1. Echo (TTE) allows assessment of ASD location, size, & associated manifestations e.g. right heart size, function & pulmonary pressure.
2. TEE is recommended if TTE is suboptimal, associated abnormalities are suspected, and is often required for diagnosis in sinus venosus.
3. Ostium secundum is most common ASD type (75% of cases), usually isolated congenital anomaly. L-R shunt causes right-sided dilatation.
4. Ostium primum ASD is usually associated with cleft anterior mitral or tricuspid valve leaflets (causing MR or TR), and VSD.
5. Sinus venosus ASD is usually associated with anomalous right-sided pulmonary veins connecting to the right atrium or SVC.
6. ASD should be considered in patients with new atrial arrhythmias.
7. Paradoxical embolism is a risk for all ASD patients regardless of size.
8. ASDs manifest fixed splitting of S2 and absence of sinus arrhythmia.
9. ASD is closed if there is right-sided volume overload or symptoms.
10. Most patients with PFO are asymptomatic. PFO closure may be considered in recurrent cryptogenic stroke despite medical therapy.
11. Perimembranous VSDs, most common, 80% in adults.
12. Subpulm./outlet, 6% non-Asians, 33% Asians, rare spont. closure.
15. Small VSDs cause small shunts and loud holosystolic murmur; no LV volume overload and no pulmonary artery hypertension.
16. Moderate-sized VSDs and small to moderate L-R shunts cause LV volume overload and pulmonary artery hypertension with mild CHF.
17. Large VSDs are detected in childhood; without early closure, cause irreversible pulm. HTN with R – L shunt (Eisenmenger syndrome).
18. TTE allows identification of VSD, number & location; PA pressures, AI, TR. MRI is useful in inlet or apical VSDs, not seen by TTE.
19. Children and adults with isolated small VSDs rarely require closure.
20. VSD closure is recommended in adults with progressive AI or TR, progressive LV volume overload, and, recurrent endocarditis.
21. Unoperated aortic coarctation has 75% mortality by 46 yrs of age.
22. Coarctation is diagnosed by HTN in upper extremities & lower BP in lower extremities, resulting in radial – femoral pulse delay.
23. TTE confirms diagnosis of coarctation and associated bicuspid AV (50% of cases). CMR & CT are preferred for imaging the aorta.
24. Auscultatory findings in coarctation include a systolic murmur & S4.
25. Surgical or percutaneous treatment of coarctation is recommended when the systolic gradient across the coarct is > 30 mmHg.
26. PDA is associated with maternal rubella & prematurity.
27. Adults with a moderate L – R PDA shunt present with left-sided cardiac enlargement, CHF symptoms and a machinery-type murmur.
28. Adults with previously unrecognized PDA may present with cyanosis and clubbing affecting the feet more than the hands.
29. TTE with Color Doppler is usually diagnostic of PDA; if nondiagnostic, cath helps identify shunting, PVR, and reactivity of the vascular bed.
30. Transcatheter/surgical closure in moderate-size PDA or endocarditis, without irreversible pulm. HTN. Otherwise, observation.
31. Valvular pulmonary stenosis is usually an isolated congenital abnormality; it may be associated with Noonan syndrome.
32. Severe pulm. stenosis may be tolerated for yrs without symptoms.
33. TTE & Doppler confirms the diagnosis and determines the severity of pulmonary stenosis and the degree of right ventricular hypertrophy.
34. Intervention is recommended when the peak systolic gradient is > 50 mmHg, when RVH is present, or when the patient is symptomatic.
35. Preferred pulmonic stenosis therapy is balloon valvuloplasty. Cardiac surveillance is needed lifelong post valvuloplasty or replacement.
36. Post‐ASD-repair: Afib incidence increases with age at repair, device dislocation causing arrhythmia, peric. effusion, aortic erosion & SCD.
38. Post‐coarct-repair: persistent/recurrent HTN (50 – 75%), CAD, aortic aneurysm or dissection, endocarditis, AV disease, recoarct; may require reop. Age at initial repair is predictive of long-term survival.
39. Tetralogy of Fallot is most common cyanotic congenital heart disease; unoperated adults are rare. 35-yr survival postop is 85%.
40. Post‐TOF-repair: PR (at RVOT annular patch); enlarged RV, TR, A/V arrhythmias; residual RVOT obstruction, PS, or VSD; RV or LV systolic & diastolic dysfunction, VT & SCD. Reop is commonly required.
41. Cyanotic congenital heart disease disposes to hemostatic problems, scoliosis, painful arthropathy/arthitis, gallstones, pulm hemorrhage or thrombus, paradoxical cerebral emboli/abscess, renal disease.
42. Cyanotic patients have compensated erythrocytosis with stable hemoglobins & require no intervention.
43. Cyanotic patients who have hyperviscosity (headaches and reduced concentration), dehydration should be excluded before phlebotomy.
44. Cyanotic patients are at risk of paradoxical embolism: from LV line air- air filters are used; from DVT- early ambulation/SCDs are used. Anticoagulation is avoided (platelet dysfunction/bleeding tendency).
45. Pregnancy has 50% maternal mortality in cyanotics & Eisenmenger.
46. Treatment options for symptomatic Eisenmenger syndrome patients include pulmonary vasodilators and lung or heart-lung transplant.
47. Cyanotic/Eisenmenger patients should be seen by adult congenital cardiac specialist annually. When hospitalized for medical or surgical problems, they should be seen & followed by such specialist.