A SUTURELESS TECHNIQUE FOR THE RELIEF OF PULMONARY VEIN STENOSIS WITH THE USE OF IN SITU PERICARDIUM

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Pulmonary vein (PV) stenosis develops as a progressive and usually lethal complication after surgical repair of total anomalous PV connection. Conventional surgical repair for the management of recurrent PV stenosis has been generally unsuccessful because of proliferative neointimal hyperplasia resulting in recurrent PV obstruction. The factors that result in recurrent stenosis after the usual types of patch venoplasty are unknown. We speculated that direct suturing of PVs and patch material may be the substrate for turbulent blood flow triggering intimal hyperplasia and eventual narrowing of the vein. On the basis of these considerations, we developed a sutureless technique for repairing PV stenosis with in situ pericardium. We present here its early but promising results.

Patients

PATIENT 1. Patient 1 was born in January 1995 with infradiaphragmatic total anomalous PV connection and severe obstruction of the descending vertical vein. On day 1, the infradiaphragmatic anomalous vein was ligated and the confluence was anastomosed to the left atrium under conditions of hypothermic circulatory arrest. At 6 months, the child had conspicuous tachypnea. Echocardiography revealed obstructed left PVs with a mean gradient of 7 mm Hg (peak 16 mm Hg) and suprasystemic right ventricular pressure. Angiocardiography demonstrated variable obstruction of all four PVs, with a mean pulmonary artery pressure of 38 mm Hg (systolic 92 mm Hg). At reoperation, obstruction of all PVs was confirmed. Under conditions of circulatory arrest, a pedicled flap of free right atrial wall–superior vena cava junction based at the inferior vena cava was used to patch the right-sided veins and carried behind the aorta to patch the left upper PV. The left lower vein was repaired with a flap created from the left atrial appendage.¹

After that operation, the child had repeated admissions with respiratory tract infections, complicated at 1 year by hemoptysis. Echocardiography revealed recurrence of obstruction. At the second reoperation, atretic left-sided veins and severe stenosis of right-sided veins were noted.

The PVs were opened, and an in situ pericardial sutureless patch was used for reconstruction. Echocardiography a year later showed patent veins, with mean gradients of 4 mm Hg on the right side and 5 mm Hg on the left. The estimated mean pulmonary artery pressure was 10 mm Hg. Perfusion lung scan showed 67% perfusion on the right side and 33% on the left. The child currently has no symptoms.

PATIENT 2. Patient 2, a female infant, was born on January 1995 with total anomalous PV connection to coronary sinus with echocardiographic evidence of partial obstruction. At 2 weeks, she underwent repair consisting of unroofing of the coronary sinus, as described by Van Praagh and Harken.² On completion of the procedure, the patient could not be weaned from cardiopulmonary bypass. At this time the right ventricular pressure was suprasystemic. The child was placed on extracorporeal membrane oxygenator and subsequently weaned after 3 days, with delayed sternal closure in 10 days as a result of persistent hemodynamic instability. Echocardiographic findings before discharge revealed unobstructed PV confluence–left atrium connection. However, echocardiography 4 months later revealed an obstructed right upper PV with a mean right ventricular pressure of 25 mm Hg. Perfusion scan showed 87% perfusion to the left lung and 13% to the right. Eleven months after the operation, echocardiography and cardiac catheterization revealed pulmonary hypertension, with a mean pulmonary artery pressure of 34 mm Hg and anatomically right PV stenosis.

At reoperation, the presence of severe bilateral PV stenosis extending from left atrium a variable distance into the intraparenchymal PVs was confirmed. An in situ pericardial baffle was used for repair, as described here. Cardiac catheterization 6 months later revealed normal venous drainage on the left side and mild obstruction on the right side. The mean pulmonary artery pressure was 16 mm Hg. Perfusion lung scan showed 42% to left lung and 58% to right. The child has no symptoms at 15 months of postoperative follow-up.

Technique. Standard cardiopulmonary bypass technique is used. The incision is made into the left atrium and extended into both upper and lower PV ostia separately (Fig. 1, A). This incision can be carried into the secondary, and if necessary, the tertiary branches of both the upper and lower lobe veins to a level at which the intima appears grossly normal. The pericardium overlying the entrance of the PVs is used to create an enlarged communication between the opened PVs and the left atrium. The neoatrium is created by suturing the pericardium to the epicardium of the left atrium, completely circumscribing the opening in the left atrium and PVs so that the PV effluent is contained by pericardium. The reconstruction is

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initiated with two parallel vertical incisions in the pericardium extending from anterior to posterior; the superior incision is slightly above the junction of the superior PV with the left atrium, and the inferior incision is slightly below the level of the inferior PV. Suturing is begun in the pericardium just above the junction of the superior PV with the left atrium (Fig. 1, *B*) taking care to avoid distortion of the PV. A few tacking sutures may be placed to secure the adventitia of the opened PV to the adjacent pericardium to maintain the PVs in the fully opened position. The suture line is continued toward the left atrium opening, joining the medial aspect of the left atrial incision. A second inferior suture is started below the inferior PV and continued in the same manner to the left atrial incision to join the superior suture line (Fig. 1, *C*). Care should be taken while suturing the inferior limb of the pericardial baffle to obliterate the communication with the oblique sinus, especially on the left side, if not already obliterated by adhesions. On both the left and right sides, it is necessary to carefully mobilize the phrenic nerve with its accompanying vessels and fat pad from the adjacent pericardium to avoid entrapment in the pericardial suture line.

Comment. The prognosis for acquired PV stenosis has historically been poor. This remains true despite early recognition of the problem by two-dimensional echocardiography and Doppler flowmetric evaluation. Repair is challenging and frequently disappointing.³ Interventional techniques are equally discouraging and carry a high mortality rate.⁴ Various procedures have been used to enlarge the stenotic veins. The use of artificial and allograft materials has been discouraged because of neointimal formation, lack of growth potential, and increased need for reoperation.⁵ Pacifico and colleagues¹ recommended the use of atrial wall or atrial septal tissue to enlarge stenotic PVs, although the durability of this technique has not been substantiated.

We, as well as others,⁶ have speculated that turbulent flow accompanying congenital PV stenosis or that occurring after imperfect surgical repair of total anomalous PVs acts as a stimulus for PV intimal injury and triggers a neointimal fibrous proliferative response. The injury response may be further amplified by the trauma associated with direct suturing of the PV intima. The sutureless *in situ* pericardial reconstruction described in this report was designed to minimize factors that could contribute to the pathogenesis of PV obstruction. This technique is similar to that described in a case report in which the authors described excision of the PVs, left atrial connection, and reconstruction with a sutureless pericardial pouch.⁷ Phrenic nerve injury, a potential complication of this procedure, was avoided in our two cases by careful mobilization of the neurovascular pedicle.

In our previous experience,⁴ recurrent obstruction has invariably occurred within a few months after surgical procedures, with or without adjunctive stent implantation, to relieve bilateral PV obstruction that has developed after repair of total anomalous PV drainage. This provides some optimism for the durability of the sutureless technique, which has successfully eliminated PV obstruction without evidence of progressive flow alteration during 18 months of follow-up.

Fig. 1. The sutureless technique for the relief of PV stenosis. **A**, The incision is made into the left atrium and extended into both upper and lower PV ostia separately. **B**, Suturing is begun in the pericardium just above the junction of the superior PV with the left atrium. **C**, A second inferior suture is started below the inferior PV and continued in the same manner to the left atrial incision to join the superior suture line. SVC, Superior vena cava; AO, aorta; IVC, inferior vena cava.

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