstrong PW, Bates ER, Green LA, Hand M, Hochman JS, Krumholz HM, Kushner FG, Lamas GA, Mullany CJ, Ornato JP, Pearle DL, Sloan MA, Smith SC. ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction--executive summary. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to revise the 1999 guidelines for the management of patients with acute myocardial infarction). *J Am Coll Cardiol* 2004; **44**: 671-719 [PMID: 15358045 DOI: 10.1016/j.jacc.2004.07.002]

28   **Makkuni P**, Kotler MN, Figueredo VM. Diverticular and aneurysmal structures of the left ventricle in adults: report of a case within the context of a literature review. *Tex Heart Inst J* 2010; **37**: 699-705 [PMID: 21224951]

29   **Naidu A**, Ricketts M, Goela A, Shoemaker G, Li S. Incidental discovery of a membranous ventricular septal aneurysm in two dissimilar patients. *Case Rep Cardiol* 2012; **2012**: 324326 [PMID: 24826245 DOI: 10.1155/2012/324326]

30   **Miyake T**, Shinohara T, Nakamura Y, Fukuda T, Tasato H, Toyohara K, Tanihira Y. Aneurysm of the ventricular membranous septum: serial echocardiographic studies. *Pediatr Cardiol* 2004; **25**: 385-389 [PMID: 15054560 DOI: 10.1007/s00246-003-0572-6]

31   **Agmon Y**, Khandheria BK, Meissner I, Gentile F, Whisnant JP, Sicks JD, O'Fallon WM, Covalt JL, Wiebers DO, Seward JB. Frequency of atrial septal aneurysms in patients with cerebral ischemic events. *Circulation* 1999; **99**: 1942-1944 [PMID: 10208995]

32   **Mügge A**, Daniel WG, Angermann C, Spes C, Khandheria BK, Kronzon I, Freedberg RS, Keren A, Denning K, Engberding R. Atrial septal aneurysm in adult patients. A multicenter study using transthoracic and transesophageal echocardiography. *Circulation* 1995; **91**: 2785-2792 [PMID: 7758185 DOI: 10.1161/01.CIR.91.11.2785]

33   **Ghosh S**, Ghosh AK, Ghosh SK. Patent foramen ovale and atrial septal aneurysm in cryptogenic stroke. *Postgrad Med J* 2007; **83**: 173-177 [PMID: 17344571 DOI: 10.1136/pgmj.2006.051094]

34   **Hanley PC**, Tajik AJ, Hynes JK, Edwards WD, Reeder GS, Hagler DJ, Seward JB. Diagnosis and classification of atrial septal aneurysm by two-dimensional echocardiography: report of 80 consecutive cases. *J Am Coll Cardiol* 1985; **6**: 1370-1382 [PMID: 4067118]

35   **Díaz-Zamudio M**, Bacilio-Pérez U, Herrera-Zarza MC, Meave-González A, Alexanderson-Rosas E, Zambrana-Balta GF, Kimura-Hayama ET. Coronary artery aneurysms and ectasia: role of coronary CT angiography. *Radiographics* 2009; **29**: 1939-1954 [PMID: 19926755 DOI: 10.1148/rg.297095048]

36   **Newburger JW**, Takahashi M, Gerber MA, Gewitz MH, Tani

LY, Burns JC, Shulman ST, Bolger AF, Ferrieri P, Baltimore RS, Wilson WR, Baddour LM, Levison ME, Pallasch TJ, Falace DA, Taubert KA. Diagnosis, treatment, and long-term management of Kawasaki disease: a statement for health professionals from the Committee on Rheumatic Fever, Endocarditis and Kawasaki Disease, Council on Cardiovascular Disease in the Young, American Heart Association. *Circulation* 2004; **110**: 2747-2771 [PMID: 15505111 DOI: 10.1161/01.CIR.0000145143.19711.78]

37   **Le Breton H**, Pavin D, Langanay T, Roland Y, Leclercq C, Beliard JM, Bedossa M, Rioux C, Pony JC. Aneurysms and pseudoaneurysms of saphenous vein coronary artery bypass grafts. *Heart* 1998; **79**: 505-508 [PMID: 9659201]

38   **Ott DA**. Aneurysm of the sinus of valsalva. *Semin Thorac Cardiovasc Surg Pediatr Card Surg Annu* 2006: 165-176 [PMID: 16638563 DOI: 10.1053/j.pcsu.2006.02.014]

39   **Gonzalez JB**, Koul S, Sawardekar U, Bhat PK, Kirshenbaum KJ, Sukerkar AN. Images in cardiovascular medicine. Sinus of Valsalva aneurysms: a unique case of giant aneurysms involving all 3 sinuses. *Circulation* 2008; **117**: e308-e311 [PMID: 18413504 DOI: 10.1161/CIRCULATIONAHA.107.730325]

40   **Moustafa S**, Mookadam F, Cooper L, Adam G, Zehr K, Stulak J, Holmes D. Sinus of Valsalva aneurysms--47 years of a single center experience and systematic overview of published reports. *Am J Cardiol* 2007; **99**: 1159-1164 [PMID: 17437748 DOI: 10.1016/j.amjcard.2006.11.047]

41   **Bricker AO**, Avutu B, Mohammed TL, Williamson EE, Syed IS, Julsrud PR, Schoenhagen P, Kirsch J. Valsalva sinus aneurysms: findings at CT and MR imaging. *Radiographics* 2010; **30**: 99-110 [PMID: 20083588 DOI: 10.1148/rg.301095719]

42   **Epstein DA**, Debord JR. Abnormalities associated with aberrant right subclavian arteries-a case report. *Vasc Endovascular Surg* 2002; **36**: 297-303 [PMID: 15599481]

43   **Davidian M**, Kee ST, Kato N, Semba CP, Razavi MK, Mitchell RS, Dake MD. Aneurysm of an aberrant right subclavian artery: treatment with PTFE covered stentgraft. *J Vasc Surg* 1998; **28**: 335-339 [PMID: 9719329]

44   **Kedora J**, Grimsley B, Pearl G. Endovascular treatment of an aberrant right subclavian artery aneurysm with use of the Zenith iliac plug. *Proc* (Bayl Univ Med Cent) 2009; **22**: 144-145 [PMID: 19381317]

45   **Soylu E**, Harling L, Ashrafian H, Anagnostakou V, Tassopoulos D, Charitos C, Kokotsakis J, Athanasiou T. Surgical treatment of innominate artery and aortic aneurysm: a case report and review of the literature. *J Cardiothorac Surg* 2013; **8**: 141 [PMID: 23725538 DOI: 10.1186/1749-8090-8-141]

46   **Bower TC**, Pairolero PC, Hallett JW, Toomey BJ, Gloviczki P, Cherry KJ. Brachiocephalic aneurysm: the case for early recognition and repair. *Ann Vasc Surg* 1991; **5**: 125-132 [PMID: 2015182 DOI: 10.1007/BF02016744]

47   **Kieffer E**, Chiche L, Koskas F, Bahnini A. Aneurysms of the innominate artery: surgical treatment of 27 patients. *J Vasc Surg* 2001; **34**: 222-228 [PMID: 11496272 DOI: 10.1067/mva.2001.115807]

48   **Seguchi M**, Wada H, Sakakura K, Kubo N, Ikeda N, Sugawara Y, Yamaguchi A, Ako J, Momomura S. Idiopathic pulmonary artery aneurysm. *Circulation* 2011; **124**: e369-e370 [PMID: 21969321 DOI: 10.1161/CIRCULATIONAHA.111.029033]

49   **Deb SJ**, Zehr KJ, Shields RC. Idiopathic pulmonary artery aneurysm. *Ann Thorac Surg* 2005; **80**: 1500-1502 [PMID: 16181901 DOI: 10.1016/j.athoracsur.2004.04.011]

50   **Veldtman GR**, Dearani JA, Warnes CA. Low pressure giant pulmonary artery aneurysms in the adult: natural history and management strategies. *Heart* 2003; **89**: 1067-1070 [PMID: 12923030]

51   **Varma PK**, Dharan BS, Ramachandran P, Neelakandhan KS. Superior vena caval aneurysm. *Interact Cardiovasc Thorac Surg* 2003; **2**: 331-333 [PMID: 17670060 DOI: 10.1016/S1569-9293(03)00076-8]

Baishideng®