

Complications of Coarctation Repair

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Surgery's role in the treatment of coarctation has been established, and the benefit to life expectancy and quality of life is undeniable. Three post-aortic coarctation repair complications are discussed, with review of existing literature: recurrent or residual aortic coarctation, postrepair aneurysm formation, and spinal cord ischemia. Incidence, potential causative factors, and outcome of surgical or transcatheter treatment for recurrent and residual aortic coarctation are reviewed. A literature review of postrepair aneurysm formation focuses on etiologic factors such as use of patch aortoplasty repair techniques, aortic arch hypoplasia, congenital abnormality of the aortic wall, and persistent hypertension after repair. The spectrum, onset, incidence, and potential risk factors for postcoarctation repair spinal cord ischemia are reviewed. Use of adenosine receptor agonists to achieve a state of ischemic resistance is under investigation to address this potential hazard of coarctation repair. Complications after surgery do occur in certain subsets of patients, but the risk of subsequent intervention is still lower than the hazards associated with the natural course of the defect.

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Aortic coarctation can be defined as a congenital narrowing of the upper descending thoracic aorta, adjacent to the site of attachment of the ductus arteriosus, sufficiently severe that there is a pressure gradient across the area.¹ This condition, which is two to five times more frequent in males as in females, appears to be a spectrum of diseases and may occur in conjunction with bicuspid aortic valve, ventricular septal defect, patent ductus arteriosus, mitral stenosis or regurgitation, or aneurysms of the circle of Willis and gonadal dysgenesis (eg, Turner syndrome).² Bonnet's classification³ of adult (postductal) and infantile (preductal) aortic coarctation has been replaced; coarctation of the aorta is currently described as (1) isolated, (2) with ventricular septal defect, and (3) with complex intracardiac anomalies.⁴ It appears that 82% of individuals with coarctation have an isolated defect, while 11% have coexistent ventricular septal defect, and 7% have other important cardiac

anomalies.⁵ While severe obstruction presents with congestive heart failure in infancy, presentation in adulthood is usually as an incidental finding or for complications associated with repair in infancy.

Paris⁶ first described the pathologic features of coarctation in 1791. Experiments performed in the 1930s showed the feasibility of cross-clamping the aorta in animals. Findings from these experiments permitted cross-clamping of the aorta both above and below the origin of the ductus arteriosus, allowing division and closure. This experience led to the decision to perform the same operation for coarctation of the aorta by applying clamps above and below the coarctation, resecting it, and sewing the aorta end-to-end. Crafoord and Nylin⁷ first described this successful surgical correction in 1945. The introduction of prostaglandin E₁ to maintain ductal patency allowed repair in stable neonates. Modifications of the technique included vertical incision and transverse closure or prosthetic patch enlargement of the stenotic isthmus (Vossschulte, 1961),⁸ and subclavian flap aortoplasty, introduced in 1961 by Waldhausen and Nahrwold.⁹ Gross¹ and other authors have described the technique of tube-graft replacement.

Successful alleviation of aortic coarctation dramatically improves long-term survival. Two thirds of patients over the age of 40 years who have uncorrected aortic coarctation have symp-

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toms of heart failure. Three fourths die by the age of 50, and 90% by the age of 60.¹⁰ Similarly, survival after repair of aortic coarctation is also influenced by the age of the patient at the time of surgery. After surgical repair during childhood, 89% of patients are alive 15 years later and 83% are alive 25 years later. When repair of coarctation is performed when the patient is between the ages of 20 and 40 years, 25-year survival is 75%; when performed in patients over 40 years of age, 15-year survival is only 50%.³ Among patients who undergo surgery during childhood, 90% are normotensive 5 years later, 50% are normotensive 20 years later, and 25% are normotensive 25 years later. In contrast, among those who are over 40 years of age at the time of surgery, half have persistent hypertension, and many of those with a normal resting blood pressure after successful repair have a hypertensive response to exercise.¹¹

While surgical correction remains the gold standard, the role of percutaneous balloon angioplasty and expandable cardiovascular stents is investigated for both primary and secondary procedures. Balloon angioplasty of native coarctation is associated with a risk of aneurysm formation and early recurrence of obstruction. Though initially limited to children considered extremely high surgical risks,¹² angioplasty is now frequently used in adults with isolated (primary) or recurrent coarctation. In general, surgical repair should be considered for patients with a transcoarctation pressure gradient of more than 30 mm Hg.¹³

After surgery, which is successful in relieving obstruction in virtually all patients, 80% remain asymptomatic for an average of 20 years after surgery.^{11,14,15} Complications from accelerated cardiovascular atherosclerosis, progression of valvular disease, and occurrence of late complications related to the repair adds to morbidity and mortality.

Operative Morbidity and Mortality

In a chart review of 244 children undergoing repair of aortic coarctation at the University of Virginia Health System (Charlottesville, VA), mortality was limited to three deaths in neonates with unremitting metabolic acidosis not responsive to prostaglandin administration, where the

operation was performed as a last resort. Most series report mortalities of less than 3%.^{16,17}

The mortality for primary surgical repair seems to be in part related to patient age and presence of associated congenital defects, and not to the procedure used.¹⁸ Long-term results are influenced by the presence of concomitant intracardiac defects requiring repair. In our series, 11% of children underwent subsequent open heart procedures, which is slightly higher than that published by Kirklin and Barratt-Boyes.⁵ All the patients in our series undergoing further surgery had uneventful postoperative courses.

Late mortality is caused by congestive heart failure, aortic rupture, myocardial infarction, or subarachnoid hemorrhage.¹⁹ The first report of long-term survival, by Maron et al,²⁰ found the mean age at death was 35.1 years and mortality incidence was especially high in patients undergoing primary repair after age 25.

Diagnosis of Postrepair Complications

Clinical evaluation and management of the patient with coarctation of the aorta continues to evolve. Traditional imaging, by plain film chest radiography, barium esophagography, and arteriography with pressure measurements across the coarctation, has been largely supplanted by doppler echocardiography and magnetic resonance imaging. Echocardiography and magnetic resonance imaging also can noninvasively evaluate the complications of surgery and balloon angioplasty, including residual or recurrent coarctation and aneurysm. Chest radiography continues to play an important role in "first discovery" imaging in asymptomatic patients.

Restenosis

The most common reason for reintervention after repair of aortic coarctation is recurrence of obstruction either at the site of previous repair or distant from it. Our results confirm published data that restenosis develops mainly in subjects who underwent repair during infancy.

Virtually all incidences of recurrence of stenosis occur only in subjects who underwent repair in infancy. Diagnosis depends on follow-up and the clinical criterion is a blood pressure gradient of more than 20 mm Hg between upper and lower

extremities. The incidence of restenosis during a 10-year follow-up study by Koller et al¹⁹ was 10.8% in infants undergoing repair before age 2 and 3.1% when repair was performed beyond 2 years of age. Whether the age or weight²¹ at primary repair plays a role is uncertain. In our series, restenosis occurred more frequently after resection and end-to-end anastomosis (3%) than after other procedures (less than 1% after subclavian flap aortoplasty). In Presbitero et al's series,¹¹ restenosis was seen less frequently after resection and end-to-end repair (8%) than after other procedures. It also suggested a better patency rate (3.6%) for resection and extended end-to-end anastomosis.²² Many cases of early restenosis may in fact be residual coarctation.²³ In most of our patients with complex coarctation, resection and end-to-end anastomosis was the preferred technique, which might explain the higher incidence of restenosis in this group of patients.

Technical factors are often responsible for persistent stenosis after repair.⁵ Insufficient resection of long narrow segments, excessive tension on suture lines, incorrect fashioning of prosthetic or subclavian flaps, or kinking of a graft are all reasons for residual gradients.

Balloon angioplasty for restenosis avoids repeat surgery and was successful in 88% of 90 consecutive cases reported by Yetman et al²⁴ when no associated arch hypoplasia was present. Sakopoulos et al²⁵ reviewed results of surgery for restenosis and a 96% success rate was achieved using the prosthetic patch technique.

Pronounced arch obstruction can be seen after a well-repaired coarctation and might result from the failure of a hypoplastic arch to grow, clamp injury at the time of the initial repair, or both.²⁶ The presence of abnormal mesodermal tissue may play a role by causing abnormal proliferation with resultant intimal and medial hypertrophy.²⁷ In a study of cases with new arch obstruction, DeLeon et al²⁶ reported a mean age of presentation of 13.5 years, after a mean interval of 10 years following repair. It is still uncertain if hypoplastic arches grow after successful repair, and the lack of alpha-actin positive cells in these arches might indicate a reduced potential for growth.²⁸

Aneurysm Formation

Large tortuous collaterals can become aneurysmal, usually in patients older than 10 years.

These saccular aneurysms occur at the origin of the intercostal arteries. The aorta itself may become aneurysmal adjacent to the site of maximal narrowing as a result of hemodynamic effects, aortic dissection, or a mycotic aneurysm.⁵

An aneurysm associated with previous repair can be defined as a change in contour or irregularities in contour at the repair site or by abnormal dimensions at the repair site, defined by the ratio of the widest measurement at the repair site to the measurement of the aorta at the diaphragm.²⁹

The most frequent cause of postcoarctation repair aneurysm is use of a prosthetic patch to relieve the coarctation. During the years that followed the first successful surgical repair of coarctation in the 1940s, a high incidence of restenosis was seen, ranging from 20% to 86%. In response to that problem, the patch aortoplasty was introduced in 1957. Appealing benefits of the technique include limited dissection (less bleeding) and shorter cross-clamp time than for coarctation resection and end-to-end anastomosis. The collateral vessels are all preserved and do not require ligation and division. The anastomosis is always tension-free and quite easy to perform. However, the frequent appearance of true aneurysms offset the advantages of the operation.

The etiology of aneurysm formation after patch aortoplasty has been attributed to several different factors. The first is resection of the coarctation ridge at the time of aortoplasty with violation of the intimal layer.³⁰ We reported³¹ a second contributing factor may be the altered hemodynamics that result from the different tensile strengths of a Dacron patch (Bard Vascular Systems Division, C.R. Bard, Inc, Billerica, MA) and the posterior aortic wall, with the pulsatile waveform being completely directed to the posterior aortic wall by the inflexible anterior patch. A decreased incidence of aneurysm when using more flexible polytetrafluoroethylene supports this theory.³² Some investigators have speculated that there is a congenital abnormality of the aortic wall at the coarctation site³³ and use of a patch aortoplasty as a reoperation after resection with end-to-end anastomosis may increase the risk of aneurysm formation.³⁴ Persistent hypertension after repair seems to contribute to the risk.³⁵ Bogaert et al³⁶ found a high correlation between arch hypoplasia and aneurysm forma-

tion after patch aortoplasty in a magnetic resonance imaging follow-up study.

Ala-Kulju and Heikkinen's series³⁷ reported the highest incidence of aneurysm formation after patch aortoplasty (33%). All of the patients were older than 15 years at the time of surgery and ranged up to 54 years of age. The mean patient age at coarctation repair was 25 years. These authors speculated that the reason for the high incidence of aneurysm formation was an abnormality that had developed in the aortic wall over a prolonged period of time because of the patients' advanced ages at the time of surgery. Our results are similar, with a 20% incidence of reoperation for aneurysm in those patients who underwent Dacron patch aortoplasty. During the early 1980s we followed the principle of using a generous prosthetic patch to create a diameter approaching that of the adult aorta. No significant correlation could be drawn between this and aneurysm formation.

Aneurysm formation has been reported after bypass grafting,³⁸ subclavian flap aortoplasty,³⁹ and has occurred in patients who did not undergo surgical treatment at all.⁴⁰ While true aneurysms are more frequent, false aneurysms associated with infections and endocarditis are seen as well. We saw only one case of mycotic aneurysm after Dacron patch aortoplasty. They occur more frequently after prosthetic patch enlargement and might appear at any time after surgery.

Spinal Cord Damage

Gross and Hufnagel⁴¹ published the first report of experimental end-to-end anastomoses in animals in 1945. Hind-quarter paralysis in some of the animals was noticed and while they thought this would not happen in humans because of collaterals, they implemented the first attempts at preventing this by "packing the entire back of the animal in ice."⁴¹

Collateral circulation between the proximal and distal segments of the stenosed aorta is typical of this condition. It is present in neonates with the condition but increases in size and extensiveness as the patient ages.

Patients with coarctation have abnormal blood flow dynamics to the spinal cord. Angiographic findings of spinal blood supply shows dilated anterior spinal arteries and reversal of blood flow, predisposing the spinal cord to ischemia second-

ary to a steal phenomenon during aortic clamping above the coarctation. However, a low incidence of complications is seen. The presence of collaterals is protective in maintaining spinal cord blood supply during clamping, while the fact that these patients seem to be physiologically adapted to anaerobic metabolism might also play a role.⁴²

Crawford and Sade⁴³ described three infants with coarctation of the thoracic aorta, patent ductus arteriosus, and ventricular septal defect who underwent repair of the coarctation in three different institutions. Despite a technically uncomplicated operation, each developed significant paraplegia postoperatively. Retrospective analysis showed that each patient had been hypothermic during the time of aortic cross-clamping, which, alone or in combination with other factors, might have contributed to the development of paraplegia in these three infants.⁴³

In an inquiry into the paraplegia rate associated with operations for coarctation of the aorta conducted by surgeons in the United Kingdom and Ireland, paraplegia occurred in 16 patients in a total of 5,492 operations, an incidence of 0.3% or once in 343 operations.⁴⁴ This low risk in uncomplicated cases obviates the need for adjunctive measures, eg, intrathecal vasodilators, cerebrospinal fluid drainage, hypothermia, and reimplantation of minor intercostal arteries as is done during other aortic aneurysm surgeries. Complicated cases, demonstrated lack of collaterals, or expectation of a longer procedure, might necessitate use of adjunctive measures as well as partial bypass techniques.

We are investigating pharmacologic methods, including adenosine receptor agonists, used to achieve a state of ischemic resistance similar to the effect in heart muscle.⁴⁵ While the efficacy has been proven when the spinal cord itself is retrogradely perfused with cold saline carrying adenosine⁴⁴, it also has been shown that the systemic administration of adenosine agonists exerts a similar effect.⁴⁶

While paraplegia remains the most dreaded outcome of spinal cord ischemia, lesser insults might occur, resulting in a spectrum from neuropathic pain and bladder dysfunction to complete paralysis.⁴⁷ Paraplegia also might present immediately after surgery, or may be delayed for days after repair.⁴⁸ In a study by Dasmahapatra

et al,⁴⁹ up to 26% of all patients develop reversible ischemic changes of somatosensory evoked potentials during surgery for coarctation repair. Statistical analysis of risk factors concluded that: (1) distal hypotension and reversible spinal cord ischemia commonly occurred during aortic occlusion, and (2) intraoperative interventions that can potentially influence distal aortic perfusion and/or arterial pCO₂ should be used with caution.

Conclusion

The role of surgery in the treatment of coarctation has been established, and while efforts are made to establish indications for each type of repair, the benefit to life expectancy and quality of life is undeniable. Complications after surgery do occur in certain subsets of patients, but the risk of subsequent intervention is still lower than the cumulative hazards associated with the natural course of the defect.

While advances in surgical technique have made this lesion and its complications easily correctable, the problem of spinal cord ischemia remains, especially when patients are reoperated on for complications of previous coarctation repair. Promising animal studies on spinal cord protection might hold the answer to this major problem.

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