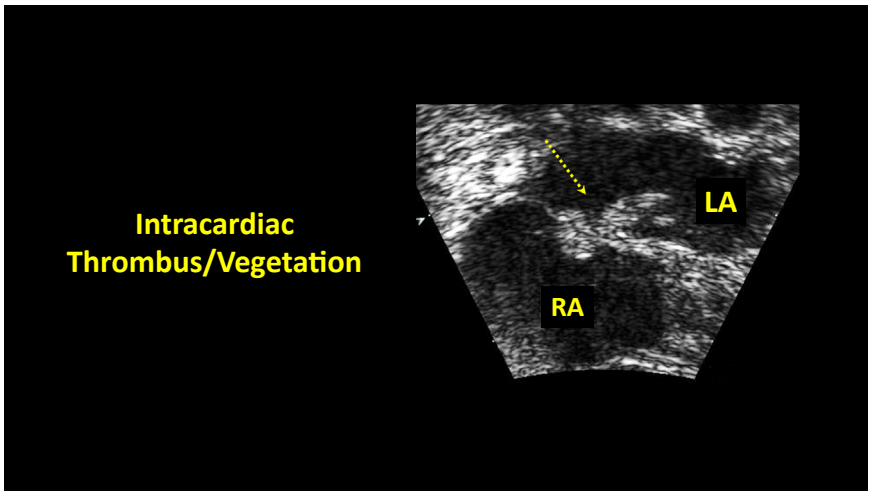

Figure 24 Image of intracardiac thrombus and vegetation from the subcostal view. *RA*, Right atrium.

hypertrophy disorders, and/or disturbances of cardiovascular and pulmonary adaptation after birth.[95] The most common pathology is asymmetrical septal hypertrophy (an anabolic result of fetal hyperinsulinemia triggered by maternal hyperglycemia).[96] Myocardial hypertrophy can extend beyond the septum and involve the free walls symmetrically. As the right and left posterior walls can become thickened diastolic dysfunction in the setting of normal systolic function may ensue.[97] Further hypertrophy may obstruct the LV outflow tract and leads to impaired muscle relaxation and diastolic filling, decreased SBF, and decreased cardiac output. The severe form of the diabetic cardiomyopathy may also lead to decreased pulmonary blood flow and reduced pulmonary venous return presenting clinically with hypoxemia. Additionally, fetal hyperinsulinemia can transiently delay surfactant synthesis and secretion leading to persistent elevation of pulmonary pressures and vascular resistance during the immediate postnatal period.[95] Although most of the alterations in cardiac morphology and systemic or pulmonary hemodynamics appear to resolve during the first 2 to 4 weeks after birth,[98-101] some patients have persistent functional abnormalities beyond 1 month.[102]

Indications for Echocardiography. Clinical phenotypes relate directly to the degree of dynamic obstruction to the LV outflow tract, diastolic and eventual systolic dysfunction, and impact on the pulmonary vasculature. In the setting of a presumed low–cardiac output state (cyanosis, tachypnoea, tachycardia, and cardiomegaly) or PH (increase oxygen requirement, BP lability), TNE is used to detect the different presentations of cardiopulmonary compromise in IDM and facilitate appropriate therapeutic intervention.

Imaging Techniques and Guidance of Clinical Decision-Making. TNE assessment of an IDM with suspected hypertrophic cardiomyopathy and/or PH includes evaluation of chamber dimensions, heart function, and pulmonary pressures, with special attention paid toward the septum and its relationship to the LV outflow tract and degree of obstruction (Table 5). The increase in basal septal thickness may lead to apposition of the anterior leaflet of the MV to the interventricular septum during systole leading to dynamic LV outflow tract obstruction. RV morphology is assessed by both linear dimensions and RV areas acquired at end-diastole and end-systole from the RV-focused apical four-chamber view. RV and LV systolic and diastolic function are evaluated as previously described. Note that left and right cardiac output calculations may be unreliable in the presence of outflow tract obstruction and influenced by shunts. Torsion and LV longitudinal systolic strain by STE may be impaired in IDMs during the transitional period,[103] even with preserved EF, suggesting that rotational mechanics may offer a more sensitive measure of ventricular function.[104] Although septal wall thickness may normalize by 1 month of age, abnormal global and segmental systolic and diastolic strain values may persist.[102]

Recommendations

> In IDMs with clinical signs of low cardiac output or PH, standard TNE should be performed to exclude CHD and evaluate the degree of dynamic obstruction to the LV outflow tract, diastolic and systolic dysfunction, and impact on the pulmonary vasculature.

Twin-to-Twin Transfusion Syndrome. *Pathophysiology and Mechanistic Phenotypes.* Twin-to-twin transfusion syndrome (TTTS) affects approximately 10% to 15% of monochorionic-diamniotic pregnancies and is a significant contributor to perinatal morbidity and mortality.[105] The resultant myocardial functional and structural phenotypes are poorly understood but may stem from the presence of placental anastomoses leading to a distinct clinical phenotype. Chronic hypovolemia and growth restriction complicates the donor twin, while chronic fluid overload, hydrops, and an adverse afterload environment are hallmarks of the recipient twin.[106,107] If left untreated, the mortality rate can approach 100% in one or both fetuses with most survivors experiencing significant morbidity including adverse neurodevelopmental outcomes.[108] Treatment of TTTS with selective laser photocoagulation of the communicating placental vessels (SLPCV) improves survival and cardiovascular outcomes by alleviating, or at least mitigating, the abnormal circulatory load and cardiac morbidity.[109,110] There is an increased incidence of structural valvular disease in affected fetuses including tricuspid, mitral, and most commonly pulmonary valves occurring predominantly in the recipient twin.[111] TTTS results in a cardiomyopathy, occurring predominantly in recipient twins, that is only partially understood; specifically, recipient monochorionic-diamniotic twins with TTTS who do