Angioplasty for coarctation of the aorta: long-term results


ABSTRACT Balloon coarctation angioplasty (BCA) was performed in seven consecutive patients (five boys and two girls) 18 months to 18 years old (mean 9.5) with isolated discrete unoperated coarctation of the aorta. A No. 8F or 9F catheter was chosen with balloon lengths of 30 or 40 mm and maximum inflation diameters 1 mm less than the smallest measured aortic diameter determined 1 cm proximal to the coarctation site. A 10 sec inflation-deflation cycle of 6 to 8 atmospheres (90 to 120 psi) was performed. The peak systolic pressure gradient (PSG) before BCA ranged from 35 to 70 mm Hg (mean 58), and immediately after BCA it decreased to 0 to 20 mm Hg (mean 7). One to two year follow-up (mean 14 months) of the seven patients revealed a PSG range of 10 to 30 mm Hg (mean 19). Repeat angiography was performed immediately proximal to the coarctation site. Three patients (43%) had evidence of aneurysm formation at or immediately distal to the balloon dilatation site. One patient had coarctation restenosis. While initial results with BCA for unoperated coarctation were encouraging, current data raise serious concerns about its long-term safety and efficacy. Circulation 75, No. 3, 600-604, 1987.

IN THE PAST 5 years, the percutaneous balloon angioplasty technique has been applied in patients with a variety of congenital and acquired heart lesions. The conditions that have been treated with this technique include: pulmonary valvular,1-6 branch pulmonary artery,7 and pulmonary vein stenosis,8, 9 aortic valve stenosis,10-15 superior and inferior vena caval obstruction,16, 17 coarctation of the aorta,18-24 and restenosis coarctation.25 The current report presents our initial and long-term results with seven patients with discrete coarctation.

Methods

Seven patients (five boys, two girls) 18 months to 18 years old (mean 9.5) underwent percutaneous balloon dilatation angioplasty for unoperated discrete coarctation of the aorta from December 1983 to December 1984. All patients had clinical evidence of coarctation as manifested by diminished femoral pulses, systolic hypertension of the upper extremities, and short grade 2/6 systolic ejection murmur at the left base and back. A chest x-ray, electrocardiogram, two-dimensional, and pulsed Doppler echocardiogram were obtained for each patient. Magnetic resonance images of the thoracic aorta were obtained in all patients after the angioplasty. Two patients underwent magnetic resonance imaging before angioplasty. Informed consent was obtained for each patient and patients were enrolled in a research protocol approved by the Research and Clinical Projects Committee (IRB). The specific technique employed for our balloon angioplasty has been previously reported.24

Follow-up evaluation consisted of repeat physical examination with arm/leg blood pressure determinations by sphygmomanometer, electrocardiography, and two-dimensional and pulsed Doppler echocardiography both 1 month and 1 year after coarctation angioplasty. Magnetic resonance images were obtained in all patients from 1 to 16.5 months (mean 7.1 months) after angioplasty. Electrocardiographically gated magnetic resonance images were obtained in coronal, sagittal, transaxial, and left anterior oblique imaging planes with a 0.6 tesla superconducting magnet to obtain 0.75 to 1 cm thick sections. Repeat aortograms and intra-aortic pullback pressure gradients were obtained in all seven patients.

Results

The hemodynamic measurements before, immediately after, and at follow-up catheterization are summarized in Table 1 and illustrated in Figure 1. Before balloon angioplasty, the peak systolic pressure gradient across the coarctation site ranged from 35 to 70 mm Hg, with a mean of 58 mm Hg. Immediately after balloon angioplasty, the peak systolic pressure gradi-
TABLE 1
Hemodynamic data: change in peak systolic gradient in response to balloon dilatation angioplasty

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>(\Delta P_0)</th>
<th>(\Delta P_1)</th>
<th>(\Delta P_f)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>35</td>
<td>0</td>
<td>10(^a)</td>
</tr>
<tr>
<td>2</td>
<td>45</td>
<td>0</td>
<td>15</td>
</tr>
<tr>
<td>3</td>
<td>70</td>
<td>0</td>
<td>30</td>
</tr>
<tr>
<td>4</td>
<td>50</td>
<td>0</td>
<td>10(^a)</td>
</tr>
<tr>
<td>5</td>
<td>65</td>
<td>10</td>
<td>15(^a)</td>
</tr>
<tr>
<td>6</td>
<td>70</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>7</td>
<td>65</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>Mean</td>
<td>58</td>
<td>7</td>
<td>17</td>
</tr>
</tbody>
</table>

Gradients were measured at cardiac catheterization before, immediately after, and 1 year after angioplasty.

\(\Delta P\) = peak systolic gradient in millimeters of mercury; \(\Delta P_0\) = initial peak systolic gradient before angioplasty; \(\Delta P_f\) = follow-up gradient at 1 year; \(\Delta P_1\) = peak systolic gradient immediately after angioplasty.

\(^a\)Denotes aneurysm formation.

ent decreased to 0 to 20 mm Hg, with a mean of 7 mm Hg. Follow-up catheterization and aortography were performed in all seven patients. The peak systolic pressure gradient ranged from 0 to 30 mm Hg, with a mean of 18 mm Hg. Three of the seven patients demonstrated aneurysm formation at or just distal to the site of balloon coarctation on the follow-up angiogram and representative angiograms of two are presented in figure 2. Magnetic resonance imaging confirmed this observation in two of the three patients (figure 3). Our results with this imaging technique have been reported.

Pulsed Doppler flow studies immediately after balloon dilatation demonstrated a return to a normal spectral waveform in six of the seven patients. Follow-up pulsed Doppler flow studies predicted residual gradients of coarctation restenosis. Two of the three patients with aneurysm formation underwent successful coarctectomy without any sequelae. Histologic sections of the resected segment from one of the patients showed aneurysmal dilatation of the ballooned coarctation segment with disruption of the intima and elastic media (figure 4). Scar tissue or cicatrix formation was sparse, leaving the thin-walled media covered only by the adventitia.

Discussion

As with any therapeutic modality, both initial and long-term results must be evaluated. Our early observations with balloon angioplasty for unoperated coarctation of the aorta were encouraging and similar to the findings of others. Always of concern, however, was the possibility of long-term adverse consequences such as aneurysm formation at or near the site of dilatation. The fact that this particular complication had occurred in one patient immediately after balloon coarctation angioplasty was particularly disturbing. More recently, aneurysms were found 6 to 12 months after balloon angioplasty in a series of patients with unoperated coarctation of the aorta.

A number of potential factors may be responsible for the development of aneurysms after percutaneous balloon angioplasty of aortic coarctation. Previous histologic studies demonstrated extensive intimal and medial tears in resected coarctation segments that were dilated, and similar findings were observed in an animal preparation of balloon angioplasty of aortic coarctation. In a recent study cystic medionecrosis was consistently found in histologic sections prepared from resected portions of the aorta from patients undergoing surgical repair of aortic coarctation. These findings, along with similar observations in an autopsy specimen of unoperated aortic coarctation, suggest that there may be an inherent wall weakness in patients with this disorder. This could explain the high frequency of aortic aneurysm and aortic dissection previously reported in humans with unoperated coarctation. Cystic medionecrosis, by contributing to the progression of intimal and medial tears, could lead to the adverse effects now being reported after balloon angioplasty.

The presence of aneurysm formation in three patients and of restenosis in one patient in our series raises serious doubts as to the long-term safety and efficacy of this procedure in patients with unoperated coarctation of the aorta. The finding of sparse scar

FIGURE 1. Peak systolic pressure gradient (in mm Hg) across coarctation site before, immediately after, and at 1 to 2 years follow-up (mean 14 months). Dotted line represents the mean gradient.
FIGURE 2. For legend see opposite page.
tissue at the balloon site in one of our patients is of particular concern, since the aneurysm could have enlarged and perhaps dissected or ruptured in the future. Based on these observations, we recommend that follow-up studies be carried out in all patients who have already undergone balloon angioplasty for unoperated coarctation of the aorta. Furthermore, we recommend that the use of balloon dilation angioplasty for treatment of this disorder be restricted until the results of these follow-up studies are known.

We are indebted to the staff of the Cardiac Catheterization Laboratory at The Brookdale Hospital Medical Center for the technical assistance and the excellent care our patients received, to Dr. Yale Rosen for his photography of the histologic results, to North Shore University Hospital for the MRI Magnetic Resonance images by Drs. Boxer, Stein, and their technical staff, and to Lenore Gottlieb and Harriet Lustbader for secretarial assistance in preparing this manuscript.

**FIGURE 3.** Magnetic resonance image (composite sagittal view) of thoracic aorta of patient 1 at 1 year follow-up. Arrow points to aneurysm.

**FIGURE 4.** Top, Photomicrograph of resected aneurysm of ballooned coarctation segment. Arrow points to ballooned site with disruption of intima and elastic media. Note sparse scar tissue formation and thin media covered by adventitia (hematoxylin-eosin stain; original magnification × 4. Middle, Magnification of inset with arrow pointing to disruption of elastic tissue (original magnification × 40). Bottom, High-power magnification illustrating disruption of elastic fibers of media (original magnification × 100).
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