



Computed tomography angiography of congenital left-sided obstructive heart lesions: a pictorial essay

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ABSTRACT

Congenital left-sided obstructive heart lesions (LSOLs) are a group of rare but serious congenital heart diseases. They encompass a spectrum of structural abnormalities that can obstruct blood flow at any point from the left atrium to the aortic arch. They are broadly divided into two main categories. The first is left ventricular inflow obstruction, including cor triatriatum sinister and congenital mitral stenosis. The second is left ventricular outflow obstruction, including subvalvular aortic stenosis, bicuspid aortic valve, supra-ventricular aortic stenosis, coarctation of the aorta, aortic arch hypoplasia, and interrupted aortic arch. When stenosis involves both left ventricular inflow and outflow obstructions, the condition is called Shone's syndrome. Computed tomography angiography (CTA) is a valuable tool for the diagnosis, treatment planning, and follow-up of LSOLs. Our goal is to review the CTA manifestations of LSOLs to better understand these congenital heart lesions.

KEYWORDS

Congenital heart disease, left-sided obstruction, inflow obstruction, outflow obstruction, computed tomography, aortic arch syndromes

In congenital heart diseases (CHDs), left-sided obstructive lesions (LSOLs) are a group of structural abnormalities that can obstruct blood flow at any point from the left atrium (LA) to the aortic arch. This usually results in inadequate end-organ perfusion with potentially life-threatening implications. LSOLs are broadly divided into two main categories. The first is left ventricular inflow obstruction, which involves blockages from the pulmonary veins to the left ventricle. The second is left ventricular outflow obstruction, which refers to obstructions from the left ventricle into the aorta.

Although echocardiography remains the first-line imaging modality for CHD, limited acoustic windows often hinder its evaluation of extracardiac structures. Cardiac magnetic resonance imaging (MRI) avoids radiation and provides excellent functional data but is limited by long acquisition times and the need for pediatric sedation. In contrast, cardiac computed tomography (CT) angiography (CTA) delivers rapid, high-resolution three-dimensional (3D) imaging with a wide field of view, simultaneously evaluating the heart, coronary arteries, great vessels, and adjacent airways. By overcoming the limitations of echocardiography and MRI, CTA serves as an essential complementary tool for comprehensive preoperative planning. This pictorial essay reviews the characteristic CTA manifestations of complex CHD with LSOLs.

Technical considerations of computed tomography angiography in pediatric left-sided obstructive lesions

Pediatric cardiac CTA requires individualized protocols that balance diagnostic image quality with radiation protection. Ultra-fast scanning (dual-source or 320-row) combined with prospective electrocardiogram (ECG) gating minimizes motion artifacts, and techniques such as "feed-and-wrap" often eliminate the need for sedation. Contrast administration should be weight-based (1.5–2.0 mL/kg) and tailored to specific cardiac anatomy.¹ Iodinated contrast media, such as 320 or 350, are utilized. The guiding principles are as follows: select the largest

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catheter possible based on weight (22 gauge for < 10 kg, larger for older children) to allow higher injection rates (contrast and saline: 1.5–5 mL/s) that optimize bolus geometry, while always adjusting the rate according to the specific catheter type. CTA for pediatric aortic obstructive lesions requires an extended scan range from the thoracic inlet to below the renal arteries to evaluate collateral circulation fully. To prevent aortic pulsation and venous streak artifacts, prospective ECG gating (or ultra-fast high-pitch scanning) and ascending aorta contrast tracking are essential. Finally, advanced dose-reduction strategies—including low-kV protocols, iterative reconstruction, and emerging photon-counting CT—paired with multiplanar and 3D reconstructions enable precise visualization of arch geometry and stenosis severity for surgical planning.²

Left ventricular inflow obstruction

Left ventricular inflow obstruction encompasses conditions including cor triatriatum sinister (CTS) and congenital mitral stenosis (MS). It usually leads to higher atrial filling pressures and can predispose patients to pulmonary edema, which can ultimately lead to the development of pulmonary arterial hypertension and even right heart failure.

Cor triatriatum sinister

CTS accounts for 0.1%–0.4% of all CHDs.³ It is characterized by a fibromuscular membrane that partitions the LA into two compartments: a posterosuperior accessory chamber (AC), which receives the pulmonary veins, and an anteroinferior LA proper, which communicates with the left atrial appendage (LAA) and mitral valve. Associated defects, primarily atrial septal defect and anomalous pulmonary venous drainage, occur in 80% of cases (Figures 1, 2).³

ECG-gated CTA with multiplanar reformation (MPR) and 3D volume reconstruction

can accurately delineate the fibromuscular septum, its thickness, fenestrations, and the volume relationship between the AC and LA proper. Crucially, CTA plays a pivotal role in distinguishing CTS from key diagnostic mimickers, most importantly a supramitral ring. This differentiation relies on the anatomical position relative to the LAA: the CTS membrane is located proximal (superior) to the

LAA, whereas a supramitral ring lies distal (inferior) to the LAA, positioned just above the mitral valve annulus (Figure 3a, b). Additionally, CTA readily differentiates CTS from intra-atrial thrombi or masses, which manifest as localized, irregular filling defects rather than a linear septum traversing the atrial cavity. Imaging reports should specify membrane location, the number and diameter of

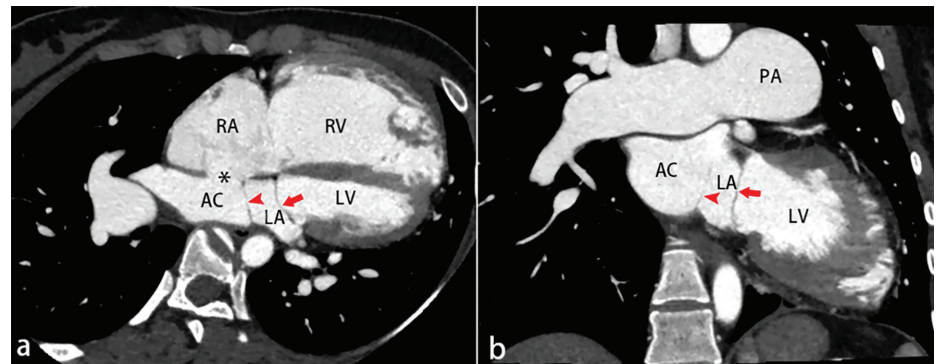


Figure 1. A 17-year-old girl with CTS. (a) Computed tomography angiography four-chamber view shows a thin, slightly hypodense membrane (red arrowhead) within the left atrium (LA). An atrial septal defect (asterisk) is also present. Due to the left-to-right shunt, there is secondary enlargement of the right atrium and right ventricle. The mitral valve (red arrow) is clearly visualized between the LA and the left ventricle. (b) Computed tomography angiography two-chamber view shows the membrane in the LA (red arrowhead). The pulmonary artery is markedly dilated, indicating pulmonary hypertension. CTS, cor triatriatum sinister; AC, accessory chamber; RA, right atrium; LV, left ventricle; RV, right ventricle; PA, pulmonary artery.

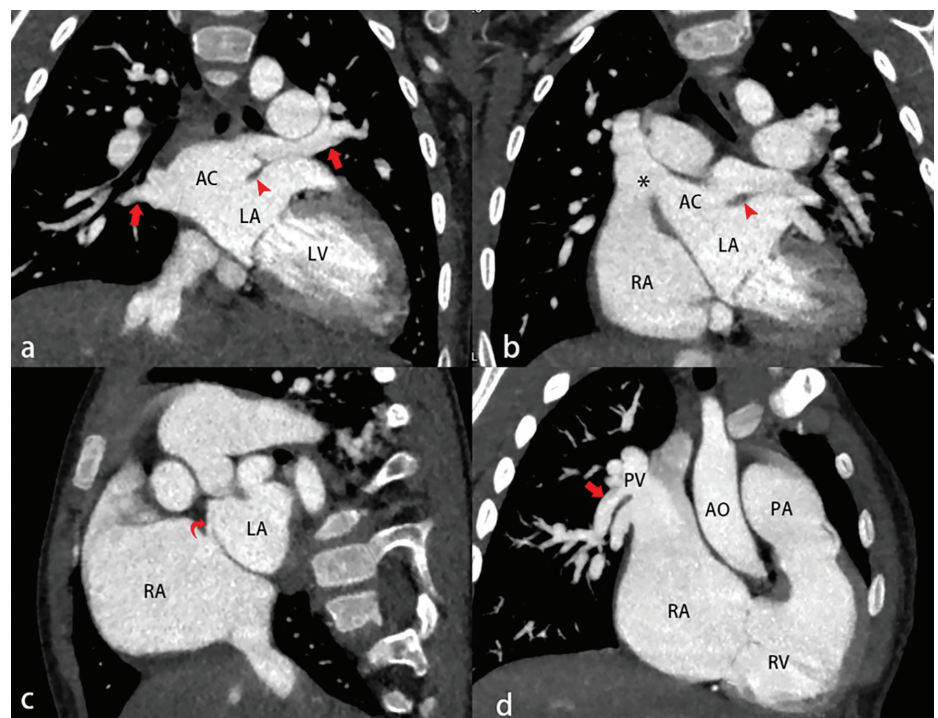


Figure 2. A 2-year-old boy with CTS, ASD, PFO, and abnormal pulmonary venous drainage. (a) Two-chamber view shows a thin, slightly hypodense membrane (red arrowhead) partially dividing the left atrium into an AC and the LA. The right inferior pulmonary vein and left pulmonary veins (red arrows) drain into the AC. (b) Discontinuity of the atrial septum, indicating an ASD (asterisk). (c) A tubular communication (red curved arrow) is seen between the left and right atria, suggesting a PFO. (d) The right superior pulmonary vein drains anomalously into the right atrium (red arrow). Additionally, right atrial enlargement and dilation of the main pulmonary artery are observed. CTS, cor triatriatum sinister; ASD, atrial septal defect; PFO, patent foramen ovale; AC, accessory chamber; LA, left atrium proper; RA, right atrium; LV, left ventricle; RV, right ventricle; PA, pulmonary artery; AO, aorta; PV, pulmonary vein.

Main points

- Left-sided obstructive heart lesions (LSOLs) encompass congenital obstructive diseases from the left atrium to the aortic arch, which are divided into left ventricular inflow obstruction and left ventricular outflow obstruction.
- LSOLs are usually associated with other congenital heart diseases, making their diagnosis more complex.
- Computed tomography angiography is valuable for the diagnosis and treatment planning of LSOLs.

fenestrations, pulmonary venous connections, and any associated anomalies. The definitive treatment for symptomatic CTS is surgical excision of the intra-atrial membrane.

Mitral stenosis

MS causes supravulvar, valvular, or subvalvular obstruction. Anatomically, Ruckman classifies it into typical (shortened chordae), hypoplastic, supramitral ring, and parachute types.⁴ Functionally, Carpentier distinguishes Type A (spared papillary muscles, including supramitral rings) from Type B (abnormal papillary muscles or chordae). Type B is the most common pediatric form, often causing mixed stenosis and regurgitation.⁵

Although echocardiography and MRI excel in hemodynamic assessment, ECG-gated cardiac CTA provides high-resolution, isotropic visualization of congenital MS subtypes. On CTA images, typical MS is characterized by shortened chordae tendineae, reduced inter-chordal spaces, and decreased inter-papillary distance (Figure 4). A supramitral ring appears as a thin fibromuscular ridge immediately superior to the mitral annulus, extending over the leaflets to restrict their motion (Figure 3a, b). Crucially, this ring must be differentiated from CTS based on its location distal (rather than proximal) to the LAA. This precise anatomical evaluation guides the surgical choice between valve repair (e.g., ring resection, chordal splitting) and replacement.⁶ Technical pitfalls, such as reconstruction phase mismatch (failing to use mid-diastole) or poor contrast opacification, can simulate or obscure stenosis.

Left ventricular outflow obstruction

Left ventricular outflow obstruction includes subvalvular aortic stenosis (SubAS), bicuspid aortic valve (BAV), supravulvar aortic stenosis (SupAS), coarctation of the aorta (CoA), aortic arch hypoplasia (AAH), and interrupted aortic arch (IAA). In this condition, the pressure load is imposed on the left ventricle, triggering compensatory hypertrophy, adverse remodeling, and even left heart failure.

Subvalvular aortic stenosis

SubAS represents approximately 14% of left ventricular outflow tract (LVOT) obstructions.⁷ The stenosis may be caused by a discrete fibrous membrane just below the aortic valve (90%), a thick fibromuscular narrowing, creating a tunnel-like outflow tract, or a combination of both.⁸

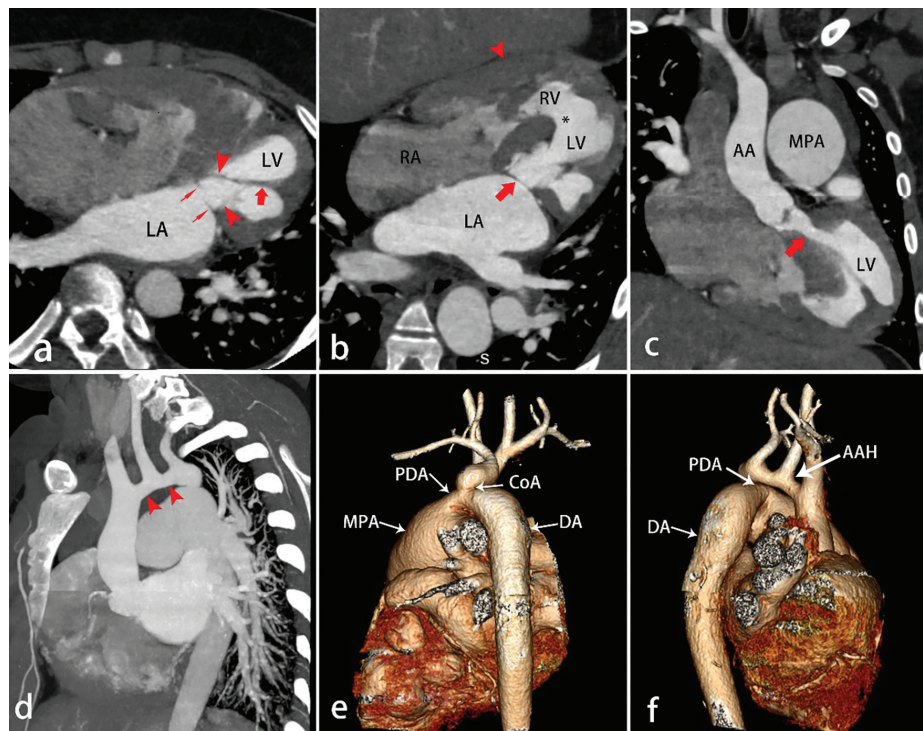


Figure 3. A 14-year-old boy with Shone's syndrome. Computed tomography angiography four-chamber view shows the parachute mitral valve (a, red arrow and arrowhead) and the supravulvar mitral ring (a, thin red arrow; b, red arrow). A ventricular septal defect (b, black asterisk) and thickened right ventricular wall (b, arrowhead) are also revealed. Oblique coronal CT angiography shows the subaortic stenosis (c, red arrow). Oblique sagittal maximum intensity projection image shows the aortic arch hypoplasia (d, red arrowhead). Volume-rendered CT angiography images show aortic arch hypoplasia, coarctation of the aorta and patent ductus arteriosus (e,f). CT, computed tomography; LA, left atrium; RA, right atrium; LV, left ventricle; RV, right ventricle; MPA, main pulmonary artery; AA, ascending aorta; DA, descending aorta; PDA, patent ductus arteriosus; AAH, aortic arch hypoplasia; CoA, coarctation of the aorta.

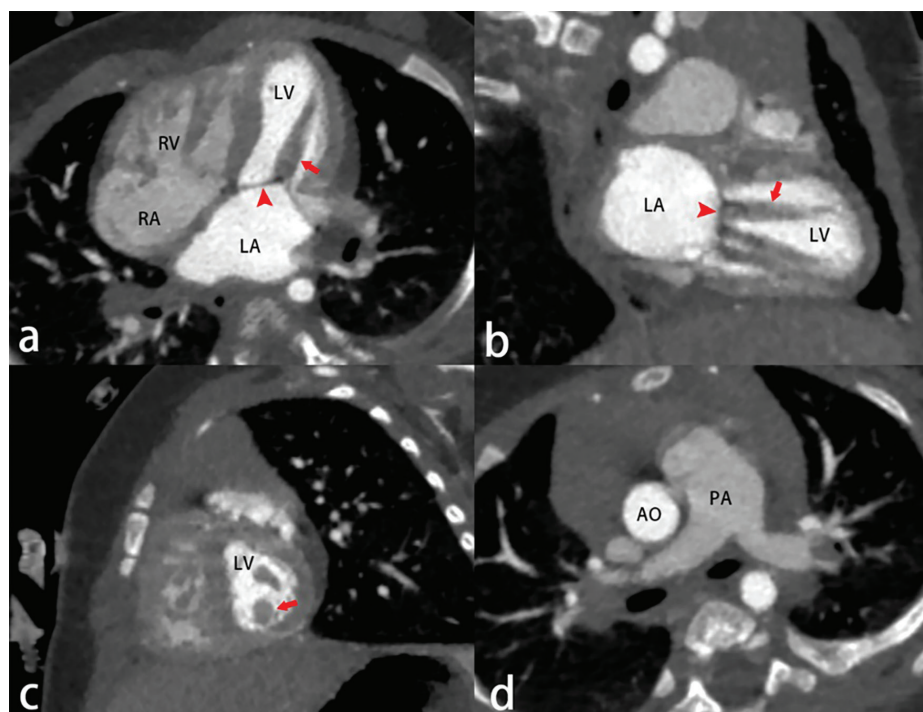


Figure 4. A 4-month-old girl with congenital mitral stenosis (MS). Computed tomography angiography four-chamber (a) and two-chamber (b) views show restricted opening of the mitral valve (red arrowhead), with thickened chordae tendineae attached to the leaflets (red arrow). The papillary muscles in the left ventricle are thickened (c). The main pulmonary artery is dilated (d). LA, left atrium; RA, right atrium; LV, left ventricle; RV, right ventricle; PA, pulmonary artery; AO, aorta.

ECG-gated cardiac CTA (optimally reconstructed in mid-systole) provides high-resolution imaging of SubAS, typically demonstrating a thin fibromuscular membrane (Figure 5), localized ridge, or tunnel-like narrowing (Figure 3c) in the LVOT. CTA also depicts associated CHDs, including BAV and ventricular septal defect (VSD). SubAS must be differentiated from other LVOT obstructive lesions, such as hypertrophic obstructive cardiomyopathy and valvular aortic stenosis. Hypertrophic obstructive cardiomyopathy can be distinguished from tunnel-like narrowing of SubAS by dynamic, asymmetric septal hypertrophy and systolic anterior motion of the mitral valve (Figure 6). Primary valvular stenosis can be identified by restriction and thickening/calcification of the valve leaflets themselves. Diagnostic pitfalls include motion artifacts or incorrect phase reconstruction, which can simulate or obscure thin membranes.

Surgical intervention is the definitive treatment for symptomatic SubAS. A surgically focused CTA report should highlight these critical elements: lesion morphology (discrete membrane vs. tunnel-type); anatomical safety margins relative to the aortic cusps; quantitative metrics of minimal LVOT area; aortic annulus size and associated anomalies; and secondary left ventricle hypertrophy and dilation.

Bicuspid aortic valve

BAV is one of the most common CHDs, with a population-based prevalence of

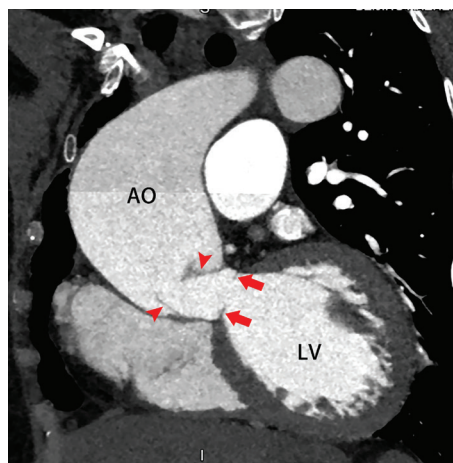


Figure 5. An 18-year-old woman with subvalvular aortic stenosis (fibrous membrane). The CT angiography oblique coronal image shows a thin, slightly hypodense membrane (red arrow) below the aortic valve (red arrowhead) in the left ventricular outflow tract, causing stenosis and secondary dilation of the left ventricle. An ascending aortic aneurysm is also present. CT, computed tomography; LV, left ventricle; AO, aorta.

0.5%–2.0%.⁹ Diagnostic key points center on identifying two-cusp morphology, commissural orientation, and classifying raphe. Two systems define its phenotypes: the Sievers classification categorizes by raphe presence (type 0: no raphe; type 1: one raphe; type 2: two raphe), and the Jilaihawi classification emphasizes three fusion patterns (tricom-

missural/functional, bicommissural raphe, and bicommissural non-raphe types) (Figure 7).¹⁰

ECG-gated cardiac CTA MPR aligned to the short-axis aortic valve plane in systolic/diastolic phases is essential to resolve motion artifacts and differentiate BAV from unicuspid or quadricuspid valves. On short-axis

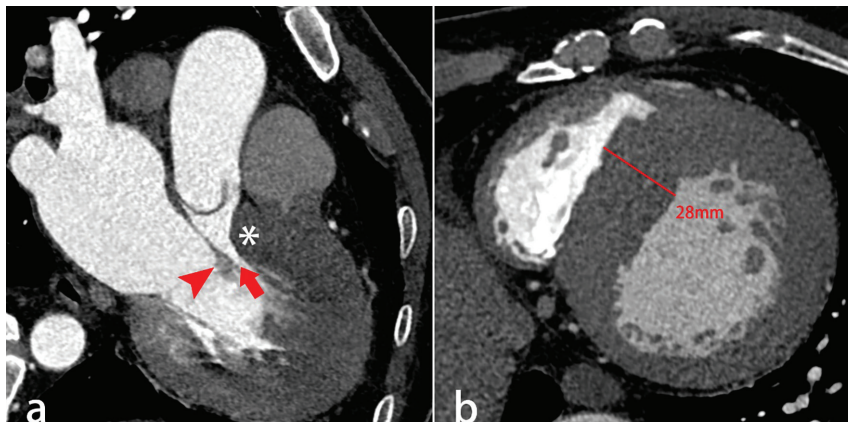


Figure 6. A 54-year-old man with hypertrophic obstructive cardiomyopathy. (a) Systolic cardiac CTA in the three-chamber view shows hypertrophy of the basal interventricular septum (white asterisk) protruding into the LVOT, accompanied by SAM of the mitral valve (red arrowhead), resulting in LVOT narrowing (red arrow). (b) Diastolic cardiac CTA in the short-axis view demonstrates a basal interventricular septal thickness of approximately 28 mm (red line). CTA, computed tomography angiography; LVOT, left ventricular outflow tract; SAM, systolic anterior motion.

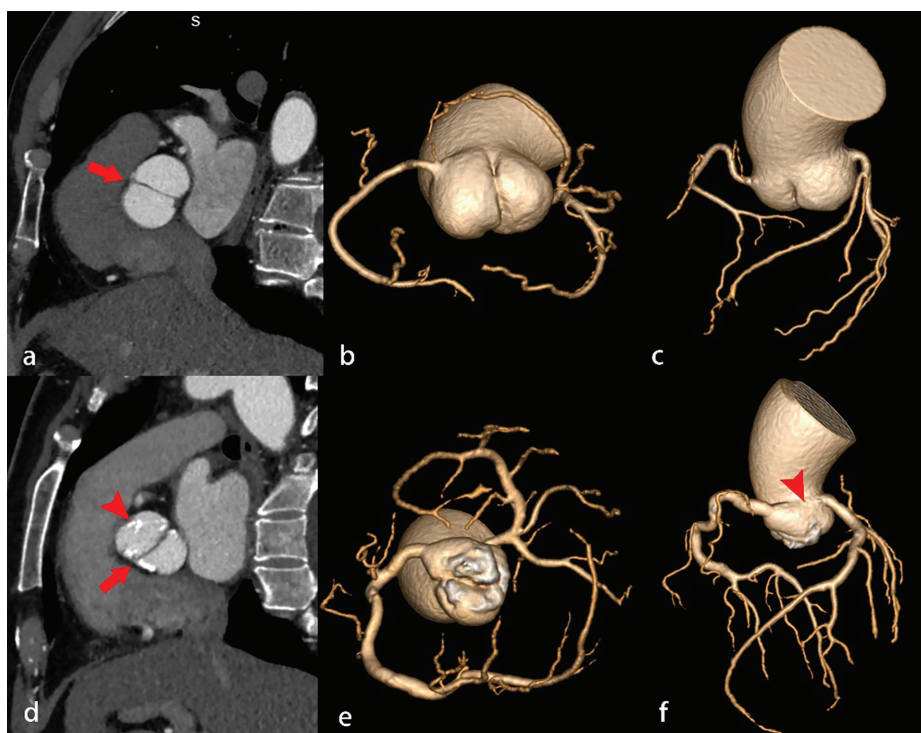


Figure 7. (a–c) A 62-year-old man with BAV. Computed tomography angiography oblique sagittal view shows a BAV with no raphe (Sievers classification, type 0; Jilaihawi classification, bicommissural non-raphe type) (a, red arrow). Volume-rendered CT angiography (b, c) demonstrates the left and right coronary arteries originating from separate sinuses of Valsalva. (d, e) A 54-year-old woman with BAV. Computed tomography angiography oblique sagittal view shows a BAV with one raphe (d, red arrowhead) (Sievers classification, type 1; Jilaihawi classification, bicommissural raphe type) and thickened, calcified leaflets (d, red arrow). Volume-rendered CT angiography (e, f) shows both the left and right coronary arteries arising from the anterior sinus. BAV, bicuspid aortic valve; CT, computed tomography.

images, the hallmark CTA features of BAV include a “fish-mouth” systolic orifice and a single diastolic commissural closure line (Figure 8). CTA comprehensively assesses secondary aortic and coronary alterations, including dystrophic calcification, infective endocarditis, aortopathy (annular-to-ascending aorta dilation, dissection, and rupture), and coronary origin anomalies. Compared with echocardiography and MRI, CTA offers excellent spatial resolution, precisely mapping calcification and coronary ostia to guide surgical repair versus replacement or valve-sparing root reconstruction.

Supravalvular aortic stenosis

Congenital SupAS, often genetically linked to Williams–Beuren syndrome, is an uncommon anomaly. Its hallmark is focal or diffuse narrowing at the sinotubular junction (STJ), classically appearing as an “hourglass” sign on CTA (Figure 9). This narrowing can also affect the peripheral arteries, such as stenosis or dilation in the pulmonary artery (Figure 10), renal artery, and coronary arteries. CTA provides superior 3D spatial resolution, offering a clear view of the distal aorta and complex peripheral branches. ECG-gated CTA is crucial for evaluating coronary abnormalities, particularly ostial stenosis in adults with Williams syndrome. Differential diagnosis should include valvular aortic stenosis, which features abnormal, thickened leaflets with a normal STJ, and aortic coarctation (Figure 11), where narrowing occurs at the aortic isthmus distal to the aortic arch. To relieve symptoms and preserve ventricular function, surgical repair is recommended for patients with symptomatic SupAS, alongside coronary revascularization if symptomatic ostial stenosis is present.¹¹

Coarctation of the aorta

CoA accounts for 4%–8% of CHDs. It features a discrete, shelf-like constriction near the arterial ligament (Figure 11), which often causes premature hypertension and abnormal pulse differences between the upper and lower extremities.

CTA can precisely detail the narrowing, post-stenotic dilation, enlarged collateral vessels (such as mammary and intercostal arteries), and associated anomalies, such as BAV. This detailed imaging easily separates true CoA from pseudocoarctation, which features aortic kinking but no real narrowing or collaterals (Figure 12).

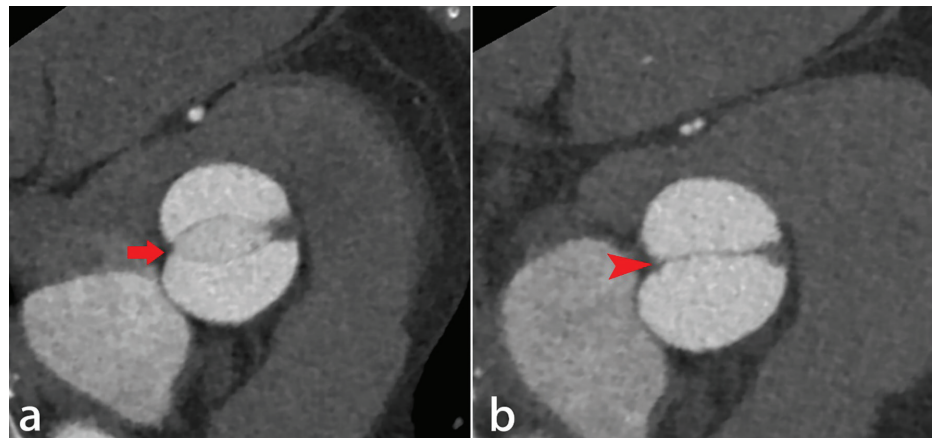


Figure 8. A 72-year-old woman with a BAV. (a) Systolic axial CTA image at the level of the aortic valve demonstrates aortic valve opening with a characteristic “fish-mouth” appearance (red arrow). (b) Diastolic axial CTA image at the level of the aortic valve shows aortic valve closure with a linear appearance (red arrowhead). BAV, bicuspid aortic valve; CTA, computed tomography angiography

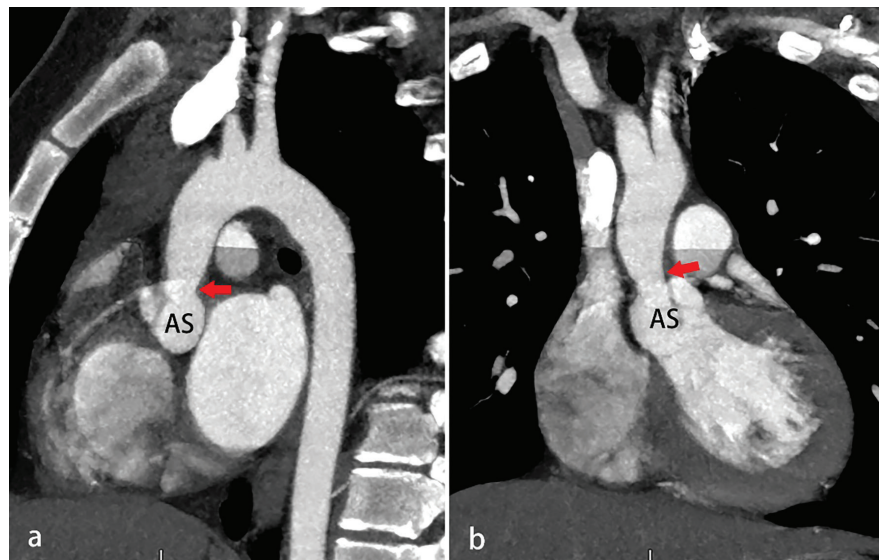


Figure 9. A 14-year-old boy with supravalvular aortic stenosis. Oblique sagittal (a) and coronal (b) MIP images from CT angiography demonstrate a markedly narrowed lumen at the sinotubular junction (red arrows), producing the typical “hourglass” appearance. MIP, maximum intensity projection; CT, computed tomography; AS, aortic sinus.

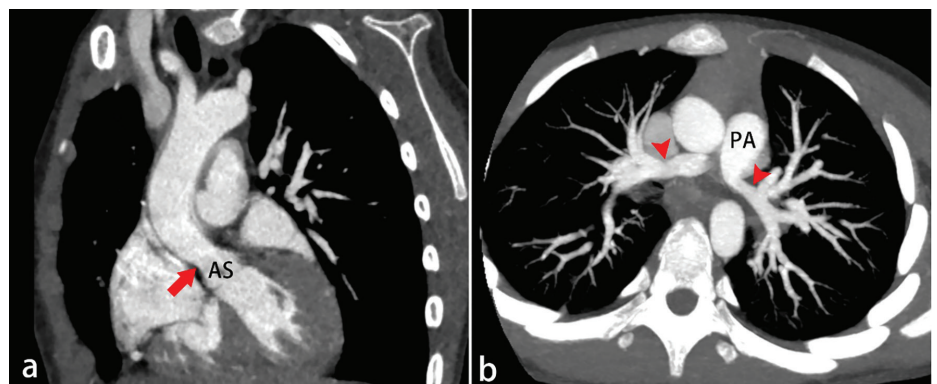


Figure 10. A 10-year-old girl with supravalvular aortic stenosis and pulmonary artery stenosis. Oblique coronal CT angiography (a) demonstrates a narrowed lumen at the sinotubular junction (red arrows). Oblique axial CT angiography (b) shows narrowing of the proximal left and right pulmonary arteries (red arrowheads). CT, computed tomography; AS, aortic sinus; PA, pulmonary artery.

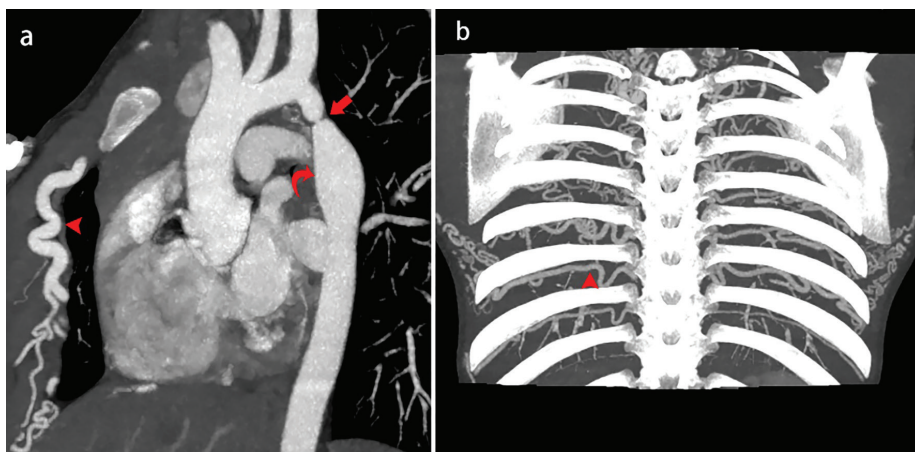


Figure 11. A 7-year-old boy with coarctation of the aorta. Oblique sagittal MIP image from CT angiography shows a significantly narrowed lumen at the juxtaductal region (a, red arrow). The adjacent descending aorta is dilated (a, red curved arrow). Dilation of the internal mammary artery (a, red arrowhead) and the intercostal arteries (b, red arrowhead) is observed, indicating formation of collateral circulation. MIP, maximum intensity projection; CT, computed tomography.

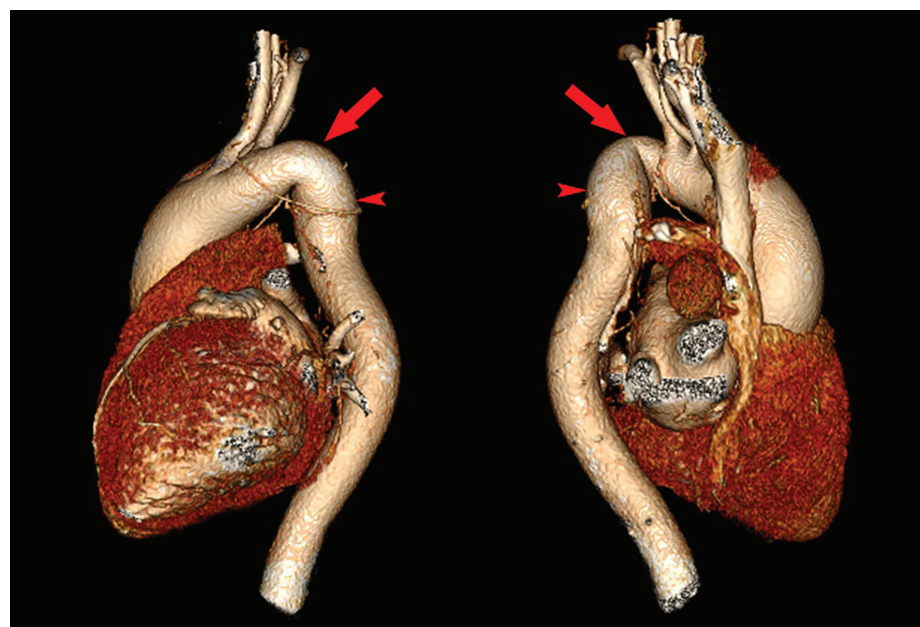


Figure 12. An 18-year-old man with pseudocoarctation of the aorta. Volume-rendered CT angiography shows elongation of the aortic arch, producing a twist or kink at the point of insertion of the ligamentum arteriosum (red arrow). The descending aorta just distal to the pseudocoarctation is mildly dilated (red arrowhead). No collateral vessel formation is present. CT, computed tomography.

It also distinguishes CoA from an IAA, where the aorta is completely disconnected. The management of CoA primarily involves surgical repair and transcatheter interventions.¹¹ CTA can precisely measure the stricture's length and minimal diameter and delineate its spatial relationship with the left subclavian artery and other aortic arch branches. The aortic isthmus ratio (minimal

CoA diameter/diaphragm-level descending aorta) should be measured, which defines severity, with a consensus cut-off of < 0.5 for repair.¹¹

Aortic arch hypoplasia

AAH is a rare CHD that usually occurs in neonates and infants. It is characterized by

a reduction in the external diameter of the aortic arch. It frequently coexists with anomalies such as VSD, CoA, or tetralogy of Fallot. AAH is commonly diagnosed using the following aortic diameter ratios relative to the ascending aorta: $< 60\%$ for the segment between the innominate and left common carotid arteries; $< 50\%$ between the left common carotid and left subclavian arteries; and $< 40\%$ at the aortic isthmus (Figure 13).¹² These criteria assume a normal ascending aorta diameter. CTA demonstrates continuous, uniform narrowing of the aortic arch, and MPR can precisely localize the affected segment. AAH should be differentiated from CoA (which shows a focal shelf-like narrowing with collaterals) and IAA (which presents as complete discontinuity). Treatment for AAH involves surgical reconstruction, such as patch augmentation or extended end-to-end anastomosis.¹³ CTA precisely maps the hypoplastic segment's length, diameter, and relationship to neck vessels.

Interrupted aortic arch

IAA is defined by a complete anatomical and luminal discontinuity between the ascending and descending aorta. The separation may be complete or connected by a remnant fibrous band. Most patients with IAA present with concomitant VSD or other CHDs, such as patent ductus arteriosus (PDA), and the descending aorta is mainly perfused via PDA. IAA is classified into three types: Type A is characterized by disruption distal to the left subclavian artery (Figure 14); Type B is characterized by disruption between the left common carotid and left subclavian arteries, and is the most common type (Figure 15); and Type C is characterized by disruption between the brachiocephalic and left common carotid arteries. A comprehensive CTA report must detail the exact site of interruption, the anatomical gap distance, the size of the PDA, and frequently associated anomalies. Differential diagnosis includes severe CoA and AAH, both of which maintain luminal continuity, unlike the absolute separation seen in IAA. CTA is vital for preoperative planning. By mapping the anatomical gap, branch origins, and associated defects, it dictates surgical strategies—such as direct anastomosis or conduit placement—ensuring a safe, single-stage repair.¹⁴

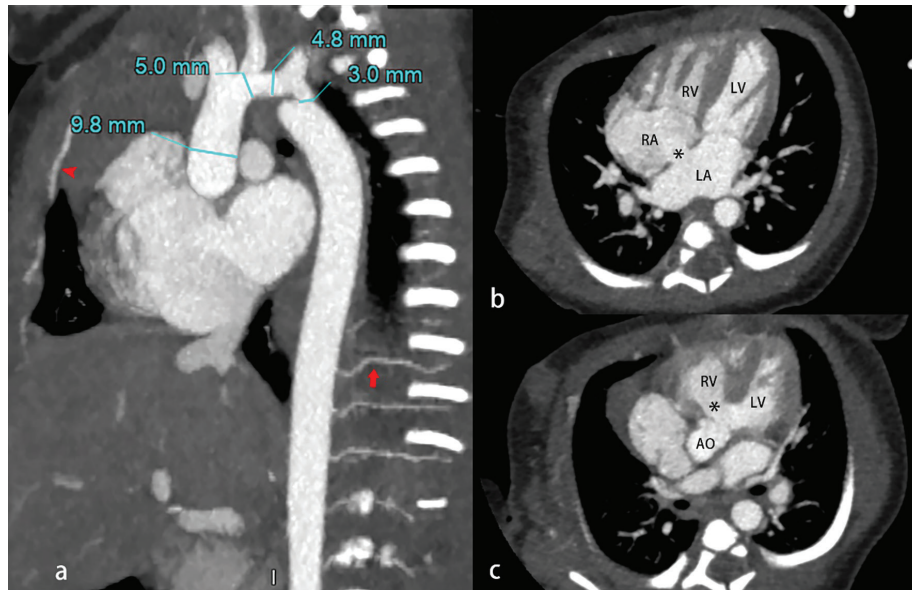


Figure 13. A three-day-old infant with aortic arch hypoplasia. Oblique sagittal MIP image from CT angiography shows a markedly narrowed aortic arch (a). The aortic diameter ratios relative to the ascending aorta are as follows: < 60% for the segment between the innominate and left common carotid arteries; < 50% between the left common carotid and left subclavian arteries; and < 40% at the aortic isthmus. Dilated internal mammary and intercostal arteries (red arrowheads) indicate formation of collateral circulation. The infant also has an atrial septal defect (b, asterisk) and a ventricular septal defect (c, asterisk). MIP, maximum intensity projection; CT, computed tomography; LA, left atrium; RA, right atrium; LV, left ventricle; RV, right ventricle; AO, aorta.

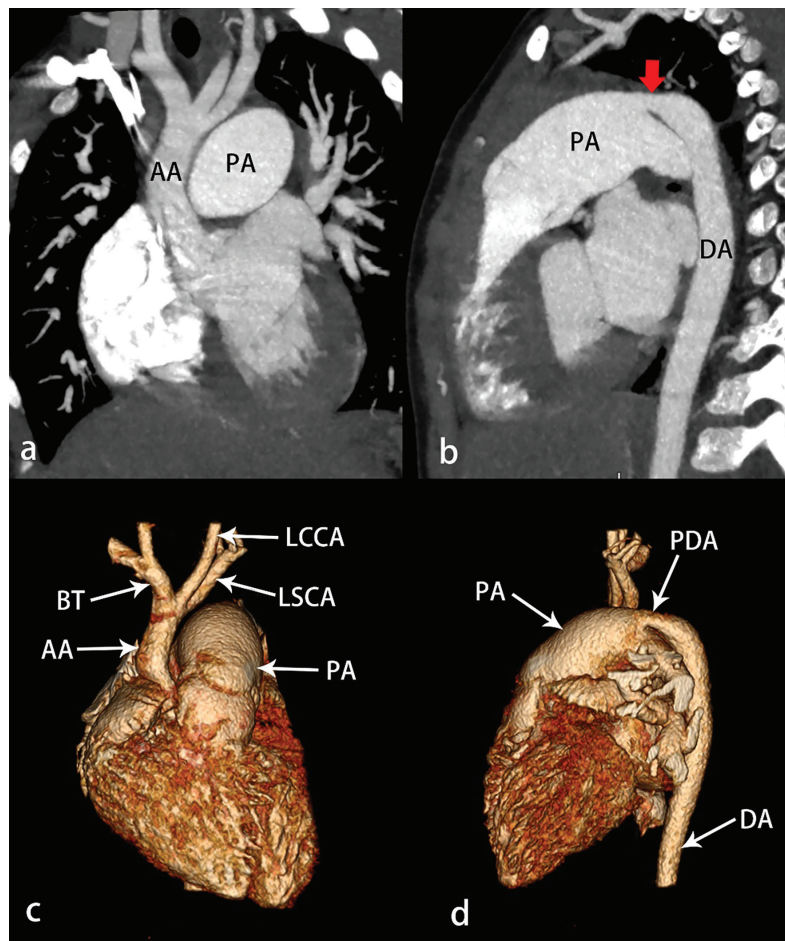


Figure 14. A 3-year-old girl with type A interrupted aortic arch. Oblique coronal CT angiography (a) and volume-rendered CT angiography (c) show disruption of the aortic arch distal to the left subclavian artery, with the ascending aorta giving rise to the brachiocephalic trunk, left common carotid artery, and left subclavian artery. Oblique sagittal CT angiography (b) and volume-rendered CT angiography (d) reveal that the descending aorta is supplied by the pulmonary artery via a patent ductus arteriosus (b, red arrow), with marked dilation of the pulmonary artery. CT, computed tomography; AA, ascending aorta; PA, pulmonary artery; DA, descending aorta; BT, brachiocephalic trunk; LCCA, left common carotid artery; LSCA, left subclavian artery; PDA, patent ductus arteriosus.

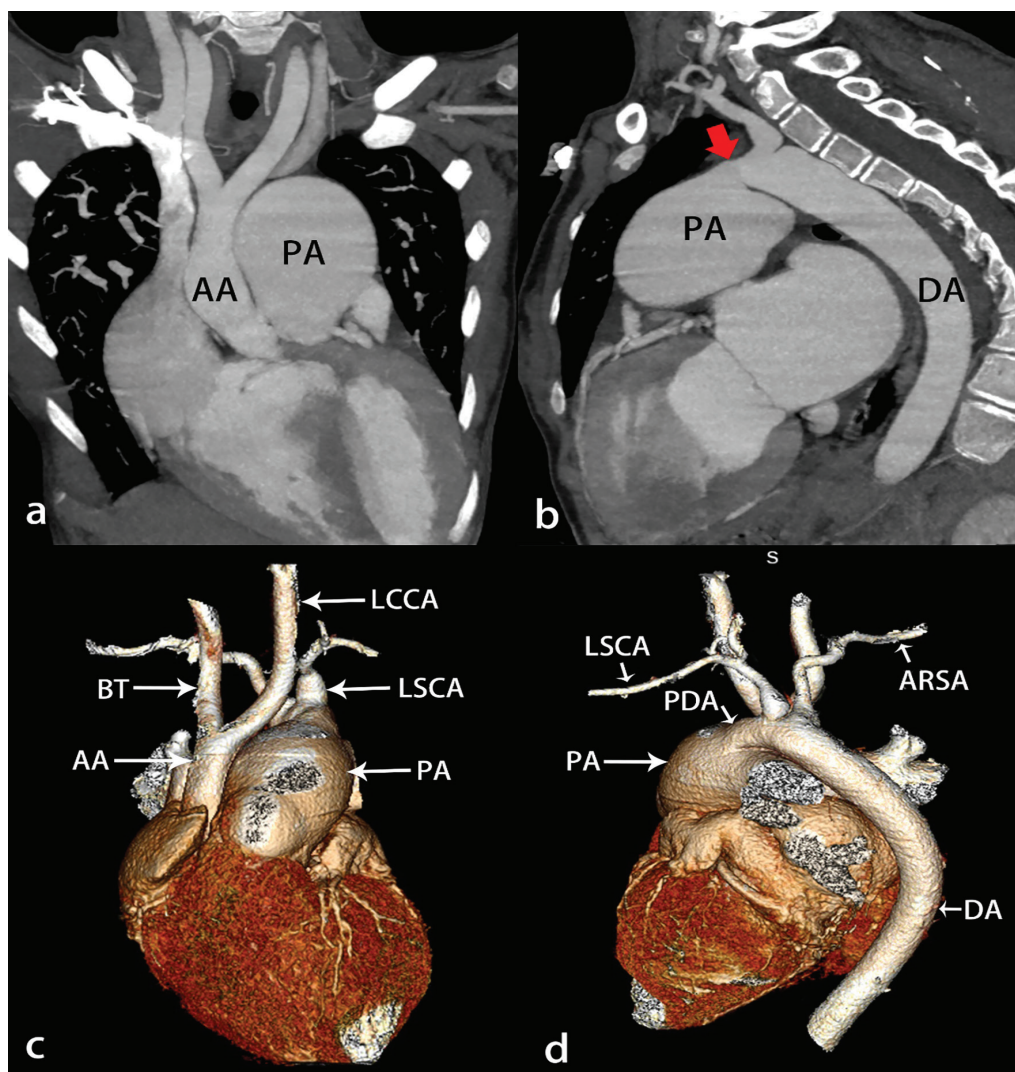


Figure 15. A 14-year-old girl with type B interrupted aortic arch. Oblique coronal CT angiography (a) and volume-rendered CT angiography (c) show interruption of the aortic arch between the left common carotid artery and the left subclavian artery. The ascending aorta gives rise only to the brachiocephalic trunk and the left common carotid artery. Oblique sagittal CT angiography (b) and volume-rendered CT angiography (d) show that the descending aorta is supplied by the main pulmonary artery via a patent ductus arteriosus (b, red arrow). The descending aorta gives rise to the left subclavian artery and an aberrant right subclavian artery. CT, computed tomography; AA, ascending aorta; PA, pulmonary artery; DA, descending aorta; BT, brachiocephalic trunk; LCCA, left common carotid artery; LSCA, left subclavian artery; ARSA, aberrant right subclavian artery; PDA, patent ductus arteriosus.

Shone's syndrome

Shone's syndrome is a rare congenital heart condition, accounting for approximately 0.6% of all CHDs.¹⁵ It is characterized by multi-level left-sided obstructions affecting both the inflow and outflow tracts of the left ventricle. The complete Shone's syndrome typically includes four anomalies: parachute mitral valve, supramitral ring, subaortic stenosis, and CoA (Figure 3). Incomplete form is often defined by at least one inflow lesion (e.g., parachute mitral valve, mitral ring, congenital MS) combined with at

least one outflow lesion (subaortic stenosis or aortic coarctation).

Diagnostic algorithm for left-sided obstructive lesions on cardiac computed tomography angiography

To prevent diagnostic oversight—especially in complex or multi-level congenital lesions—the cardiac CTA evaluation of LSOLs should follow a strict proximal-to-distal anatomical pipeline. Figure 16 provides a comprehensive overview of the CTA diagnostic flowchart for LSOLs, which can serve as a practical reference in clinical practice.

Congenital LSOLs encompass a spectrum of stenotic lesions that can obstruct blood flow at any point from the LA to the aortic arch. They are broadly divided into two categories: left ventricular inflow obstruction and left ventricular outflow obstruction. Pathological features and CTA characteristics for LSOLs are summarized in Table 1. Familiarity with the various types and CTA imaging features of LSOLs is crucial for accurate diagnosis, classification, and treatment planning.

Diagnostic Algorithm for LSOLs on Cardiac CTA

A proximal-to-distal anatomical pipeline to prevent diagnostic oversight in complex or multi-level congenital lesions.

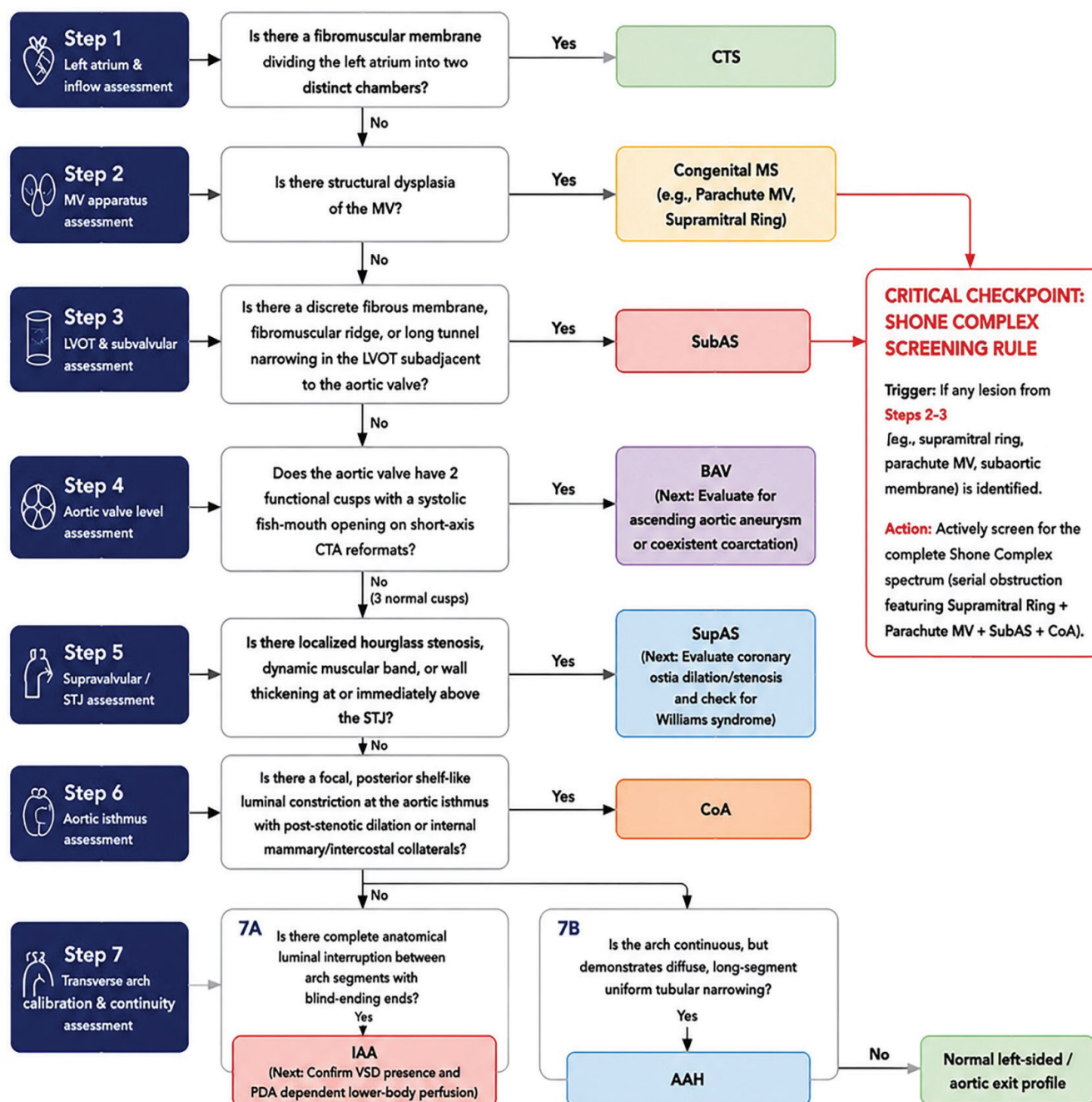


Figure 16. Diagnostic algorithm for LSOLs on cardiac CTA. CTS, cor triatriatum sinister; MS, congenital mitral stenosis; SubAS, subvalvular aortic stenosis; BAV, bicuspid aortic valve; SupAS, supravalvular aortic stenosis; CoA, coarctation of the aorta; AAH, aortic arch hypoplasia; IAA, interrupted aortic arch; MV, mitral valve; LVOT, left ventricular outflow tract; STJ, sinotubular junction; LSOL, left-sided obstructive lesions; CTA, computed tomography angiography.

Table 1. Pathological features and CTA characteristics for left-sided obstructive heart lesions			
Pathology	Pathological features	Characteristic CTA findings	Associated cardiovascular anomalies
CTS	Fibromuscular membrane divides the left atrium into a proximal and distal chamber.	Intra-atrial linear membrane dividing the LA.	ASD, anomalous pulmonary venous drainage.
MS	Congenital dysplasia of the mitral apparatus (leaflet thickening, chordal fusion, supramitral ring, parachute mitral valve).	Leaflet thickening with restricted opening; single prominent papillary muscle in Parachute mitral valve; supramitral ring.	Shone complex (often coexists with SubAS and CoA), VSD, ASD.
SubAS	LVOT obstruction caused by a discrete membrane, localized ridge, or fibromuscular tunnel.	Membranous; circumferential soft-tissue ridge; or fibromuscular tunnel in the LVOT; secondary concentric left ventricle hypertrophy.	VSD, BAV, Shone complex.
BAV	Congenital fusion of two aortic cusps, producing two functional leaflets instead of three.	"Fish-mouth" systolic orifice and single diastolic commissural closure line; raphe presence.	CoA, AAH, ascending aortic dilation, VSD.
SupAS	Focal or diffuse narrowing at the sinotubular junction.	"Hourglass" constriction/wall thickening at the sinotubular junction.	Williams syndrome, stenosis or dilation of pulmonary artery, renal artery, or coronary arteries, VSD.
CoA	Focal infolding of the aortic wall at the aortic isthmus near the ductus/ligamentum arteriosum insertion.	Discrete "shelf-like" luminal constriction at the isthmus with post-stenotic dilation.	BAV, AAH, VSD; tortuous collateral networks.
AAH	Long-segment tubular narrowing of the transverse aortic arch without focal disruption.	Uniform, long-segment tubular narrowing of the arch; absence of a discrete shelf.	CoA, VSD, BAV, Hypoplastic Left Heart hypoplastic left heart spectrum
IAA	Complete anatomical and luminal discontinuity between segments of the aortic arch.	Complete loss of arch luminal continuity; descending aorta supplied entirely by a large PDA.	VSD, PDA.
Shone's Syndrome	Rare congenital anomaly characterized by serial left-sided obstructive lesions: 1) Supramitral ring 2) Parachute mitral valve 3) Subaortic stenosis 4) Coarctation of the aorta	Multiphase CTA demonstrates two or more (incomplete) or all four (complete) classic lesions: 1) Supramitral ring 2) Parachute mitral valve 3) Subaortic stenosis 4) Coarctation of the aorta	BAV, VSD, AAH.
BAV obstructs only when stenotic. CTS, cor triatriatum sinister; MS, congenital mitral stenosis; SubAS, subvalvular aortic stenosis; BAV, bicuspid aortic valve; SupAS, supraaortic stenosis; CoA, coarctation of the aorta; AAH, aortic arch hypoplasia; IAA, interrupted aortic arch; LA, left atrium; ASD, atrial septal defect; VSD, ventricular septal defect; LVOT, left ventricular outflow tract; PDA, patent ductus arteriosus; CTA, computed tomography angiography.			

Footnotes

Conflict of interest disclosure

The authors declared no conflicts of interest.

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