

situation that requires prevention, because diagnosis is not easy and thus treatment may be too late to save the lung parenchyma. The remaining lung must be examined before chest closure to ensure proper anatomic positioning. Stabilization has been advocated, with suggested techniques including suturing the lung to the parietal pleura, stapling the middle and lower lobes together, and apposition of fibrin glue or other hemostatic products.⁷⁻¹⁰ Although the first 2 techniques have been extensively used at our institution and have certainly contributed to keeping the rate of torsion extremely low, with only 1 anecdotal case, they may produce air leaks in emphysematous lungs.

Coseal is a synthetic polymer that does not carry the risk of any blood-borne infection related to a bovine origin, and it has already been widely tested in preventing prolonged air leaks after major pulmonary resections.^{11,12} No complications have been described with its use, and in this setting it allows prevention of lobar torsion. This technique is fast and simple, and it is certainly less likely to injure the parenchyma than other means of fixation. It may show some limitations with a thoracoscopic approach, however, as it has not yet been tested in that setting. The sealant should be applied on inflated lungs to avoid unintentional restriction of lung expansion.

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Intrauterine rupture of anterior tricuspid valve papillary muscle: Tricuspid valve chordae replacement on the first day of life

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Severe neonatal cyanosis not responding to medical or ventilatory treatment requires stabilization by extracorporeal membrane oxygenation (ECMO) unless a repairable structural cause can be found indicating emergency surgery. We present a case of tricuspid valve repair on the first day of life after intrauterine rupture of the anterior tricuspid valve

papillary muscle due to premature closure of the ductus arteriosus.

CLINICAL SUMMARY

A neonate presented with profound cyanosis immediately after birth. Ventilation and administration of prostaglandin did not improve oxygenation. Echocardiography showed right ventricular hypertrophy and dilatation, severe tricuspid valve insufficiency, reduced low-velocity antegrade pulmonary artery flow, normal pulmonary artery bifurcation, no patent ductus arteriosus, and right to left shunt at the foramen ovale. Despite ventilation with nitric oxide and administration of milrinone, hypoxemia persisted. Detailed echocardiography revealed flail anterior leaflet of the tricuspid valve with severe regurgitation and hyperechogenic papillary muscles of the hypertrophied right ventricle (Figure 1).

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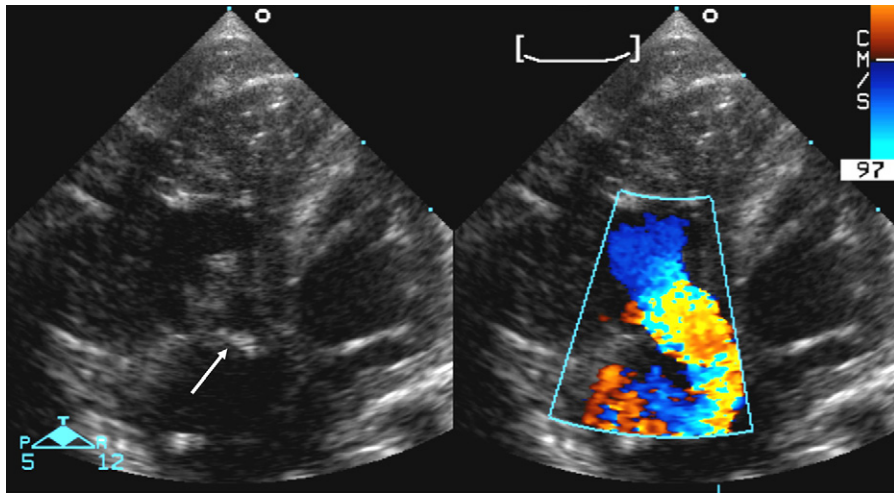


FIGURE 1. Apical 4-chamber view showing flail anterior leaflet of the tricuspid valve with echobright density, severe regurgitation, and enlargement of the right-sided chambers.

Surgery on the first day of life revealed a ductus aneurysm with constriction at the pulmonary site and thrombus formation in the remainder to the aortic site. The papillary muscle of the anterior tricuspid valve leaflet was ruptured, showing scars and calcification. The torn-off papillary muscle head was left attached to the corresponding chordae while the necrotic base was resected for

histopathologic examination. The function of the tricuspid valve was restored by chordae and papillary muscle replacement with repeatedly knotted (8 to 9 knots) artificial 5.0 polytetrafluoroethylene threads (WL Gore and Associates, Flagstaff, Ariz) to receive a twisted stump that had its base with a simple u-stitch at the endocardial site of the ruptured papillary muscle. We did not touch the most

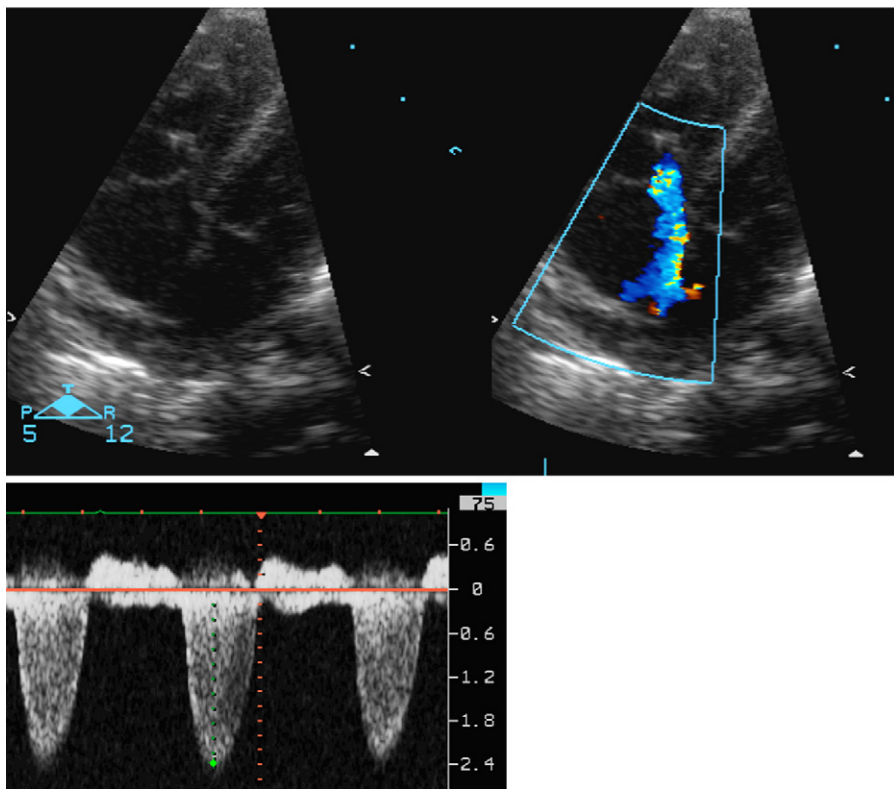


FIGURE 2. Postoperative apical 4-chamber view showing tricuspid valve leaflet coaptation, mild regurgitation, and systolic right ventricular/atrial pressure gradient.

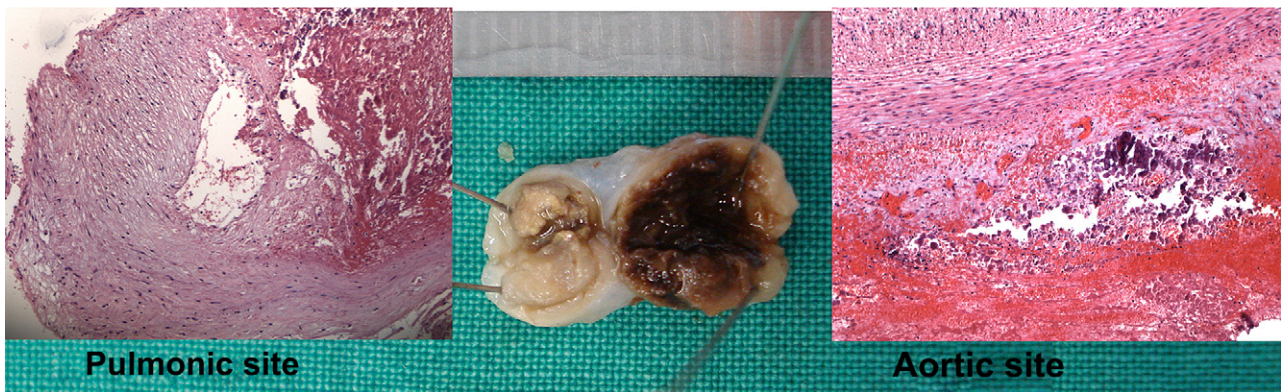


FIGURE 3. Specimen and histopathologic examination showing fibrotic occlusion at the pulmonic site and dilatation of the duct with secondary thrombosis at the aortic site.

delicate neonatal tissue of the anterior tricuspid valve leaflet but established its neo-chordal/-papillary muscle connection, interweaving the last knots of the twisted neo-chordal/-stump within the subvalvar original chordae insertions at the torn-off papillary muscle head. With utmost care during the last knottings, the length of the artificial stump created was adjusted to neither produce restriction nor prolapse of the anterior tricuspid valve leaflet in a rather individual trial and error, eyeballing fashion. The patient was weaned from bypass on low-dose epinephrine and dopamine; ventilation with nitric oxide was necessary for the next few days; and extubation followed on the eighth postoperative day, when echocardiography showed mild tricuspid regurgitation without elevated right ventricular pressure (Figure 2). The further course was uneventful.

Histopathologic examination of the duct (Figure 3) demonstrated fibrotic closure at the pulmonary side and organized thrombus formation with partial calcification at the aortic side. The ruptured papillary muscle was partially calcified.

DISCUSSION

Prenatal ductal constriction and closure is a rare but serious condition. In the majority of patients, it is related to maternal intake of nonsteroidal anti-inflammatory medication, but it may also occur spontaneously or be associated with structural cardiac lesions as tetralogy of Fallot or truncus arteriosus.^{1,2} During fetal life, premature closure causes right ventricular hypertrophy and dilatation, tricuspid and pulmonary valve regurgitation, increase in pulmonary artery pressure, and right ventricular dysfunction and papillary muscle ischemia, sometimes resulting in muscle rupture with flail tricuspid valve leaflet.^{3,4} The increase in pulmonary artery pressure may cause hypertrophy of the media, leading to postnatal persistent pulmonary

hypertension. Early induction of delivery should be discussed, resulting in decrease of right ventricular and pulmonary artery pressure.^{1,5}

If intrauterine diagnosis of ductal closure is not possible, postnatal echocardiographic features include right heart dilatation, right ventricular hypertrophy, tricuspid regurgitation, and hyperechogenicity of the tricuspid valve papillary muscle. With postnatal decrease of pulmonary vascular resistance, most cases need no specific treatment or require only oxygen supplementation. However, mechanical ventilation, administration of nitric oxide or pulmonary vasodilators, and ECMO may be necessary in severely hypoxic and acidotic infants.^{3,4}

CONCLUSIONS

Tricuspid papillary muscle rupture as demonstrated in the present case may be related to muscle ischemia in the presence of rapidly increasing right ventricular pressure after premature closure of the duct.^{1,2} Consecutive prolapse of the anterior leaflet and severe neonatal tricuspid regurgitation is a rare cause of persistent neonatal cyanosis and a life-threatening situation with the need for immediate postpartum surgical intervention to avoid ECMO for bridging to surgical repair.

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