postoperative compromise of the airways. To prevent left recurrent laryngeal nerve injury, we dissected the nerve and brought it above the aorta, preventing its entrapment between the aorta and the tracheobronchial tree. We found this technique particularly helpful in the patient described herein, as we extended the slide tracheoplasty into the left main bronchus because of the narrowing of the origin of the left main bronchus.

In isolation, we would repair a hypoplastic aortic arch with an end-to-side anastomosis [5]. To prevent bronchial compression in this patient, we augmented the aorta with a homograft pericardial patch. Furthermore, we used a pericardial flap, previously described [3] to securely seal the site of tracheal repair and to prevent subsequent erosion of the aorta and its branches into the tracheobronchial tree. This pericardial flap is easy to place, and it functions as native pretracheal fascia. In conclusion, slide tracheoplasty involving the left main bronchus and concomitant hypoplastic aortic arch repair can be successfully performed in a high-risk low-weight neonate.

References

Repair of an Unusual Aortic Coarctation Using an Extracellular Matrix Patch
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The surgical treatment of neonatal aortic coarctation is usually accomplished with either a termino-terminal anastomosis or a subclavian flap [1, 2]. When anatomy is not suitable for these procedures, a patch may be used to enlarge the narrowed isthmus [3]. Although technically simple, this solution is frequently burdened by the late development of aneurysmal dilatation of the aorta. It occurs at the level of the patch, on the opposite native wall, regardless of the kind of patch [4]. This complication is probably determined by the disruption of the circumferential fibers of the aortic wall where the patch has been inserted [5]. The recent introduction of extracellular matrix (ECM) patches could represent a solution because they are showing capable of being colonized and replaced by the original tissue, restoring its integrity [6]. The ECM patches employed for coarctation surgery should prevent aneurysmal development.

A 3-week-old male was referred to our center by his family pediatrician, with the suspect of aortic coarctation. At clinical examination he showed a continuous murmur and an arterial pressure difference between upper and lower limbs (107/62 mm Hg vs 46/31 mm Hg). Echocardiography confirmed the diagnosis but showed a quite unusual anatomy. The aortic arch was normally developed; at its end a very small left subclavian artery originated. Immediately after the left subclavian, the aortic lumen reduced to 2 mm and maintained this dimension for more than 3 cm, then returned to a normal diameter. The patent ductus arteriosus (PDA) was well evident and entered the thoracic aorta at the end of the narrowed segment, extremely far from the left subclavian. A 40 mm Hg pressure gradient with diastolic runoff was measured (Fig 1).

The patient underwent surgery through a left thoracotomy and anatomy appeared exactly as described by echocardiography. Because of the extension of the narrowing and the small left subclavian, either a termino-terminal anastomosis or a subclavian flap were not feasible, and we decided for an enlargement patch. After heparin infusion, proximal and distal aortic cross-clamping, the PDA was ligated with a silk string and the entire narrow tract incised longitudinally, from the foot of the left subclavian to almost 1 cm downstream of the PDA. The opened aortic segment was then enlarged with a CorMatrix (CorMatrix, Alpharetta, GA) patch tailored and sutured with a polydioxanone running suture (Fig 2).

Accepted for publication June 3, 2013.
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Postoperative course was completely uneventful. Echocardiographic examination showed a normal reconstructed thoracic aorta, with no residual gradient, and no pressure gradient between the limbs was detected. The patient was disconnected from mechanical ventilation after 2 days and discharged from the intensive care unit 1 day later. Subsequent echocardiograms showed no differences with the postoperative one and clinical examination appeared totally negative. The patient was discharged from hospital 7 days after the operation.

During the first months of follow-up, the patient remained completely asymptomatic, with regular growth, no clinical findings, and normal echocardiographic reports. However, about 4 to 5 months after the operation, recoarctation findings were detected; pressure gradient between limbs (136/100 mm Hg vs 86/45 mm Hg), continuous murmur, and echocardiographic evidence of a significant distal restenosis, with a 64 mm Hg pressure gradient and diastolic runoff.

Balloon angioplasty was then indicated. Aortography showed a focal significant restenosis, located in a very distal position, while the rest of the reconstructed aorta appeared completely normal (Fig 3). Measured pressure gradient was 70 mm Hg. Restenosis was easily and completely dilated, gradient lowered to 10 mm Hg, and no complications occurred (Fig 4). Echocardiography confirmed the good result, with a residual gradient of 20 mm Hg, without diastolic runoff.

In the next 6 months after angioplasty, follow-up has always been completely normal. The patient has shown no symptoms and a normal growth, clinical examination has always been negative, and echocardiograms have demonstrated a good anatomy, with neither restenosis nor dilatation and a pressure gradient of 24 mm Hg, without diastolic runoff.

Comment

Aneurysmal dilatation is a frequent late complication after aortic coarctation repair when an enlargement patch has been used [4]. Though not completely clear, the mechanism seems to be related to the disruption of the wall anatomy. Resecting the circumferential muscular and elastic fibers of the aorta and inserting a nonviable patch may unbalance the wall stress toward the native part of the aortic, which could weaken and dilate [5].

The ECM patches are a promising device that could change the concept of tissue reconstruction. Acting as a scaffold for tissue regrowth, they behave like a patch only initially. In 5 to 6 months they become gradually colonized, reabsorbed and replaced by the tissue on which they are sutured [6]. Some studies also report evidence of
contractile activity, 1 year after, when an ECM patch has been used on a muscular structure [7].

Established that only a patch enlargement could be feasible, we decided to use ECMs to rebuild the aortic anatomic integrity and avoid the mechanisms that could lead to aneurysmal dilatation. After 1 year, no evidence of aortic dilatation has been seen. On the contrary, a restenosis developed 5 to 6 months after surgery. It is well documented that any kind of foreign material may impair the healing process of a suture through an inflammatory mechanism [8]. Our first hypothesis was for a scarring of the suture line at the end of the patch. However, some interesting elements are to be considered. We used a re-absorbable suture and this should limit the presence of foreign material. The suture was a very long single running line, while restenosis was focal. Restenosis developed 5 to 6 months after surgery, the time taken by the ECM to be replaced by native tissue. Angioplasty was easy, with optimal result and no signs of recoil after 6 months. A careful examination of the images shows that focal narrowing did not occur at the distal end of the patch but slightly upstream where the PDA entered the aorta. Considering these observations, we made another intriguing hypothesis; restenosis could be determined not by a scarring process but by a regrowth of ductal tissue on the ECM patch, which could have created a true new coarctation.

Unfortunately this hypothesis is not supported by any histologic evidence. However, it could be helpful, suggesting the removal of any residual ductal tissue when using an ECM patch to correct an aortic coarctation.

References