Aortic Wall Injury Related to Endovascular Therapy for Aortic Coarctation

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***Abstract*—**Aortic wall complications can occur in unrepaired aortic coarctation (CoA) and after surgical repair or endovascular treatment. This review summarizes the available literature and current understanding of aortic wall injury (AWI) surrounding the management of CoA, focusing specifically on acute and follow-up AWI after endovascular treatment. There have been 23 reported cases of aortic rupture after endovascular treatment for CoA, including angioplasty alone, bare metal stenting, and primary covered stent therapy. Even if these published cases represent only a minority of ruptures that have actually occurred, the incidence is substantially <1%. The incidence of acute aneurysm formation was 0% to 13% after angioplasty, 0% to 5% after bare metal stent placement, and <1% after covered stent placement. The reported incidence and natural history of both acute and new AWI during follow-up after endovascular therapy for CoA varies considerably, likely secondary to ascertainment and reporting biases and inconsistent definitions. Although important AWI after endovascular treatment of CoA seems to be declining in frequency with increasing experience and improving technology, it remains one of the most important potential adverse outcomes. Long-term surveillance for new AWI and monitoring of existing AWI is mandatory, with institution of appropriate treatment when necessary. A central research focus in this population should be determination of the appropriate treatment for both native and recurrent CoA across various ages with regard to limiting recurrent CoA and preventing associated aortic wall complications, in addition to determining the appropriate treatment of various AWI. Consistent definitions and reporting are necessary to truly understand the incidence of, risk factors for, and measures protective against AWI after angioplasty or stent implantation for CoA.** **(*Circ Cardiovasc Interv*. 2015;8:e002840. DOI: 10.1161/CIRCINTERVENTIONS.115.002840.)**

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Aortic wall complications can occur in both unrepaired aortic coarctation (CoA) and after surgical repair or endovascular treatment. In this review, we summarize the literature on and current understanding of aortic wall injury (AWI) surrounding the management of CoA, focusing specifically on endovascular treatment. Before reviewing AWI related to balloon angioplasty (BA) and stent therapy, it is important to set the context by providing an overview of historical, anatomic, and physiological factors that may be related to aortic wall complications in CoA patients, as well as a brief discussion of AWI in patients with surgically repaired CoA.

Definitions of AWI

A clear nomenclature and uniform definitions are essential in the discussion of AWI. Effective endovascular therapy for CoA, particularly BA, typically results in a therapeutic tear or controlled tear, which is a tear through the intima and at least partially into the media, but not involving the adventitia. Therapeutic tears form when the angioplasty balloon is larger than the narrowing to be dilated (the CoA segment) and the intima/media is stretched beyond its yield point. Therapeutic tears may or may not be visible by angiography, but they are almost always seen by intravascular ultrasound (IVUS).[1] When therapeutic tears are evident on angiography, contrast is confined to the aortic wall in the radial dimension, and to the CoA segment axially, and there may be a small intimal flap (Figure 1).[2] By definition, a therapeutic tear should involve only the narrow region of the CoA segment, but the location around the circumference of a CoA is not predictable.[3] The need for a therapeutic tear to achieve a lasting result was noted early in the experience with CoA BA.[4] However, they have been implicated in the development of pathological AWI, such as false aneurysms, dissections, and ruptures. When and how a therapeutic tear becomes pathological is not well understood.

An aortic aneurysm is a localized, permanent dilation of the aortic wall, defined in most surgical literature as having a

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diameter of at least 50% more than that of the expected normal aorta diameter at the level of the diaphragm. A true aneurysm is an aneurysm contained by all 3 layers of the aortic wall; the intima, media, and adventitia. A false aneurysm or pseudoaneurysm is an outpouching through a defect that is partly or completely through the arterial wall and contained only by the adventitial layer or periarterial connective tissue. False aneurysms commonly have a neck between the vessel lumen and the aneurysm sac, corresponding to points of penetration and containment, respectively.[5]

In studies of endovascular treatment of CoA, a distinction is not always made between true and false aneurysms, and the anatomic definition of an aneurysm has varied from clearly stated absolute or relative size criteria to no specific criteria at all. For example, the prospective multicenter Coarctation of the Aorta Stent Trial (COAST) defined an aortic aneurysm as any localized widening or dilation of the aorta, without specific size criteria,[6–8] whereas studies conducted by the Congenital Cardiovascular Interventional Study Consortium (CCISC) defined an aneurysm as a >10% expansion of the aorta outside the stent or normal aorta that was not present before the intervention.[9,10] One single-center study defined an aortic aneurysm as a defect in the aortic wall extending >3 mm beyond the presumed adventitial plane in the radial dimension, with an angle of incidence >30° relative to the adjacent aorta and a discrete length.[2] Pedra et al[11] further classified the size of aneurysms, with a small aneurysm being >3 mm and ≤50% the diameter of the descending aorta at the level of the diaphragm, a moderate aneurysm ≥50% of the diameter of the aorta, and a large aneurysm ≥50 mm. Most reports do not clearly state the criteria used to define post-BA or poststent aneurysms. This ambiguity makes it difficult to determine the incidence, distribution of type and size, and natural history after endovascular interventions, and complicates decision making about the timing and method of treatment.

An aortic dissection is a tear into the intima and media that extends beyond the intimal disruption in the axial dimension, permitting extraluminal contrast to track proximally or distally.[2] A dissection often occurs in the absence of preceding aneurysm formation.[5] Waller et al[12] distinguished between a therapeutic tear and aortic dissection with respect to coronary BA, defining a therapeutic tear as involving ≤50% of the vessel circumference and extending <1 cm longitudinally, whereas a dissection is defined as a defect that involves >50% of the circumference and extends >1 cm longitudinally. Dissection is generally not defined precisely in series of CoA therapy.

An aortic rupture is a complete disruption of the aortic wall, with angiographic evidence of extravasation of contrast beyond the confines of the aorta and into the mediastinum or pleural space if not contained.[2] An aortic rupture can also manifest as an aortobronchial or aorta-to-pulmonary artery communication.

For the purposes of discussion, the definitions of dissection and rupture are useful, but are not necessarily those applied in clinical series of CoA therapy, which typically do not define these forms of AWI. In this review, AWI will be used to refer to all of the above anomalies, both when they occur acutely during treatment and when observed later, during follow-up.

Aortic Wall Complications in Unrepaired CoA

The natural history of CoA was first described in the early 20th century, with a subsequent report confirming the poor prognosis, namely, 50% mortality by 32 years of age and aortic rupture in 21% of cases.[13] Spontaneous aneurysm formation is also a well-described phenomenon in unrepaired CoA, most commonly involving the ascending aorta, with eventual rupture.[14,15] False aneurysms involving the descending aorta have also been described, thought to be secondary to endothelial injury from the high-velocity jet emanating from the stenosis proximally, and are sometimes complicated by infective endarteritis and formation of mycotic false aneurysms.[16,17] A more recent consensus summarized the dismal prognosis of untreated CoA, which is characterized by 80% mortality from associated complications, with approximately one quarter of deaths from aortic dissection or rupture.[5]

Anatomic and Physiological Factors Potentially Contributing to AWI in CoA Patients

Cystic Medial Necrosis

Cystic medial necrosis (CMN), characterized by depletion and disarray of the medial elastic fibers, is commonly associated with CoA both proximal and distal to the narrow segment. Whether CMN in CoA patients is congenital or acquired is controversial, with evidence supporting both arguments. Even so, it has been implicated in the pathophysiology leading to AWI.[18] Turner syndrome (45 X,O) is a genetic syndrome commonly associated with hypertension, CoA, bicuspid aortic valve (AOV), and aortic dilation, which are all risk factors for AWI.[19,20] Similar to patients with connective tissue abnormalities, CMN has been reported in association with Turner syndrome, further increasing the risk of AWI in these patients.[19]

Bicuspid AOV

A bicuspid AOV is present in more than half of patients with a CoA overall, and approximately two thirds of those with a native or repaired CoA and an aneurysm involving the ascending or descending aorta.[21] However, other studies found no

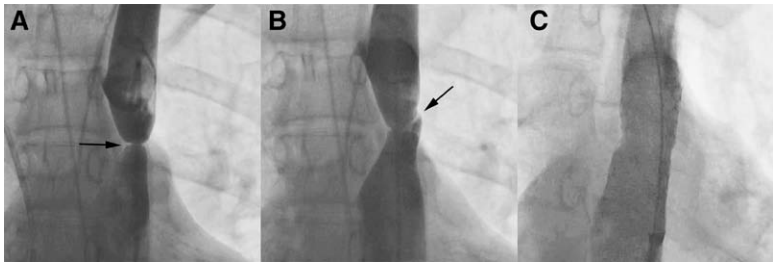

Figure 1. This series of angiograms were obtained in an adult with a native aortic coarctation (CoA). **A**, This angiogram before any intervention shows a discrete CoA at the aortic isthmus. **B**, After balloon angioplasty, the caliber of the CoA is larger and there is a therapeutic tear along the superior-rightward border of the dilated segment. **C**, After bare metal stent placement, the flap associated with the therapeutic tear is no longer visible, and there is no other evidence of aortic wall injury.

association between bicuspid AOV and postoperative aneurysms at or near the CoA repair site.[22] Isolated bicuspid AOV, occurring in 1% to 2% of the population, is identified in 9% to 24% of cases of aortic dissection, with most showing some degree of CMN in addition to underlying hypertension. The association between CoA, bicuspid AOV, and CMN suggests an interaction among these factors or a unifying arteriopathy, which could predispose to AWI.[23]

Systemic Hypertension

Late hypertension is documented in 30% to 40% of patients after CoA repair.[24–26] The underlying pathophysiology includes residual CoA, increased aortic stiffness, hyperactivity of the renin–angiotensin system, impaired baroreflexes, peripheral vascular dysfunction, and abnormal arch geometry.[25,26] In addition, older age at the time of CoA surgery has been associated with a higher incidence of late hypertension.[27] In turn, hypertension has been identified as one of the major contributing factors in the development of AWI and resulting mortality both in native and postoperative CoA.[27]

Vascular Properties and Calcification

Abnormal arterial compliance has been implicated as a risk factor for AWI. In addition, advanced age is considered a risk factor for AWI, likely secondary to aortic wall calcification and consequently diminished aortic elasticity.[9] Although there are noninvasive methods of characterizing gross aortic or arterial stiffness, assessment of focal elasticity at the site of and adjacent to a CoA may best be accomplished with balloon compliance testing, as required in the COAST.[6–8]

AWI and Abnormalities in Patients With Surgically Repaired CoA

In 1944, Clarence Crafoord performed the first surgical repair on a patient with CoA by resecting the stenotic segment and repairing the aorta with an end-to-end anastomosis. Various other techniques were subsequently trialed, including patch augmentation, interposition and bypass tube grafts, and subclavian flap arterioplasty.[28] Aortic aneurysm formation after surgical CoA repair is a well known late outcome, and there is an extensive literature focused on the occurrence and management of this issue, which is not exhaustively reviewed here.

Frequency and Risk Factors for AWI After Surgical CoA Repair

Aortic aneurysms have been reported in 9% to 20% of patients after CoA repair, with a mean duration from surgery to diagnosis of >12 years.[27,29–31] Aneurysm rupture has accounted for ≈7% of mortality after CoA repair.[27] In general, false aneurysms tend to arise at the site of suture lines or aortic isthmus restenosis, whereas true aneurysms usually develop opposite a patch. It has been speculated that synthetic patch repair of CoA increases wall strain and produces significant regional strain gradients that predispose to aneurysm formation. After subclavian flap repair, aneurysms can occur as a result of degeneration of the residual pathological aortic tissue.[27,29–31] The frequency of postoperative aneurysms varies depending on the type of repair, and is highest after Dacron patch aortoplasty (≤51%), which has led to avoidance of this technique if possible.[29,32] The reported incidence of aneurysms after other surgical techniques also varies considerably: 17% after subclavian flap aortoplasty, 6% after tube graft repair, and 3% after end-to-end anastomosis.[28,33] In addition, advanced age and postoperative hypertension have been implicated in the development of AWI.[26,30] Association with bicuspid AOV or CMN should also support aggressive control of postoperative hypertension, along with more frequent monitoring, although these factors have not specifically been associated with a higher risk of adverse aortic outcomes.[30]

Postoperative Follow-Up of AWI

The relatively high incidence of aortic aneurysms after surgical repair of CoA has led to a frequent practice of routine surveillance using computed tomography (CT) or magnetic resonance imaging (MRI). Echocardiography is inadequate for detection of aneurysms after CoA repair, with a sensitivity of 24%.[22] MRI is the gold standard for evaluating AWI after surgical repair or BA.[34] Although MRI has historically been limited in patients with stents because of magnetic susceptibility and metallic stent artifact, with new sequences and MRI compatible stents it is becoming a feasible and attractive alternative in many patients.[35,36] In most patients with CoA stents, CT is considered the best method of surveillance for AWI and other stent-related complications. It has been suggested that CT should be performed 3 to 6 months after the procedure in the absence of significant procedural complications or clinical concerns, and then at least every 5 years.[35,37]

Treatment of Postoperative AWI and Abnormalities

The natural history of postoperative aortic aneurysms is associated with a variable (36%–100%) rate of rupture within 15 years of detection, making conservative management inappropriate.[30,38] Postoperative aneurysms are often stable for many years, but may grow rapidly and lead to sudden death from development of an aortobronchial fistula, infection, rupture, or dissection.[38] Treating postoperative CoA-related aneurysms with observation alone is associated with a short-term mortality of 36%,[39] compared with 9% to 23% for surgical repair.[39–41] In addition to the significant mortality associated with surgical repair of postoperative complications including aortic aneurysm, there is a high incidence of nonlethal morbidity.[40,41] Because of the associated high mortality and morbidity with surgical repair despite the known natural history of aortic aneurysms, the decision to intervene and appropriate timing in asymptomatic patients with an aortic aneurysm after CoA repair is difficult.[42]

Thoracic endovascular aneurysm repair (TEVAR) has emerged as an alternative to surgery for postoperative aortic aneurysms, and has been most widely applied in cases of large fusiform aneurysms after patch aortoplasty.[39,43] Factors that may preclude TEVAR include a small proximal lumen diameter, a short landing zone, and an acute angle of the thoracic arch. An important limitation in the pediatric population is the large caliber of current TEVAR systems, which may not be compatible with small ileofemoral vessels in children. Reported case series show that TEVAR is relatively safe and

effective for treating aneurysms associated with CoA repair, with major complications and mortality uncommon and few reported cases of post-TEVAR aortic rupture.[39,41,43–46]

AWI After Endovascular Treatment of CoA

Endovascular treatment of CoA was introduced >3 decades ago, with BA first reported in 1982,[47] and placement of bare metal stents (BMSs) used by 1989.[48] Transcatheter therapy, including BA alone, BA followed by stenting, and primary stenting with BMS or covered stents, has since become a widely accepted alternative to surgical repair of both native and recurrent CoA.[41,47,48] AWI is one of the most important potential complications of both BA and stenting, but the data on this topic are variable and have not been synthesized comprehensively. The true incidence of pathological AWI, including aneurysm, dissection, ,and rupture, is unknown because of varying definitions and terminologies. Moreover, a clear distinction is generally not made in the literature between true and false aneurysms.

Acute and Early AWI Related to Catheter Therapy

Aortic Rupture

Aortic rupture is the most serious and feared complication of endovascular treatment for CoA, and may quickly lead to circulatory collapse and death.[36] The first fatal case after endovascular treatment for CoA was reported in 1983 by Finley et al[49] after attempted BA in an infant. Subsequent experience supports an incidence of <1% after both BA and stent placement.[9,50–53] A current review of the literature identified 23 cases of aortic rupture after endovascular treatment for native or recurrent CoA (Table 1), including 12 individual case reports and 11 cases included in larger series, although many more have likely occurred.[2,49,52,54–73] The CoA was native (no previous surgery or endovascular therapy) in 7 patients, recurrent after previous surgery in 14, and unspecified in 2. Of the 14 patients with recurrent CoA, 12 originally underwent surgical repair using various techniques (end-to-end anastomosis in 4, patch aortoplasty in 2, subclavian flap aortoplasty in 2, a Norwood arch reconstruction in 1, and unspecified in 4) and 1 had previous BA but no surgery. The rupture occurred after BA in 9 patients, BMS implantation in 11, covered stent placement in 2, and an unspecified intervention in 1. Predilation was reported in only 2 of the 13 patients who underwent stenting.

Eleven patients died after aortic rupture, including 2 of the infants. In 6 of the deaths, the rupture was recognized immediately after the intervention in the catheterization laboratory, whereas 2 became symptomatic in the recovery room, and 3 presented 1 to 5 days later. Overall, 7 patients, including 6 who died, were identified to have an aortic rupture an hour or more after the procedure, which highlights the importance of timely identification and treatment. Six of these 7 patients had stents placed, and their delayed decompensation suggests that the rupture did not occur immediately and may have progressed from a lesser AWI. Because a single biplane angiogram may not provide adequate visualization of the entire aortic circumference to evaluate AWI after intervention, angiography from multiple projections or with a 3-dimensional technique may be most sensitive for immediate identification of AWI.

During the early experience with BA, it was suggested that postoperative periaortic fibrous scarring may protect against aortic dissection or rupture, making BA safe for treatment of recurrent CoA.[74] However, the relative predominance (59%) of reported ruptures in patients with postoperative CoA suggests that the resulting scar is not necessarily protective. In fact, it has been speculated that the rigid fibrous tissue cannot expand with BA and may confer a risk of rupture. In support of this theory, the only reported postrupture autopsy after BA for recurrent CoA showed a longitudinal tear traversing the intact end-to-end anastomosis suture line.[56] The reported cases of rupture comprised a fairly even number of patients who had undergone BA and stent placement, suggesting that both modes of treatment carry an inherent risk of this complication. Although the relative risk of rupture with BA and stenting cannot be determined from these limited data, the balance of reported cases might suggest that neither type of intervention is substantially riskier than the other. As can be seen in Table 1, potentially important information, including data on balloon and aortic diameters, was not provided in many of the reports. As a result, it is not possible from the available literature to identify technical risk factors for angioplasty/stent-related aortic rupture. Autopsy findings were described for 3 patients who died from aortic rupture after stenting: in the first, the rupture was adjacent to the stent edge, a location felt to be vulnerable to AWI during CoA stenting; in the second, although the stent overlapped an associated aneurysm which subsequently ruptured, the covering did not; in the third, the rupture was within the distal half of the second stent, which was placed after the first stent migrated distally and created AWI in that location.[63,67,70]

Aortic Aneurysm and Dissection

As demonstrated in Table 2, there is a remarkable variability in the reported frequency of aneurysms forming acutely after BA for CoA, anywhere from 0% to 13% of patients.[11,50,54,67,75–83] The earliest registry study of BA for CoA, which included 907 patients and is not included in Table 2, reported 4 transmural tears, which may represent any or various of the defined AWI types.[84] In addition, many series did not specifically report on AWI. As also summarized in Table 2, the reported incidence of AWI after BMS therapy for CoA is typically lower than with BA, on the order of 0% to 5%,[2,7–11,22,37,52,61,68,85–97] and many clinicians consider stenting to be the first line therapy in adults and large children. Among patients treated with balloon expandable covered stents, acute aneurysms have been reported in <1%.[2,9–11,22,37,52,68,85,86,92–94,97–102] In 5 studies of BMS treatment for CoA that included a total of 119 patients, there were no reported aortic aneurysms or dissections,[85,103–107] and some early studies did not mention assessment for AWI.[92] In series that reported ≥1 cases with AWI, 55 out of a total of 3541 procedures (1.6%) reportedly experienced acute AWI, and new AWI were noted on follow-up in 108 patients (3%), as detailed in Table 2. This does not include series in which AWI was not specifically mentioned, or in which no AWI was reported (at least 5 studies including 119 patients), or the 907-patient registry report mentioned above that did not include details on type or timing of 4 AWI.[84] Incorporating those cohorts, acute AWI was reported in 1.3% of CoA interventions (59 of 4567

procedures) and either acute or late AWI was reported in 3.7% (167 of 4567 procedures). Given the variability in reporting and of follow-up, deriving a composite estimate of acute AWI incidence from the reported literature is probably fraught with bias, but simple compilation of the reports mentioned above may be the best available estimates of the frequency of AWI after endovascular CoA therapy.

Of note, in the prospective, multicenter single-arm COAST, which evaluated acute and follow-up AWI systematically, 4 of 112 patients (3.6%) were initially reported to have small aortic aneurysms after prestent BA, all of which were treated with a covered Cheatham-platinum stent (NuMED, Hopkinton, NY).[7] In a subsequent report, the authors noted that core laboratory review determined those 4 injuries to be therapeutic tears rather than aneurysms. Two additional patients were determined to have small aneurysms after compliance testing, one of which was treated with a covered Cheatham-platinum stent.[8] No aneurysms were noted after stent implantation, but 1 patient (0.9%) developed a localized dissection after BMS placement, which was noted on CT imaging the following day to have healed without intervention.[7] These findings point both to the inconsistencies in defining aneurysms between providers and the importance of multimodal imaging for detecting AWI. In the multicenter CCISC study of CoA stenting, only 1% of patients developed aneurysms acutely.[9] In a large single-center series of stent therapy for native and recurrent CoA, acute aneurysms were diagnosed in 2% of 153 patients; the authors concluded that serious injury to the aortic wall is uncommon, and can be minimized with a focus on technical measures.[2] Data from other studies are summarized in Table 2, but are difficult to synthesize concisely given the variability in findings and data.

In most reports, the term aneurysm is used without further specification. However, given the presumed mechanism of AWI in most cases, extension of the intimal disruption into and through the media, it is likely that most aneurysms associated with endovascular CoA therapy are actually false aneurysms. Like therapeutic tears, aneurysms and dissections associated with BA tend to be focused at the dilated segment, while in patients treated with stenting, the location is more variable, sometimes at the level of the CoA and other times adjacent to the ends of the stent. By angiography, protrusion of stent ends beyond the luminal plane can be seen after stent therapy in some cases in the absence of AWI, but the implications of this appearance and its potential connection to AWI are unclear (Figure 2). Furthermore, in a few reported cases, AWI has been associated with stent migration or fracture.[37,103] Although stenting, and covered stent therapy in particular, seems to be associated with a lower risk of AWI than BA alone, there is limited information on patient-related or procedural risk factors for any of the aortic wall complications discussed in this review. Unfortunately, the quality and quantity of data in this regard are limited, given the variability in definitions and the lack of systematic or serial follow-up imaging.

Natural History of Early AWI and Development of New Aortic Wall Complications Over Time

Therapeutic tears, a desired outcome after BA, have been implicated in the development of AWI. However, clear documentation of such progression is lacking. A study by Sohn et al[1] using IVUS identified therapeutic tears in all 17 patients after BA for CoA (12 with native CoA and 5 with recurrent CoA), with no acute aneurysms. IVUS was more sensitive than angiography in detecting therapeutic tears, suggesting that many therapeutic tears go unnoticed, as IVUS is not commonly used during endovascular CoA procedures. None of the 9 patients who underwent follow-up imaging an average of 2.3 years later had aneurysms, and in 4, IVUS and angiography demonstrated evidence of healing and remodeling, with diminution in size or disappearance of the therapeutic tears. Similarly, Walhout et al[51] documented therapeutic tears by angiography in 8 of 29 adolescents and adults after BA for native CoA, with no aneurysms. On 3-month follow-up angiography, half of the tears remained unchanged, and the other half had healed. The tendency for therapeutic tears to heal was also seen in an animal model of BA for CoA, which documented resolution of therapeutic tears within 2 months of the procedure and no evidence of aneurysm formation.[108] Nevertheless, to understand fully the prevalence and remodeling of therapeutic tears after CoA intervention, it will be necessary to standardize definitions and to use more sensitive imaging modalities at the time of intervention and during follow-up.

The collective understanding of the evolution and significance of aneurysms after endovascular treatment of CoA is limited by a lack of long-term follow-up data with comprehensive and consistent serial imaging. The available literature, which is summarized in Table 2, indicates that significant progression of acute AWI or formation of new aneurysms is uncommon but does occur, as also demonstrated in Figure 3. Although the published data are variable and limited, they can nevertheless serve as an important foundation for understanding this issue.

On intermediate follow-up in the COAST at 2 years, which included CT, MRI, or angiography in 86% of patients, 4 new aortic aneurysms were identified. One patient was found by MRI to have a large aneurysm at the margin of the previously placed BMS stent, and 3 patients who underwent planned cardiac catheterization for stent re-expansion had angiographic evidence of small stent-related aneurysms that were not apparent on previous MRI or CT imaging. All these were treated with a covered Cheatham-platinum stent.[8] In the CCISC study, which reported a 1% incidence of acute pathological AWI, 27% of patients were evaluated with MRI, CT, or repeat catheterization a median of 12 months after intervention, 9% of the patients with follow-up imaging were found to have aneurysms, most of which were small and at the level of the CoA, and 3% had a dissection or intimal tear.[10] In another long-term follow-up study of 58 adolescents and adults with native CoA after BA, 4 (7%) had developed aneurysms at the site of dilation by 1 year: in one of these patients, the aneurysm was larger on follow-up MRI 20 years later, and in 2 others with late imaging there was no appreciable change in size.[50] Five of 36 patients who underwent stent placement or BA for native CoA reported by Pedra et al[11] developed small aneurysms or dissections acutely, but on imaging an average of 1 to 1.5 years later, both dissections had disappeared and there was no progression of the aneurysms. In the report from Boston,

Table 1. Reported Cases of Aortic Rupture Related to Endovascular Therapy for Aortic Coarctation

Publication	Age, y	Previous CoA Intervention	Angioplasty or Predilation	Stent	CoA Diameter, mm	Proximal/Distal Ao Diameter, mm
Rupture after BA						
Finley[49]	0.1	None, nCoA	Yes	None	NR	NR
Hellenbrand[54]	NR	Yes, type NS	Yes	None	NR	NR
Kulick[55]	30	None, nCoA	Yes	None	7	20/NR
Balaji[56]	8	Patch aortoplasty (age, 12 d)	Yes	None	NR	14/NR
Roberts[57]	25	ETEA (age, 5 y)	Yes	None	NR	23/NR
Qureshi[2]	0.1	Stage 1 Norwood (age, 1 wk)	Yes	None	NR	NR
Motz[58]	0.1	Yes, type NS	Yes	None	NR	NR
Ogasawara[59]	22	SFA (age, 2 y), 3 previous BA	Yes	None	NR	NR
Wu[60]	9	Patch aortoplasty (age, 1 mo) Turner syndrome	Yes	None	7.8	12.2/13.9
Rupture after stent implantation						
Suàrez de Lezo[61]	0.1	SFA (age, 6 wk)	Yes	Palmaz (NS)	NR	NR
Korkola[62]	44	None, nCoA	Yes	BMS 40 mm (NS)	NR	NR
Varma[63]	65	None, nCoA	No	Palmaz P5014	6	24/30
Tan[64]	33	ETEA (age, 3 y)	No	Palmaz P4014	8	18/22
Collins[65]	51	None, nCoA	No	CCPS 50 mm	6	22/26
Alcibar[66]	62	ETEA (age, 29 y)	No	Palmaz P4014	5	16/18
Rodés-Cabau[52]	NR	None, nCoA	NR	NR	NR	NR
Reich[67]	NR	BA (4 y prior)	NR	Palmaz P4014	NR	NR
Moltzer[68]	NR	Yes, type NS	NR	BMS (NS)	NR	NR
Erdem[69]	NR	NR	NR	BMS (NS)	NR	NR
Alcibar[70]	57	None, nCoA	No	CCPS 45 mm	0	NR
Butera[71]	NR	ETEA, BA	NR	BMS	NR	NR
Ostovan[72]	26	NR	NR	BMS (NS)	NR	16/NR
Endovascular procedure not specified						
Brown[73]	NR	Yes, type NS	NR	NR	NR	NR

(*Continued*)

Angio indicates angiogram; Ao, aortic; BA, balloon angioplasty; BMS, bare metal stent; cath, catheterization; CCPS, covered Cheatham-platinum stent; CoA, aortic coarctation; CT, computed tomography; ETEA, end-to-end anastomosis; nCoA, native aortic coarctation; NR, not reported; NS, not specified; and SFA, subclavian flap aortoplasty.

there was no progression of acute AWI at follow-up, and new aneurysms were seen in 6% of patients a median of 2.8 years after intervention.[2] Other series with relatively short follow-up similarly reported no progression of acute aneurysms.

In most studies, the number of aneurysms detected during follow-up was sufficiently small that evaluation of risk factors would not be possible. In the CCISC report, it was noted that the incidence of AWI decreased over the study period,[10] and in the study by Hoffman et al,[22] the authors reported a higher incidence of follow-up aneurysms in patients who were initially treated beyond 1 year of age and those who had ≥3 CoA procedures. There is a suggestion in the literature that late aneurysm formation may be more common after treatment of native than recurrent CoA.[41] The data are limited, but based on the cases in which the location of new aneurysms was reported, they seem to occur at similar locations as acute

Table 1. Continued

Balloon Diameter, mm	Procedural Comments	Timing of Rupture Diagnosis	Treatment	Outcome
NR	Lost guidewire position after BA, angiographic catheter used to cross CoA	Immediately after dilation (angio)	None	Died
NR	NR	Immediately after dilation	None	Died
18	Balloon slipped below CoA on first inflation, waist revolved with second inflation	Immediately after second dilation (angio)	Emergent surgery	Survived
15, 12	15-mm balloon ruptured during second inflation	Immediately after second balloon dilation (angio)	None	Died
15, 19, 23	15-mm balloon ruptured during first inflation	Immediately after dilation (angio)	Emergency thoracotomy	Died
NR	…	NR	Stent, coil embolization	Survived
6, 7	Angiogram after second dilation showed small aneurysm	5 d after cath, poor feeding and emesis, cardiac arrest	None	Died
NR	…	Immediately after balloon dilation (angio)	Emergent surgery	Survived
14	…	Immediately after dilation (angio)	16-mm endograft extension implanted surgically	Survived
7	Balloon ruptured during deployment, stent migrated distally	Immediately after postdilation of stent (angio)	Second stent placed	Survived
20	…	8 h after cath, sudden chest/back pain (CT)	Second stent, followed by emergent surgery	Survived
20, 25	Stent migrated distally during second inflation of 25-mm balloon; second stent placed with 15- and 20-mm double balloons	Circulatory collapse in recovery room, returned to cath laboratory (angio)	Attempted covered stent	Died
20	Second inflation to address residual waist	Immediately after second inflation (angio)	CCPS	Survived
18	Second inflation to fully deploy distal stent	1 h after cath, chest pain (CT angio)	Second 50-mm CCPS	Survived
16	…	Immediately after stent deployment (angio)	CCPS	Survived
NR	Contained rupture	Immediately after stent deployment	Emergent surgery	Survived
NR	…	28 h after cath	None	Died
NR	…	Immediately after stent deployment	CCPS	Died
NR	…	Immediately after stent deployment (angio)	CCPS	Survived
NR	Preexisting aneurysm appeared excluded, but was not on autopsy	2 d after cath, massive hematemesis	None	Died
NR	Preexisting aneurysm coil occluded	2 h after cath	NR	Died
18	…	Immediately after stent deployment (angio)	Balloon inflated, transferred to operating room	Died
NR	…	NR	Emergent surgery	Survived

AWI, both in patients treated with BA alone and those in whom a stent was implanted.

Covered Stents and AWI Associated With Endovascular CoA Therapy

Balloon expandable covered stents have become important tools in the management of CoA, although they are not yet approved in the United States for this indication. Even with the reduced risk of AWI after staged BMS expansion, covered stents have been advocated for primary therapy on the basis of their additional sealant effect, which may prevent or limit acute AWI and allow a more aggressive approach in high-risk situations.[66,109] Covered stents are also used to exclude existing aneurysms after previous endovascular repair or surgery (Figure 3).[40,109] In addition to the fact that it is covered, the most commonly used balloon expandable covered stent, the

Table 2. AWI After Endovascular CoA Therapy as Reported in Selected Single Center, Multicenter, and Review Articles

Author	Study Type*	No. of Patients	Acute AWI		
			Acute Aneurysm	Acute Dissection	Treatment of Acute AWI
Balloon Angioplasty					
Hellenbrand[54]	Multicenter, ReCoA	200	NR	2 Small and 1 moderate (5 mm)	Surgical repair 1 moderate dissection
Tynan[75]	Multicenter, nCoA	140	2	NR	NR
Booth[76]	nCoA and ReCoA	34	1 Small	1	Surgical repair 1 dissection
Fawzy[77]	nCoA	23	1 Small	1 Small	Surgical repair 1 dissection
Paddon[78]	nCoA	17	1	1 (Same patient, 2 cm)	Surgical repair 1
Cowley[79]	nCoA	36	NR	NR	NR
Fawzy[50]	nCoA	58	None	1	Surgical repair 1
Reich[67]	ReCoA	99	NR	NR	NR
Adjagba[80]	nCoA and ReCoA	25	1	None	None
BMS implantation					
Suárez de Lezo[87]	nCoA and ReCoA	48	1 (after treatment of aortic rupture)†	None	None
Ledesma[88]	nCoA and ReCoA	54	1	None	None
Harrison[89]	nCoA and ReCoA	27	NR	NR	NR
Johnston[90]	nCoA and ReCoA	32	NR	NR	NR
Mahadevan[91]	nCoA and ReCoA	37	None	1	None
Ringel[7]	Multicenter, nCoA, and ReCoA	105	4 Small (after prestent balloon)	1 Small	CCPS 4 aneurysms
Meadows[8]	Multicenter, nCoA, and ReCoA	105	2 Small	None	1 CCPS
Covered Stent Implantation					
Tzifa[98]	Multicenter, CCPS, nCoA, and ReCoA	30	None	1 Small	None
Bruckheimer[99]	CCPS and nCoA	22	None	1 Small	CCPS 1
Tanous[100]	CCPS, nCoA, and ReCoA	22	1 (Leading to aortic rupture)	None	CCPS 1
Sadiq[101]	CCPS, nCoA	56	None	1	NR
Vanagt[102]	CCPS, nCoA, and ReCoA	51	None	NR	None
BMS and covered stent implantation					
Cheatham[92]	Multicenter, BMS or CCPS, nCoA, and ReCoA	21	None	None	None
Shah[93]	BMS or CCPS, nCoA, and ReCoA	44	1 Small	1 small	None
Forbes[9]	Multicenter, nCoA, and ReCoA	555	6	9	Surgical repair 3 dissections, covered stent 3 dissections
Forbes[10]	Multicenter, nCoA, and ReCoA	578	NR	NR	NR
Qureshi[2]	nCoA and ReCoA	153	1	1 (4 cm)	Covered stent 2
Chakrabarti[37]	BMS or CCPS, nCoA, and ReCoA	88	None	None	None
Moltzer[68]	BMS or CCPS, nCoA, and ReCoA	24	None	None	None
Butera[71]	nCoA and ReCoA	143	None	NR	None
Sohrabi[94]	BMS or CCPS and nCoA	120	None	None	None
BA and stent implantation					
Macdonald[95]	BA or BMS, nCoA, and ReCoA	BA:13 Stent:15	None	BA: 1 small Stent: None	None
Zabal[96]	BA or BMS, nCoA	BA:32 Stent:22	BA: 2 small Stent: none	None	Surgical repair 1
Pedra[11]	BA, BMS or CCPS, and nCoA	BA:15 Stent:21	BA: 2 small Stent: 1 small	BA: 2	None
Rodés-Cabau[52]	Multicenter, BA, BMS or CCPS, and nCoA	BA:31 Stent:19	BA: 1 small (3 mm) Stent: none	NR	None
Forbes[97]	Multicenter, BA or stent, and nCoA	BA:61 Stent:217	BA:0 Stent:0	BA:6 Stent:0	NR
Hoffman[22]	Multicenter, BA or BMS, nCoA, and ReCoA	BA:105 Stent:43	NR	NR	NR

(*Continued*)

This table does not include reports with 10 or fewer patients, and does not include series in which AWI was not mentioned or in which no cases of AWI were observed acutely or during follow-up. AWI indicates aortic wall injury; BA, balloon angioplasty; BMS, bare metal stent; CCPS, covered Cheatham-platinum stent; Max, maximum; Min, minimum; nCoA, native aortic coarctation; NR, not reported; NS, not specified; and ReCoA, recurrent aortic CoA.

*Study type is single center unless specified otherwise.

Table 2. Continued

Follow-Up		
Duration Mean, y (Min–Max), No. of Patients	New AWI	AWI Treatment
None	NR	NR
NR	6	Surgical repair
None	NR	NR
1.3 (0.3–4), 100%	1 Small aneurysm	None
5.4 (1.5–11), 100%	None	None
10.6, 56%	7 Aneurysms (3 small, 4 large)	Surgical repair 2 large aneurysms
12 (1–22)	3 Small aneurysms (2–2.3 cm), 1 large aneurysm (4 cm)	Surgical repair 1 large aneurysm
Median 13.4, 100%		
Median 20.7, 100%	1 small aneurysm (2.1 cm)	Surgical repair during ReCoA repair 1
nCoA: 2.2	2 Aneurysms	None
ReCoA: 3.2, 100%		
1.9, 100%	2 Small aneurysms	Coiled 2
2.1 (0.2–4.3), NR	2 Small aneurysms	None
1.8 (0.1–3), 100%	1 Aneurysm (2 cm)	Surgical repair 1
1.5, 97%	1 Dissection (2 days postprocedure)	Surgical repair 1
NR	3 Small aneurysms	None
0.1, 96%	NR	NR
2, 86%	3 Small aneurysms, 1 large aneurysm	4 CCPS
0.9	None	None
Median 1, 100%		
1.5 (0.1–2.6), 100%	None	None
1 (0.8–1.3)	None	None
Median 1, 100%		
Median 3.8, NR	None	None
NR	1 Aneurysm	CCPS 1
NR	1 Small aneurysm, 1 large aneurysm	Surgical repair 1 large aneurysm
1.7 (0.1–9.8), 91%	1 Large aneurysm	Surgical repair 1
NR	NR	NR
Median 1 (0.1–7.7), 100%	13 Aneurysms, 5 dissections	Intervention (type NS) 4 aneurysms
Median 2.5 (0.2–12.5), 93%	5	NR
Median 2.9 (0.3–6.1), 97%	1 Aneurysm	CCPS 1
2.8 (0.7–6.4), 92%	1 Aneurysm	CCPS 1
4.8, 94%	3 Aneurysms (BMS group)	CCPS 3
2.6 (1–5), 100%	2 aneurysms (CCPS group)	CCPS 2
BA:9 (3–10), 100%	None	None
Stent:3 (1–5), 100%		
3.6, NR	None	None
BA: 1.5	Stent: 1 moderate aneurysm	CCPS 1
Stent: 1, NR		
2.7, 84%	BA: 9 aneurysms Stent: 1 aneurysm	Surgery 1, Stent and coils 4
BA: 2.1,77%	BA: 7 aneurysms, 1 dissection	NR
Stent:1.9,68%	Stent:3 aneurysms, 1 dissection	
10 (0–30.3), 100%	BA:15 aneurysms Stent:2 aneurysms	NR

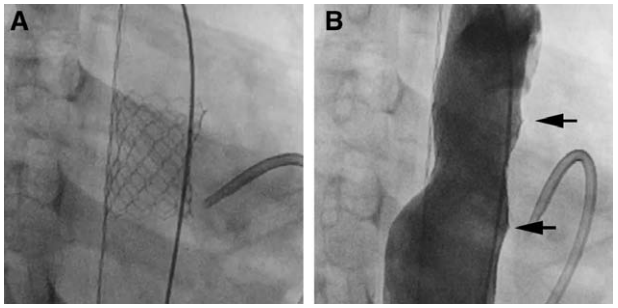

Figure 2. A, This fluoroscopic image demonstrates a bare metal stent at the level of the aortic isthmus. **B**, An angiogram in the same projection in the same patient reveals protrusion of the superior and inferior stent edges (arrows) and distortion of the aortic lumen, with no evidence of aortic wall injury.

covered Cheatham-platinum stent, is designed with the aim of preventing AWI, with rounded stent ends that are intended to reduce trauma, and composition from platinum–iridium, which is more pliable than other stent materials and thus allows better conformability to the aortic wall.

On the basis of a growing literature, it seems that covered stents are associated with a lower incidence of intrastent aneurysm formation and aortic rupture (<1% in most studies) than BA or BMS therapy, both acutely and at follow-up, and confer the advantages of sealing tears within the stented region and excluding preexisting aneurysms.[43,86] Studies in which AWI was reported after covered stent therapy are summarized in Tables 1 and 2. In addition, in 4 studies that included a total of 91 patients who received a covered stent for CoA,[72,87,69,110] and 2 series with 78 patients who received either a BMS or covered stent, no aneurysms or dissections were reported.[69,72] Although the benefits of covered stents in reducing overall AWI are supported by the literature relative to simple BA, it is less clear whether they

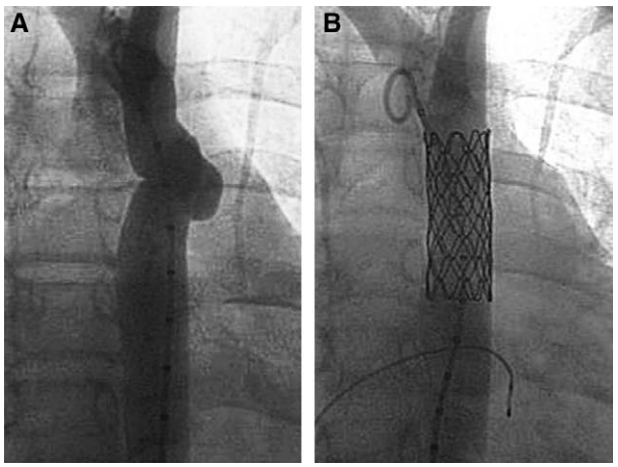

Figure 3. These images are from a patient who underwent balloon angioplasty for a native aortic coarctation (CoA) at 3 years of age. Immediately after intervention, there was a small aneurysm that was not treated. The patient was followed closely with serial computed tomography, and by 12 years of age was noted to have a moderate size aneurysm that had increased substantially over the prior few years. **A**, Before reintervention, angiography in a frontal projection showed a residual CoA shelf on the rightward aspect of the descending aorta and opposite that an aneurysm. **B**, After placement of a Cheatham-platinum stent, the aneurysm was effectively excluded and the lumen/caliber of the descending aortic normalized.

maintain the same advantage compared with BMS. Only short- and intermediate-term follow-up exist, and various instances of AWI have been described after covered stent therapy, including aortic rupture, aneurysm, and dissection (Tables 1 and 2), often at the edges of the stent.[94,111] The only 2 published studies that have directly compared BMS and covered stents show conflicting data. In a comparative study of 71 patients who received a BMS with 72 who underwent covered stent implantation for native or recurrent CoA, Butera et al[71] identified 3 false aneurysms during a mean follow-up of 81 months in the BMS cohort, but no AWI in the covered stent group with a mean follow-up of 35 months. In contrast, Sohrabi et al[94] reported false aneurysms in 2 of 60 patients ≈1 month after covered stent implantation used to treat native CoA, but no AWI at a mean follow-up of 2.6 years in the 60 patients who received a BMS. Both aneurysms were located proximal to the covered stent and were thought to be a result of prolonged and possibly traumatic wire manipulations. In their review of a 10-year experience with covered stent therapy for native CoA, Sadiq et al[101] reported a single case of AWI, a small aortic dissection discovered within 24 hours after the procedure. A CT scan at the time of diagnosis revealed that the proximal aspect of the covered stent was not fully apposed to the vessel wall, potentially creating turbulent flow that may have contributed to the dissection; on a repeat CT 6 months later, the dissection was unchanged. Although these and other follow-up studies have been inconsistent with respect to the benefits of covered stents over BMS, it has been proposed that the sealant effect provided by covered stents may allow a more aggressive approach to primary dilation of CoA, especially in the adult population where the risk of aortic rupture is higher.[109] Covered stents almost certainly afford protection against AWI, but aneurysms, dissections, and ruptures have all been reported after primary covered stent therapy, so they are clearly not a panacea. A recent editorial proposed that BMS is appropriate for most patients with CoA, whereas covered stents should be reserved for those determined to be at high risk or with a preexisting aneurysm.[112] It has been suggested that patients with complex CoA who may benefit from covered stent placement include those with aortic inflammatory disease, severe stenosis with a proximal aorta-to-CoA diameter ratio >3, atresia or near atresia of the aorta, ascending aortic dilation, a preexisting aneurysm, or an irregular aortic wall.[71] It should be acknowledged that current experience with covered stents may be biased toward use in patients with such features, which many implanters consider to increase the risk of AWI relative to more typical cases, which could potentially confound unadjusted comparisons between covered stents and BMS. Ultimately, further investigation into the long-term outcomes of covered stent placement will be necessary to clarify the relative benefits and appropriate use of this technology. In the meantime, most interventional cardiologists are in agreement that covered stents should be readily available for rescue therapy, and it seems prudent to treat acute AWI after BA or BMS therapy with an additional covered stent if available.

Other Technical Considerations Related to AWI Resulting From Endovascular CoA Therapy

Because few risk factors have been identified for important AWI during CoA interventions, the relationship between technical aspects of CoA therapy and AWI are generally

speculative. However, it is important to discuss the potential role of procedural variables in AWI, because even though there are few data to shed light on this area, it is critically important, and consideration of potentially relevant factors may enhance the collective understanding of and insight into this issue. Assuming that more pronounced oversizing of the balloon or stent relative to the CoA predisposes to a higher risk of important AWI, one of the most intuitive measures to reduce the likelihood of catastrophic aortic rupture, and potentially other significant AWI, is to approach angioplasty and stent implantation conservatively. Gradual dilation and enlargement of the CoA may allow the operator to identify therapeutic tears and small AWI at an early stage and consider whether to implant a covered stent or take a staged approach by allowing the initial tear/injury to heal before performing a second procedure to relieve the CoA completely.

Investigators have proposed various measures to reduce or mitigate the risk of AWI related to angioplasty or stenting for CoA, but there is limited evidence about the effectiveness of these strategies. Hijazi et al[113] recommended that the diameter of the balloon or enlarged stent must never exceed the diameter of the descending thoracic aorta at the level of the diaphragm or 3× the diameter of the CoA segment, and if there is a discrepancy between angiography and precatheterization imaging, IVUS may help resolve the discrepancy. In a study by Oliver et al,[21] the only risk factors for AWI after surgical or endovascular treatment for CoA were advanced age and a bicuspid AOV. The higher risk of AWI in older patients may be related to histological changes in the aging aorta that make it less resilient to expansion and potentially susceptible to injury.[114]

Another technical consideration that has been implicated in AWI is balloon rupture during dilation, although there are no data supporting this association. The mechanistic explanation for this theory is that an abrupt, high-velocity jet of balloon expansion medium directed toward the aortic wall may serve as another potential source of trauma, and subsequent inflations may enlarge the tear and lead to further damage. Although balloon rupture is less common with equipment used to treat CoA in the current era, care should be taken to evaluate for AWI after a balloon ruptures in a region where the vessel wall may be thin and predisposed to rupture, and further inflations should be performed with caution. In addition, traction on or distortion of the aorta (either in the distal arch or a tortuous aorta) because of straightening of the balloon during inflation may play a role in AWI, both at the site of CoA and remotely. The aortic isthmus serves as a natural fulcrum in the aorta, insofar as it functions as a point of transition from a relatively mobile arch to a fixed and immobile descending aorta, which may predispose to AWI during balloon-related distortion; this phenomenon may be altered, and potentially exacerbated, by scarring from previous surgery.[115] Also, manipulation of guide wires and catheters in the region of the freshly dilated CoA should be avoided if possible.[116]

Controversy remains about whether to perform BA previous stent placement. The CCISC study identified prestent BA as a risk factor for aortic wall complications, along with location of the CoA at the abdominal aorta and age >40 years.[9] Subsequent studies suggested that BA before stenting may cause disruption of the endothelium, with further sheath and wire maneuvers causing additional trauma to the vessel wall.[9,10,69] However, the authors of another large single-center study proposed that predilation before stent placement may help to avoid AWI by revealing the location and compliance of the stenosis, as well as the compliance of the surrounding aorta.[2] In the COAST study, which required low-pressure BA before stent placement to identify patients with a noncompliant CoA that would not benefit from stenting or might be at risk for rupture, all instances of acute AWI occurred after the initial BA.[6–8]

Stenting may prevent development of a dissection or aneurysm from a therapeutic tear by keeping the layers of the aortic wall laminar so that any tear within the stented region may be less likely to extend. There remains a risk of injury at the ends of the stent with both BMS and covered stents. Duke et al[117] recommended incomplete initial stent expansion followed by repeat dilation at a later procedure to further protect against pathological AWI, and although staged treatment makes sense in high-risk situations, there is no evidence that this approach is necessary or effective. In a study of 22 patients who underwent primary stent placement at a median age of 9 years for native or recurrent CoA, followed by stent redilation a median of 18 months later because of somatic growth or neointimal proliferation, the second stent expansion was successful in 20 patients without any complications. Both patients in whom the redilation was unsuccessful had Williams syndrome (1 developed a small dissection acutely after redilation, and at 3-year follow-up the other had a large false aneurysm that was surgically addressed). On the basis of this and other studies, secondary expansion of CoA stents seems to be a safe and successful procedure, making it feasible to perform staged therapy for high-risk patients or place stents in nonadult sized patients with a plan for further expansion over time to account for somatic growth.[118]

Treatment of AWI Related to Endovascular CoA Therapy

There is limited evidence from which to determine whether and when to treat AWI after BA or stent therapy. The risks of rupture or progression have not been defined in large series, so it is not clear that intervention is necessary to prevent evolution and adverse outcome. Nevertheless, there are circumstances in which treatment seems appropriate, particularly if it can be performed with relatively low risk and morbidity (eg, with endovascular methods). As previously described, TEVAR has been used as a less invasive alternative to surgical repair of postoperative aneurysms.[39,43] Balloon expandable or self-expanding covered stents have been used to exclude a wide range of aortic wall anomalies to prevent progression and possible rupture, and, as discussed above, are commonly used to treat acute aortic dissection and rupture after endovascular therapy for CoA.[41,111] Some clinicians have referred patients for elective surgical repair of post-BA/stent aneurysms, and others have proposed situational treatment. For example, Pedra et al[11] described a benign history of smaller aneurysms after endovascular interventions, which they elected to follow clinically with strict control of blood pressure levels and close imaging surveillance, whereas intervening on larger

aneurysms to reduce the likelihood of rupture. A comparative study of BMS or covered stents with endovascular stent grafts has not been performed, and the choice between these techniques seems to be based on institutional experience, but for most discrete false aneurysms, as opposed to longer fusiform aneurysms, balloon expandable covered stents may be a simpler and more flexible option. Further evaluation of the natural history of AWI after endovascular interventions is necessary to better understand when treatment is appropriate.

Conclusions

The reported incidence of acute AWI after endovascular therapy for CoA varies considerably, for reasons that are not entirely clear, but likely have to do at least, in part, with reporting bias and inconsistent definitions. Data on the incidence of new AWI during follow-up and the natural history of both acute and nonacute AWI are similarly variable, with clear ascertainment bias and a general paucity of data. Nevertheless, although important AWI after endovascular repair of CoA seems to be declining in frequency with improving experience and technology, it still stands as one of the most serious and important potential adverse outcomes. Accordingly, long-term surveillance for new AWI and monitoring of existing AWI is mandatory, with institution of appropriate treatment when necessary. Given the lack of long-term follow-up, a central research focus in this population should be determination of the appropriate treatment for both native and recurrent CoA across various ages with regard to limiting recurrent CoA and preventing associated morbidity and mortality, especially that resulting from AWI, in addition to determining the appropriate treatment of aortic aneurysms, dissection, and rupture. To truly understand the incidence of, risk factors for, and measures potentially protective against AWI after BA or stent implantation for CoA, standardized definitions and reporting are necessary. Moreover, the fact that most stents currently implanted for CoA in the United States are used off-label hampers a consistent approach to reporting device or therapy-related adverse events.

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