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Radiology

KEY WORDS: Congenital heart disease, CT pulmonary angiography, pediatric heart disease, cyanotic and acyanotic heart disease.

ROLE OF CT PULMONARY ANGIOGRAPHY IN CONGENITAL HEART DISEASES IN PAEDIATRIC POPULATION

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ABSTRACT

Background: Heart defects are among the most common birth defects, occurring in 1% of live births (2-3% including bicuspid aortic valve). Multidetector computed tomography (CT) angiography is a fast method to evaluate vascular structural anomalies and CT can assess cardiac structures, thoracic vessels, the lungs and tracheobronchial tree in a single examination.
Aims: The purpose of this study is to describe the role of CT in the evaluation of congenital cardiovascular disease in children and anatomical anomalies in pulmonary vasculature.
Materials and methods: A retrospective study of CT pulmonary angiography was done in our department during the period of January to December 2017 for various congenital heart diseases.
Results and conclusion: 40 patients were included out of which 12 children had acyanotic heart disease and 28 had cyanotic heart disease. The anomalies studied included Tetralogy of Fallot (15), Pentalogy of Fallot (2), Hemitruncusarteriosus (1), transposition of great arteries (1), aortic coarctation (6), hypoplastic left heart (2), double outlet right ventricle (2), Interrupted aortic arch (2) and Total anomalous pulmonary venous return (3).
We suggest evaluation of complex cardiac anomalies primarily by CT pulmonary angiography study rather than only 2D Echo evaluation for better surgical outcome.

Heart defects are among the most common birth defects, occurring in 1% of live births (2-3% including bicuspid aortic valve). In 2013, 34.3 million people had congenital heart disease (CHD). In 2010, they resulted in 223,000 deaths, down from 278,000 deaths in 1990.

For congenital heart defects that arise without a family history (de novo), the recurrence risk in offspring is 3-5%. This risk is higher in left ventricular outflow tract obstructions, heterotaxy, and atrioventricular septal defects.

Echocardiography and catheter cardio angiography are the primary cardiac imaging modalities, but both have its own limitations. New generation Multidetector computed tomography (CT) and magnetic resonance (MR) imaging have important roles in overcoming these limitations. CT angiography is a fast method to evaluate vascular structural anomalies by fast image acquisition times and their capacity to obtain volumetric data. It is particularly useful for unstable patients, where rapid acquisition allows patientsto be in the scanner for only a short period of time. The use of and advances in ECG gating permits good assessment of cardiac chambers and coronary arteries with removal of cardiac pulsation artefact. Combined with isotropic spatial resolution, this allows assessment of even very small coronary arteries and other vascular structures. Good anatomical depiction is obtained within seconds/split-seconds. The pulmonary parenchyma is wellassessed with CT and can be interrogated in the same examination. Therefore with a single examination, CT can assess cardiac structures, thoracic vessels, the lungs and tracheobronchial tree.

For the complete evaluation of complex Congenital Heart Disease (CHD), high and spatial resolution is necessary, which is possible with CT and MR imaging. In this study, we will discuss and illustrate the CT findings (cardiac and extra cardiac abnormalities) in patients with CHD and the advantages and disadvantages of CT in this setting.

AIMS

The purpose of this study is to describe the role of CT in the evaluation of congenital cardiovascular disease in children and anatomical anomalies in pulmonary vasculature. Clinical indications, imaging techniques, and illustrations of relevant conditions are presented.

MATERIAL AND METHODS:

A retrospective study of CT pulmonary angiography was done in our department during the period of January to December 2017 for various congenital heart diseases. No gender bias was followed. Total number of cases included in this study is 40. CT scan was done on Multidetector scanner from Philips Brilliance 64 slice CT. Written informed consent was obtained. Breath holding technique was explained to the patient who was able to follow breath holding. Short acting anaesthesia was administered for pediatric patients.

1. Plain CT scans of thorax followed by
2. Early arterial phases: - Non-ionic contrast 12 to 40 ml was injected intravenously contrast media (lopamidol350mg) through cubital vein by pressure injector at a rate of 1.5 ml/sec. Scan was obtained 6 sec after starting of the intravenous contrast. Scan parameters were as follows:- Slice thickness- 1mm.Collimation:- 0.6 mm. Pitch: - 1.5, MAS=200, Kvp120.

Volumetric data was obtained from the vessels in axial plane and was reconstructed in sagittal, and coronal planes along with 3D reconstruction.

Extracardiac structures

The following vascular structures were evaluated: pulmonary arteries (including peripheral branches), thoracic aorta (throughout its length), superior and inferior vena cava, patent ductus arteriosus, pulmonary veins, airways, lungs and collateral circulation.

Observations:

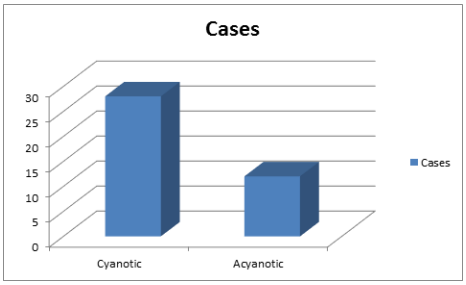
In total, 40 patients were included out of which there were 21 males (52%) and 19 females (48%).The oldest patient was 16 years old while the youngest patient was 1 day old.

Table 1: Age distribution

Age of presentation	Patient (%)
0 – 5	67.5
6 – 10	12.5
11-15	10
15-18	10

Majority of the patients presented between zero days to 5yrs.

12 children had acyanotic heart disease and 28 had cyanotic heart disease. There is a higher incidence of cyanotic heart diseases in our study.



Majority of the patients had situs solitus.(95 %)
However, only one patient had situs ambiguous - Large liver was seen crossing over to contralateral side of abdomen in central position. Spleen was not visualized. Stomach was seen to the right side of the spine and both the lungs were trilobed. These features are s/o right sided heterotaxy syndrome.

One patient had situs solitus with dextroposition.

Mild to moderate cardiomegaly was noted in almost all patients.

Dextrocardia was seen in 1 patient.

There was strong correlation between CT pulmonary angiographic findings and echo findings. Valvular pathologies were not evaluated by angiography. Detailed morphology of coarctation of aorta was better depicted by CT angiography.

VSD was seen in 27 (67.5 %) patients while ASD was seen in 9 (22.5%) patients.

PDA was seen in 14 (35%). MAPCAs were seen in 13 (32.5%) patients.

Left sided aortic arch was seen in most of the patients (87.5%).
The other variations seen included:

1. Right sided arch with normal branching patternn = 4(10 %)
2. Right sided arch with mirror branching- n = 1 .
3. First branch - common origin of left and right CCA. Second branch - left subclavian A. Third branch – Aberrant right subclavian artery.

Only 9 out of the 40 children (22.5 %) had lung changes in the form of consolidation.

3 patients had anomalies in the pulmonary veins (total anomalous pulmonary venous return.)

20 patients had anomalies of the pulmonary artery of which 9 patients had dilated main pulmonary artery suggestive of pulmonary arterial hypertension.

11 patients had features pulmonary arterial hypoplasia in which the pulmonary artery was narrowed or not visualized.

Double SVC was seen in 3 patients.

Table 2: The types of cardiac malformations seen in our study were:

Study	No of cases
Tetralogy of Fallot	15
Pentalogy of Fallot	2
Truncus arteriosus	0
Hemitruncusarteriosus	1
transposition of great arteries	1
aortic coarctation	6
hypoplastic left heart	2
DORV	2
Interrupted aortic arch	2
Partial anomalous pulmonary venous return	0
Total anomalous pulmonary venous return	3{Cardiac type 0, Supracardiac type 2, Infracardiac type 1}
Interrupted aortic arch	2

Discussion :

AORTA

COARCTATION OF AORTA –

It was first described by Morgagni in 1760. Coarctation refers to a stenosis of the proximal descending thoracic aorta that is almost always opposite the insertion of the ductus arteriosus—that is, at the junction of the distal aortic arch and the descending aorta just below the origin of the left subclavian artery (Fig 3a). Coarctation at or immediately proximal to the left subclavian artery is rare and compromises that vessel. The aorta just distal to the coarctation is typically dilated. CT is an effective and rapid imaging modality for the morphologic assessment of vessels, determination of the degree of stenosis, and visualization of collateral vessels [1] (Fig 3b). Uniform narrowing of the aortic arch (tubular hypoplasia) can be more frequently observed in neonates. A localized coarctation and tubular hypoplasia may coexist or may occur independently. [2]Coarctation is commonly associated with PDA and VSD, and the triad of these three has been named as coarctation syndrome. We found 6 cases of coarctation of aorta.

INTERRUPTED AORTIC ARCH-

Interruption of the aortic arch (IAA) is a congenital anomaly characterized by complete discontinuity of blood flow between two portions of the aorta. This malformation may exist as a long-distance physical separation between adjacent segments or in the form of discontinuity between adjacent lumens of vessels that are otherwise connected externally. More often it is considered as a variant of coarctation of Aorta.[3]

There are three major types of interrupted aortic arch:

- Type A: Aortic interruption between the left subclavian artery and the descending aorta.
- Type B: Aortic interruption between the left subclavian artery and the left common carotid artery.
- Type C: Aortic interruption between the left common carotid artery and the innominate artery.

We found 2 cases of interrupted aortic arch.

GREAT VESSELS

Truncus Arteriosus-

In truncus arteriosus, a single arterial trunk arises from the ventricle via a single arterial valve to supply the systemic, pulmonary, and coronary arterial circulations.[4] Aortopulmonary window is a communication between the ascending aorta and the pulmonary trunk in the presence of separate aortic and pulmonary valves . This defect is classified as proximal, distal, or total depending on its location.[5]

Subtypes: Collett and Edwards classification-

- Type I = pulmonary trunk arises from proximal truncus.
- Type II and III= no pulmonary trunk; branch PA arise from posterior and lateral mid-segments of truncus.
- Type IV=pulmonary circulation dependent on MAPCAs.

Hemitruncus arteriosus: It is a rare congenital cardiac anomaly characterized by the anomalous origin of one of the branch pulmonary arteries (PA) from the ascending aorta and a normal

origin of the other PA from the right ventricular outflow tract (RVOT) in the presence of two semilunar valves(Fig 7a & 7b). The lung connected to the normally arising PA receives the entire cardiac output from the right ventricle, while the other lung is exposed to both pressure and volume overload due to unrestricted shunting from the aorta.[6]

We found 1 case of hemitruncus arteriosus in our study.

TGA-

Transposition of the great arteries TGA is the second most common cyanotic heart defect after TOF. Complete transposition of the great arteries is a combination of atrioventricular concordance and ventriculoarterial discordance (ie, the ascending aorta arises from the right ventricle and the pulmonary artery from the left ventricle). The systemic and pulmonary circulations are parallel and independent closed circuits. Therefore, the blood between the two parallel circulations should be mixed through the VSD, PDA, or atrial septal defect to prevent severe cyanosis and metabolic acidosis.

It is subdivided into simple and complex TGA. Depending on the position of the aorta, it is also subdivided into dextro (D) and levo (L or congenitally corrected (cc) TGA.

We found 1 case of TGA in our study.
Patent ductusarteriosus (PDA)

The PDA lies between the proximal descending aorta and the origin of the left pulmonary artery(Fig 6a & 8g). Some conditions are duct dependant and therefore treatment is give to maintainduct patency, but it can remain patent in pathological circumstances either in isolation oras part of other complex congenital cardiac conditions. This should therefore be a pointof review for any congenital cardiovascular CT.

Cardiac:
Left predominant:
HYPOPLASTIC LEFT HEART SYNDROME-

Hypoplastic left heart syndrome (HLHS) refers to a spectrum of cardiac abnormalities that includes underdevelopment of the left ventricle, mitral valve, aorta, and aortic valve. This syndrome is the most severe form of left-sided obstructive lesions, and it is among the most severe forms of congenital heart disease (CHD). It consists, in varying degrees, of a small left ventricle associated with aortic atresia, a hypoplastic ascending aorta, mitral valve atresia or hypoplasia, and a small left atrium (Fig 6).

We found 2 cases of HLHS in our study.

Right predominant:
TOF-

TOF represents the most common cyanotic cardiac disease, accounting for almost 12% of all congenital heart anomalies. The morphologic features of tetralogy of Fallot include (a) subpulmonary infundibular stenosis (Fig 1a), (b) ventricular septal defect (VSD), (c) overriding of the aorta, and (d) right ventricular hypertrophy (Fig 1c). Anterosuperior deviation of the infundibular septum is considered the developmental cause for subpulmonary infundibular stenosis and VSD in tetralogy of Fallot. Hypertrophy of the infundibular septum and anterior muscle bundles contributes to pulmonary outlet obstruction in tetralogy of Fallot. Hypoplasia of the pulmonary valve and main pulmonary artery is common.

Several other abnormalities may occur in association with tetralogy of Fallot, including a right aortic arch in 25% of cases, an atrial septal defect in 10% of cases (so-called pentalogy of Fallot), and coronary artery anomalies in another 10% of cases.[7]

We found 15 cases of TOF in our study.

PULMONARY ATRESIA WITH VSD -

When pulmonary atresia with VSD is seen with atrioventricular and ventriculoarterial concordance, the lesion shows the morphologic characteristics of extreme tetralogy of Fallot . There is no pulmonary blood flow from either ventricle. Detailed morphologic evaluation of the central pulmonary artery and various pulmonary arterial feeding vessels is important for surgical planning . The central pulmonary artery may be either absent or present, and the branch pulmonary arteries may be either confluent or nonconfluent. The source of pulmonary blood flow may be multifocal, with a PDA or major aortopulmonary collateral vessels that supplement or replace primary pulmonary arterial blood flow. This source of pulmonary blood flow significantly affects the condition of the pulmonary vasculature and parenchyma.[2]

DORV-

In double outlet right ventricle, both great arteries are connected to the morphologic right ventricle. In the determination of a ventriculoarterial connection, an artery is regarded as connected to a ventricle when more than half its semilunar valve is connected to that ventricle (the "50% rule"). Double outlet right ventricle may occur with any atrial arrangement, atrioventricular connection, or ventricular topology.[8]

Loss of continuity between the mitral valve and the neighboring semilunar valve is prerequisite for the diagnosis. A VSD is always present and may be subaortic (50%), subpulmonary (30%), noncommitted, or doubly committed.

4 Subtypes-

1. (based on location of VSD in relation to great arteries) DORV with subaortic VSD blood from LV flows via VSD to aorta and blood from RV flows mainly to PA= physiology similar to VSD
2. DORV with subpulmonic VSD (Taussig-Bing syndrome) blood from LV flows via VSD to PA and blood from RV flows mainly to aorta= physiology similar to TGA.
3. DORV with doubly committed VSD absent infundibular septum.
4. DORV with non-committed VSD, VSD remote from aortic and pulmonary valves.

EBSTEINS ANOMALY-

Ebstein anomaly is defined as displacement of the attachment of the tricuspid valve leaflets from the atrioventricular junction to the right ventricular cavity with resultant atrialization of the inlet of the right ventricle. Displacement of the tricuspid valve attachment almost always involves only the septal and posterior leaflets and is maximal at the commissure between these two leaflets. The nondisplaced anterior leaflet is usually large and redundant, with a mobility that varies depending on the degree of tethering to the right ventricular wall.[9]

Septal defects:
ASD

There are four types of ASDs, secundum ASDs (most frequent), ostium primum defect, sinus venosus, and coronary sinus (least frequent).[10] The latter two (sinus venosus and coronary sinus sub type) do not involve a developmental abnormality of the septum as such but physiologically behave as inter ASDs. Sinus venosus ASDs are associated with partial anomalous pulmonary venous drainage. Therefore, if this type of ASD is demonstrated on echo, aCT may be done to assess for anomalous pulmonary veins.

VSD

VSDs may occur anywhere, but are most commonly found at points of junction of the embryologic components of the septum (i.e., the main [or posterior] ventricular septum, the bulbar [or infundibular] septum, and the membranous septum). By dividing the septum into four components (membranous septum, inlet, trabecular septum, and outlet or infundibular septum), VSDs may be classified into four main categories according to their location and the appearance of the margins of defects.

ATRIOVENTRICULAR CANAL DEFECTS-

Atrioventricular septal defect (AVSD) refers to a spectrum of cardiac malformations that include abnormalities of the interatrial septum, the interventricular septum, and the atrioventricular (AV) (mitral and tricuspid) valves. An AVSD results from the endocardial cushions of the heart failing to fuse properly. AVSDs are also

referred to as AV canal defects or endocardial cushion defects. AVSDs are usually categorized as partial, intermediate, or complete.

Veins:

Systemic veins:

Left SVC is more common in patients with congenital heart disease, but patients often also have a right SVC, which may or may not communicate via an innominate vein. Most left SVCs drain to the coronary sinus and then to right atrium, but occasionally they drain to the left atrium and cause hypoxia.

Inferior systemic veins:

Most notably, in heterotaxy syndromes and left sided isomerism, the inferior vena cava (IVC) can be interrupted with azygos or hemiazygos continuity.

Pulmonary veins:

PAPVC-(partial anomalous pulmonary venous connection) The pulmonary veins from some portions of both lungs show an anomalous connection. Partial pulmonary anomalous veins are often found incidentally or in association with other cardiac anomalies like ASD. When shunt is small there are no significant hemodynamic changes. In larger connections significant left-right-shunting may occur. The usual sites of connection are the SVC and the right atrium. Scimitar syndrome (hypogenetic lung or pulmonary venolobar syndrome) is a rare form of PAPVC in which right lower and sometimes middle lobe forms a vein that runs downward and medially in crescent course passing through diaphragm to enter IVC.[2]

Subtypes-

- Left pulmonary veins drain either into left innominate vein or coronary sinus. Associated with ASD.
- Right pulmonary veins may drain into SVC (associated with sinus venosus ASD) or into IVC (Scimitar syndrome or venolobar sequestration).

TAPVC-

Total anomalous pulmonary venous connection is characterized by connection of the pulmonary veins from both lungs to form a confluence behind the left atrium and connection of a venous channel from this confluence to a systemic vein, the right atrium, or both (Fig 5). Total anomalous pulmonary venous connection is described as supracardiac, cardiac, infracardiac, or mixed depending on the site or sites of connection. Supracardiac and cardiac types are rarely obstructive, but bilateral infracardiac types are almost always obstructive because the blood passes through the hepatic sinusoids.[2]

We found 3 cases of TAPVC in our study.

Other findings

There can be many additional associated congenital abnormalities, which should bereviewed at time of scanning, for example duplication cysts and oesophageal atresia. Vertebral anomalies (Fig 9a) may also be demonstrated on congenital cardiac CTs, particularlyin children with the VACTREL syndrome.

Inference

The review of literature of pulmonary angiography studies for congenital heart disease have evaluated the cardiac morphology, however, have not indicated the incidence rate. Our current study of pulmonary angiography in complex cardiac anomalies have evaluated the cardiac anomalies as well as presented the incidence rate of the various complex cardiac anomalies. Not all the congenital heart disease could be presented in our study – limitation due to small sample size and also isolated septal defects were not included in the study as the diagnosis was primarily made on 2D echo. None of these patients (septal defects) had undergone CT pulmonary angiography. However, the role of CT angiography with advent of faster scanning machines cannot be entirely ruled out in evaluation of congenital heart disease. We suggest evaluation of complex cardiac anomalies primarily by CT pulmonary angiography study rather than only 2D Echo evaluation for better surgical outcome.

Figure 1: TOF

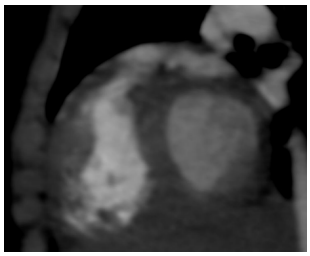


Figure 1a. Sagittal reformatted image in a case of Tetraology of Fallot reveals infundibular pulmonary stenosis.

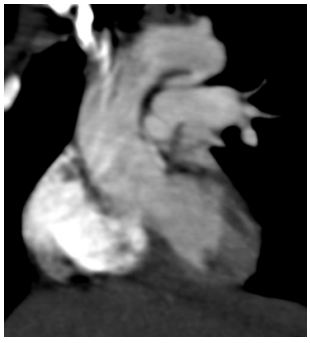


Figure 1b: Oblique coronal reformatted image in a case of TOF reveals patent ductus arteriosus.

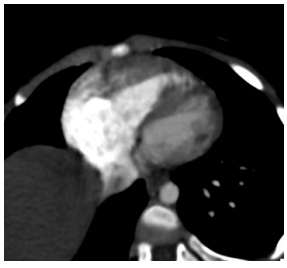


Figure 1c: Axial image in a case of TOF reveals right ventricular hypertrophy.

Aortopulmonary window

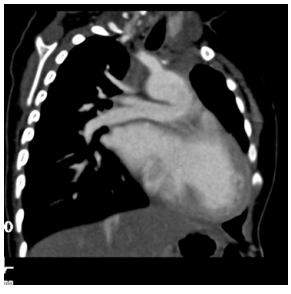


Figure 2a: Oblique coronal reformatted image reveals wide communication between arch of aorta and main pulmonary artery suggestive of aortopulmonary window.

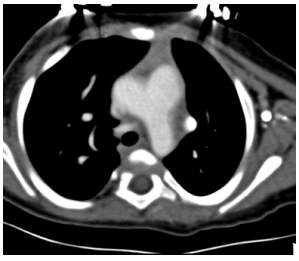


Figure 2b: Axial image in the same patient reveals the

aortopulmonary window.

Coarctation of aorta

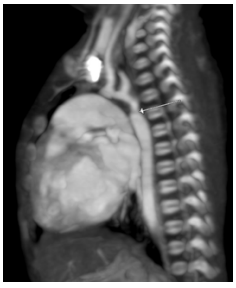


Figure 3a : MIP image in a case of coarcatation of aorta reveals short segment narrowing seen involving arch of aorta distal to left subclavian artery with poststenotic dilatation of descending aorta.

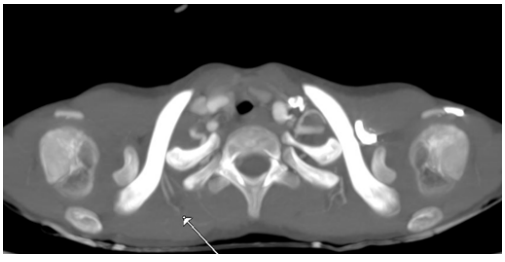


Figure 3b: MIP image in another case of coarctation reveals posterior intercostals collaterals (arrow).

Infracardiac TAPVR:

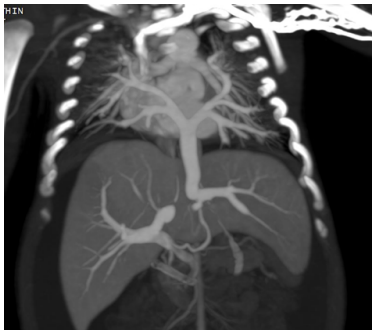


Figure 4a: MIP coronal image of infracardiac TAPVR showing confluence of superior and inferior pulmonary veins draining into the vertical vein (blue arrow)seen traversing inferiorly through posterior mediastinum and through hiatus for esophagus in diaphragm and continues as a venous channel in the left lobe of liver.

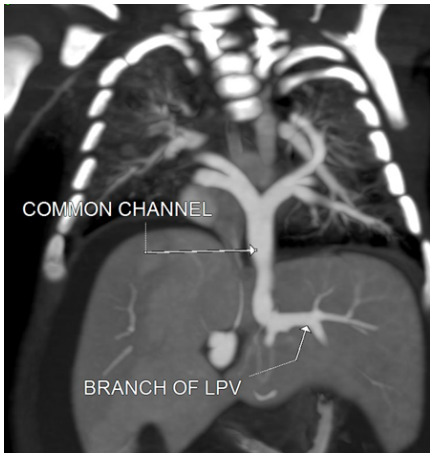


Figure 4b: MIP coronal image of the same patient showing that the venous channel is communicating with left portal vein.

Supracardiac TAPVC

Four pulmonary veins (two from each lung) converging directly behind the hypoplastic left atrium to form a common dilated vertical vein seen posterior to the bifurcation of pulmonary artery crossing the left main bronchus anteriorly to join the left innominate vein, which is further draining into the SVC and ultimately into the dilated Right Atrium.

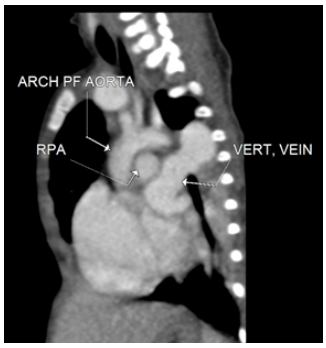


Figure 5a: Oblique sagittal MPR image

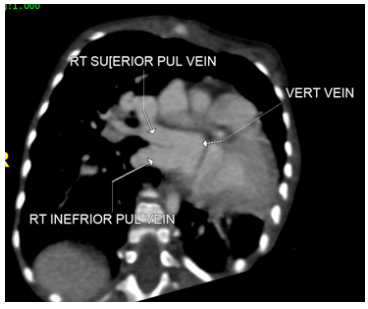


Figure 5b: Oblique coronal MPR image

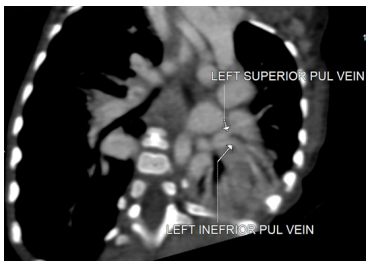


Figure 5c: Oblique coronal MPR image

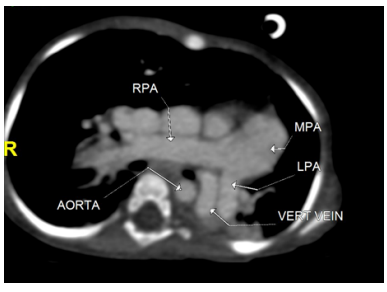


Figure 5d: Oblique axial MPR image

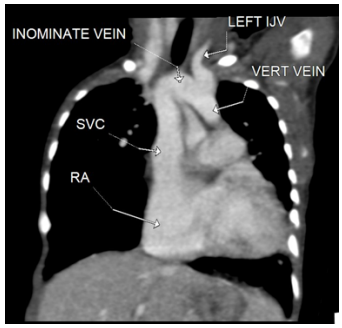


Figure 5e: Coronal MPR image

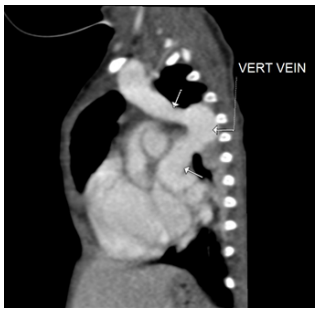


Figure 5f: Oblique sagittal MPR image

Hypoplastic left heart syndrome:
Two right-sided and two left-sided prominent pulmonary veins forming a confluence posterior to the hypoplastic LA. This confluence is communicating with a dilated and tortuous supra-cardiac vertical vein, via a vascular loop at the hilum of right lung between the upper right pulmonary vein and the vertical vein. The vertical vein is draining into the superior vena cava at the site of its origin from the right and left brachiocephalic veins. LV is hypoplastic with PDA (blue arrow).

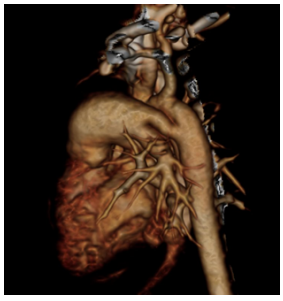


Figure 6a: 3d VR image

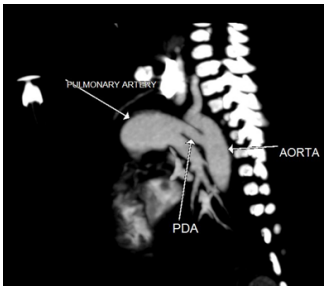


Figure 6b: Sagittal MIP image

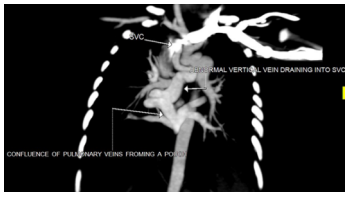


Figure 6c: Coronal MIP image

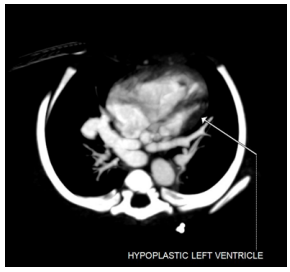


Figure 6d: Axial MIP image

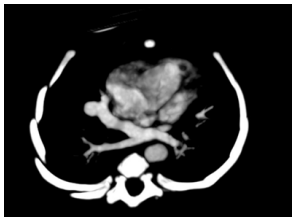


Figure 6e: Axial MIP image

Hemitruncus arteriosus
RPA arising from aorta
MPA continuing as LPA
Aberrant origin of right pulmonary artery from the posterior aspect of ascending aorta. - representing ***hemitruncus arteriosus.***
Over-riding of aorta.

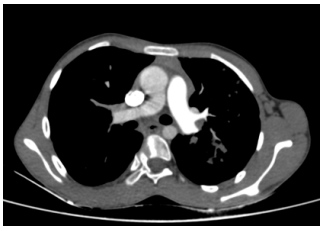


Figure 7a: Axial MPR image depicting RPA arising from aorta and MPA continuing as LPA.

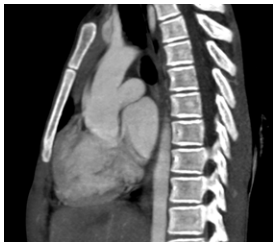


Figure 7b: Sagittal MPR image depicting RPA arising from aorta.

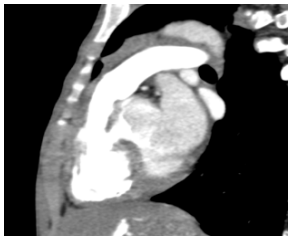


Figure 7c: Sagittal MPR image showing VSD.

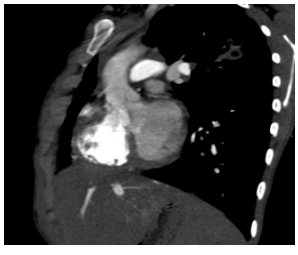


Figure 7e: Sagittal MPR image depicting over-riding of aorta.

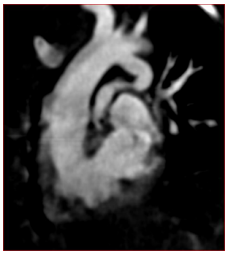


Figure 8a: Sagittal MIP image depicting severe stenosis of the pulmonary artery (blue arrow) at the infundibular level

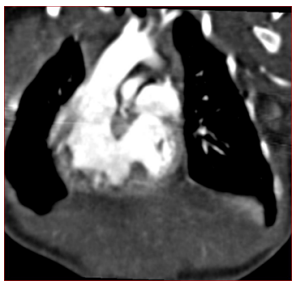


Figure 8b: Coronal MIP image depicting ventriculo-arterial discordance in the form of aorta (blue arrow) arising from the right ventricle and pulmonary artery arising from the left ventricle

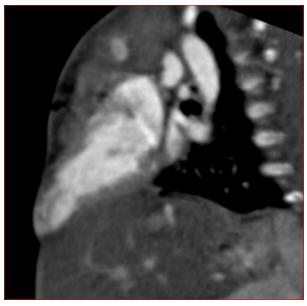


Figure 8d: Sagittal MPR image showing partial ectopia cordis.

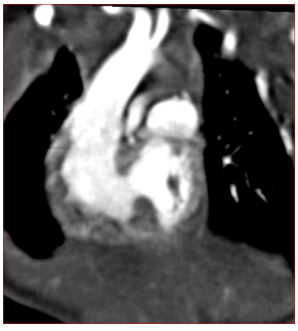


Figure 8 e: Oblique coronal MIP image depicting VSD.

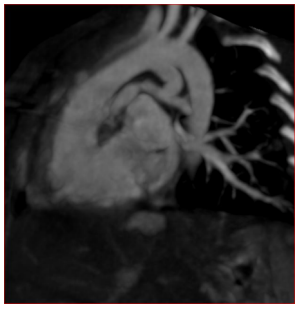


Figure 8 g: Oblique coronal image depicting PDA (blue star)

Associated anomalies:

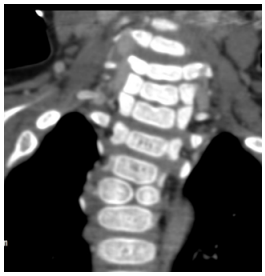


Figure9 a: Bone window settings reveal butterfly D3 vertebra.

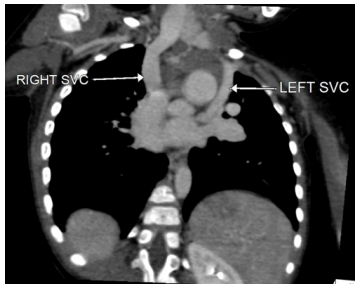


Figure 9c: Coronal MIP reformat reveals double SVC.

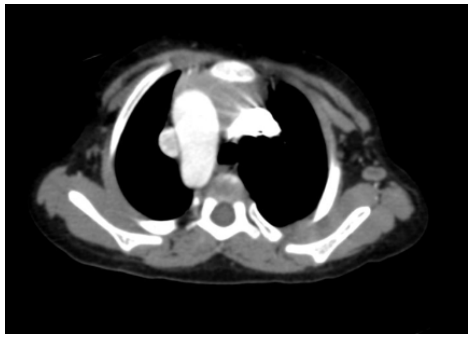


Figure 9d: Axial image reveals right sided aortic arch.

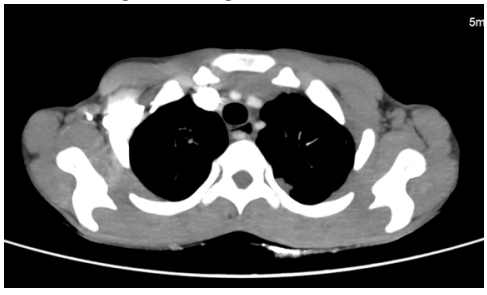


Figure 9e: Axial MIP image depicting aberrant right subclavian artery. INTERRUPTED AORTIC ARCH

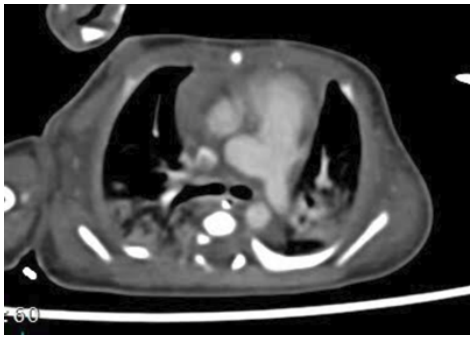
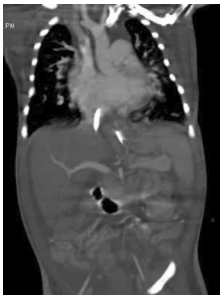


Figure 10 a: Axial MPR imaging depicting aorto-pulmonary window (blue star) seen as communication between the ascending aorta and MPA.



PENTALOGY OF FALLOT

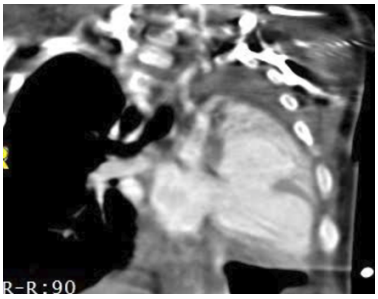


Figure 11 a: Oblique coronal MPR image depicting severe hypoplasia of left ventricle, VSD and right ventricular hypertrophy. Also noted is severe left hemithoracic volume loss. An ASD is also noted.

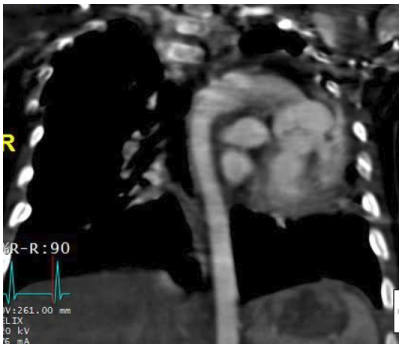


Figure 11b: Oblique coronal MPR image depicting over-riding of aorta.

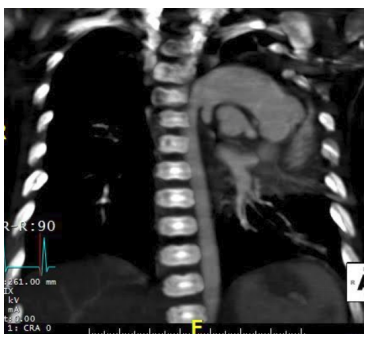


Figure 11c: Oblique coronal MPR depicting PDA.

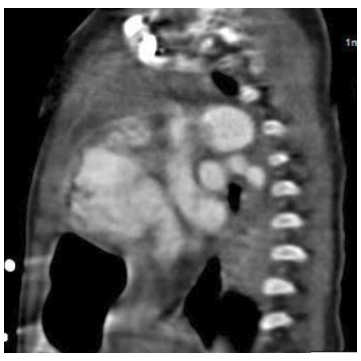


Figure 11d: Oblique sagittal MPR showing infundibular pulmonary stenosis.

REFERENCES

1. Sebastiŕ, C., et al., Aortic Stenosis: Spectrum of Diseases Depicted at Multisection CT. RadioGraphics, 2003. 23(suppl_1): p. S79-S91.
2. Goo, H.W., et al., CT of Congenital Heart Disease: Normal Anatomy and Typical Pathologic Conditions. RadioGraphics, 2003. 23(suppl_1): p. S147-S165.
3. Shirani, S. and M. Soleymanzadeh, Diagnosis of aortic interruption by CT angiography. Polish Journal of Radiology, 2013. 78(1): p. 72-74.
4. Johnson, T.R., Conotruncal cardiac defects: a clinical imaging perspective. Pediatr Cardiol, 2010. 31(3): p. 430-7.
5. Didier, D., et al., Morphologic and functional evaluation of congenital heart disease by magnetic resonance imaging. J Magn Reson Imaging, 1999. 10(5): p. 639-55.
6. Garg, P., et al., The anomalous origin of the branch pulmonary artery from the ascending aorta. Interact Cardiovasc Thorac Surg, 2012. 15(1): p. 86-92.
7. Leschka, S., et al., Pre- and Postoperative Evaluation of Congenital Heart Disease in Children and Adults with 64-Section CT1. Vol. 27. 2007. 829-46.
8. Westra, S.J., et al., Three-dimensional helical CT of pulmonary arteries in infants and children with congenital heart disease. AJR Am J Roentgenol, 1999. 173(1): p. 109-15.
9. Choi, Y.H., et al., MR imaging of Ebstein's anomaly of the tricuspid valve. AJR Am J Roentgenol, 1994. 163(3): p. 539-43.
10. Nicolay, S., et al., CT imaging features of atrioventricular shunts: what the radiologist must know. Insights into Imaging, 2016. 7(1): p. 119-129.