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REVIEW ARTICLE

Comprehensive Management of Coarctation of the Aorta; Modern Approaches and Updated Strategies for Adolescents and Adult

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Abstract

In adolescents and adults with coarctation of aorta (CoA), whether it manifests as native CoA or recurrent CoA, transcatheter CoA intervention has emerged as widely accepted alternative to surgery. Rapid advancements in techniques and refinement of stent technology have driven a significant paradigm shift in clinical practice. This manuscript offers detailed review of the current management strategies for CoA, with particular focus on adolescents and adults. It provides updated insights into current indications for intervention, procedural tips and techniques, and addresses both immediate and long-term outcomes of CoA repair in adults. Through this comprehensive review, we aim to highlight the evolving landscape of CoA management and the benefits of modern interventional approaches.

Keywords: Coarctation of aorta, congenital heart disease, left ventricular outflow tract obstruction, stent implantation for coarctation.

1. INTRODUCTION

Coarctation of the aorta (CoA) was first described in 1760 by the anatomist Morgagni Thiene[1,2]. It was among the earliest congenital heart defects (CHD) to be surgically corrected, with the Swedish surgeon Crafoord performing the first end-to-end anastomosis in 1944[3].

CoA refers to the narrowing of aortic lumen, with severity ranging from mild posterior shelf wall thickening or infolding to complete aortic luminal atresia. This differs from interrupted aortic arch, which involves a true discontinuity of the aortic wall[4]. Classical CoA involves narrowing of the aortic isthmus between the left subclavian artery and ligamentum arteriosum, often referred to as juxtaductal, with a posterior discrete narrowing of the aorta. However, atypical CoA may occur at any other site from the transverse arch (pre-ductal) to the descending abdominal aorta proximal to its

bifurcation (post-ductal). The extreme end of anatomical severity of CoA includes a long segment of tubular hypoplasia, transverse arch hypoplasia and aortic atresia.

Despite CoA being one of the most common congenital cardiac diseases (CHD), accounting for approximately 5-8% of all CHD or 4 per 10,000 live births, with a male preponderance [5], it is not uncommon for it to present in adulthood due to variations in the defect's spectrum. CoA may occur as an isolated lesion or be associated with bicuspid aortic valve (BAV) in 45-75% of cases[6,7]. It is also seen in Shone's complex (which includes subaortic membrane, parachute mitral valve and supramitral ring), as well as non-cardiac association like cerebral artery aneurysm and various syndromes such as Turner Syndrome, DiGeorge syndrome, Alagille syndrome, William-Beuren syndrome and Noonan syndrome[8].

Early diagnosis is essential to prevent irreversible cardiac remodelling and complications that could hinder management. With significant advancements in multi-modality imaging, it is now possible to diagnose and assess coarctation severity at an earlier age, allowing for well-planned interventions that minimize post interventional complications. Traditionally, surgical repair has been the primary corrective approach. However, advancements in interventional techniques and stent technology have enabled non-surgical management of CoA, especially in adult population who had a lower survivorship after surgical repair of CoA[9].

Regardless of the quality of the repair, or the age at diagnosis, re-coarctation remains a significant concern, leading to premature cardiovascular disease and necessitating long-term follow-up surveillance.

2. HISTOPATHOLOGY AND PATHOPHYSIOLOGY

2.1 Histology and pathogenesis

Examination of CoA specimens often reveal an abnormal histology of arterial wall, with intimal and medial wall thickening protruding into the aortic lumen. Medial wall degeneration in pre- or post- stenotic segments may increase the risk of development of aortic aneurysm or dissection in the coarctation segment. Most studies propose the ductal tissue hypothesis theory, suggesting that migration of ductal tissue into the CoA segment will lead to narrowing in that area[10]. However, this ductal tissue theory does not explain CoA occurring at a distance from ductus arteriosus such as abdominal CoA. A recent histological examination of specimens from eight individuals with abdominal CoA showed intimal fibroplasia in all patients. The author attributed this to abnormal aortic embryogenesis occurring during the third and fourth week of gestation, which is responsible for the development of abdominal aortic narrowing[11].

It has also been suggested that abnormal genetic factors such as NOTCH 1, MCTP 2, FOX 1 or abnormal vascular endothelial growth factors, may influence the development of aorta or aortic arch, leading to decrease in vascular smooth muscle content and increase collagen in the aorta above and below the CoA[12,13]. In older patients, aortic intimal proliferation contributes to the narrowing of CoA. Histopathological evaluations of recurrent CoA after balloon angioplasty showed neointimal proliferation with aortic intimal fibrosis at the ductal insertion region. Some studies suggest the hemodynamic theory, where reduced anterograde intrauterine blood flow to the fetal arch leads to

underdevelopment of the aorta[7,14].

Regardless of the etiology behind the development of CoA or hypoplasia, which is not completely understood, CoA can be considered as a diffuse intrinsic aortopathy, where endothelial dysfunction and abnormal elastic properties might progress even after repair[15].

2.2 Pathophysiology and mechanism of hypertension in CoA

Aortic narrowing can lead to significant hemodynamic consequences, primarily through increased left ventricle (LV) systolic afterload. This results in LV hypertrophy, and LV diastolic and systolic dysfunction. Additionally, there are also pre- or post-stenotic vessel dilatation, and development of collateral vessels to maintain distal perfusion[16].

The mechanism of hypertension in CoA is unique and multifactorial. It is not only due to the anatomical narrowing, but also the lower body hypoperfusion, which triggers the renin-angiotensin-aldosterone system as a compensatory mechanism, leading to worsening hypertension. Additionally, the abnormal intrinsic properties of the arterial wall, combined with long-standing aortic narrowing, caused increased arterial stiffness and decreased arterial compliance especially in elderly[7]. Prolonged LV afterload burden results in impaired relaxation and compliance of the LV, leading to a decrease in the diastolic perfusion pressure required for coronary flow and myocardial perfusion[17].

3. CLINICAL PRESENTATION AND EVALUATION

3.1 Clinical presentation

The clinical presentation of CoA varies based on the severity spectrum and associated lesions. In older children and adults, CoA typically manifests as upper body hypertension or sign of lower body hypoperfusion, such as delayed pulse or low pressure in lower extremities. Long-standing upper body hypertension leads to autoregulatory vasoconstriction, resulting in symptoms like chronic headache and epistaxis. Lower body hypoperfusion with autoregulatory vasodilation presents as lower extremities fatigue, claudication, abdominal angina and cold extremities. A continuous murmur along the left sternal border radiating to the interscapular area is often found in classical CoA with collateral vessels formation[18,19].

Measurement of blood pressure (BP) in all four limbs is crucial, as differences greater than 20mmHg should raise suspicion of CoA. However, variabilities might occur with a right or left aberrant subclavian artery that arises distal to the CoA, leading to unequal BP in each upper limb. Challenges increase if both aberrant subclavian arteries arise distal to CoA, causing a decreased pressure in all extremities. A 24-hours ambulatory blood pressure measurement (at the right arm) is now recommended to detect mean arterial pressure [20].

In adults, CoA can present either as native or in previously repaired CoA (post surgery or post catheter therapy). Adult patients may exhibit signs of LV failure, such as reduced effort tolerance, shortness of breath, or pulmonary edema. If left untreated, CoA can lead to more catastrophic sequalae like premature coronary artery disease, aortic aneurysm or dissection, endocarditis, and cerebrovascular accident[21].

3.2 Evaluation and diagnosis

Electrocardiography (ECG) may reveal signs of LV hypertrophy. The chest radiograph may display a ‘figure of 3’ pattern, indicating areas of CoA indentation with post-stenotic dilatation. Additionally, rib

notching suggestive of collateral vessels formation, may be observed, guiding further diagnostic workup.

Transthoracic echocardiogram (TTE) is the primary modality for diagnosing and monitoring CoA in both children and adults. TTE is effective in diagnosing suspected CoA, assessing the severity, evaluating ventricular function, and identifying associated cardiac lesions such as ventricular septal defect (VSD) or Shone's complex. The severity of CoA can be estimated by using pulse and continuous-wave Doppler by calculating the pressure gradient across the CoA, and the presence of diastolic run-off. However, the presence of collateral vessels may sometimes cause an underestimation of the gradient across the CoA. Limitations of TTE include its operator dependency and variation in acoustic windows among adult patients, which can preclude its use. Additionally, it has limited visualization of extracardiac structures and estimation of the severity of collaterals[10].

Cardiac computed tomography (CT) provides valuable information and allows for detailed assessment of cardiac, extracardiac structure, aortic morphology, and collateral vessels formation. Advances in CT technology, with its highest spatial resolution of non-invasive imaging modalities, along with 3D reconstruction softwares and kernels, offer attractive options for careful pre-planning of interventional or surgical procedures. It also reduces artefact effect in post stent CoA patients. CT can detect CoA related complications such as aneurysm, restenosis or residual stenosis. Recent CT acquisition techniques have significantly reduced the radiation dose associated with cardiac CT scans compared to the last 20 years.

Cardiac magnetic resonance imaging (CMR) provides non-invasive tissue characterization through late gadolinium (LGE) imaging. It not only offers anatomical information, but also advanced hemodynamic assessment with 4D flow CMR. CMR allows for the measurement of left ventricular volumes and function, as well as residual gradient or re-coarctation. Thus, CMR can play a crucial role in contemporary diagnosis and management of CoA, without the use of ionising radiation or contrast exposure[10].

3.3 Cardiac Catheterization

Cardiac catheterization and angiography are the gold standards for hemodynamic assessment of the CoA gradient in native or re-coarctation cases. They not only allow visualization of the aorta but also provide an assessment of complications such as severity of collaterals and aneurysm formation. With the emergence of advanced non-invasive diagnostic imaging tools, comprehensive planning for surgical or interventional procedures, (such as determining the type of future stent needed) can be achieved. Cardiac angiography complements this information, allowing for the anticipation of complications during stenting or balloon angioplasty. Invasive gradient measurement with a peak-to-peak gradient > 20mmHg indicates hemodynamically significant CoA[14].

4. MANAGEMENT APPROACH

4.1 Indications of CoA intervention

The primary goal of management in CoA is to alleviate the obstruction while optimizing long-term outcome. Indications of intervention following the recommended guidelines by European Society of Cardiology (ESC) in 2020 and the American heart association/American College of Cardiology (AHA/ACC) in 2018: Repair of coarctation or re-coarctation (whether surgically or transcatheter) is indicated in

hypertensive patients with a significant non-invasive pressure gradient between the upper and lower limbs (peak to peak > 20mmHg), with preference for catheter treatment (stenting) when technically feasible. (Class I, level C). Table 1[20,22].

Table 1: Indications of intervention in coarctation of aorta (20), Adapted from European Society of Cardiology ACHD guideline, 2020 and American Heart Association College of Cardiology ACHD guideline, 2018

Indications for treatment	Class	Level
Surgical or catheter-based treatment; with preference for catheter treatment (stenting) indicated in hypertensive patients with increased non-invasive gradient between the upper and lower limbs (peak to peak > 20mmHg)	I	C
Catheter treatment (stenting) should be considered in hypertensive patients with ≥ **50% narrowing relative to aortic diameter at the diaphragm, even the peak-to-peak gradient < 20mmHg**	II a	C
Catheter treatment (stenting) should be considered in normotensive patients with an increased non-invasive gradient confirmed with invasive measurement (peak to peak > 20mmHg)	IIa	C
Catheter treatment (stenting) should be considered in normotensive patients with ≥ **50% narrowing relative to aortic diameter at the diaphragm, even the peak-to-peak gradient < 20mmHg**	IIb	C

Most guidelines strongly recommend intervention in CoA in the presence of significant peak to peak gradient (more than 20mmHg) across the CoA, especially in hypertensive patients. However, in conditions, such as left ventricular dysfunction, severe aortic insufficiency or presence of significant collateral vessels, the gradient may not be significant. In these cases, guidelines suggest considering intervention if the narrowing is ≥ 50% relative to the aortic diameter at the level of the diaphragm.

In smaller or young infants with significant CoA, surgical treatment is typically preferred for its durability, especially when other cardiac procedures need to be addressed simultaneously. In older children and adults, endovascular therapy such as stent implantation is often favoured over surgery when technically feasible, as it allows for a faster recovery and an earlier return to work. In some adult patients with comorbidities or extensive collateral vessels formation, surgical repair may be associated with higher risk of perioperative morbidity and mortality[23].

4.2 Surgical repair in CoA

Surgical repair for CoA has been practiced for over 70 years, with well-documented long-term outcomes. The first reported surgery in 1945 involved resecting the diseased segment with end-to-end anastomosis. This technique has since evolved to subclavian artery flap repair, interposition tube jump grafts, prosthetic patch aortoplasty, and to the current extended end-to-end anastomosis. These newer surgical techniques aim to achieve favourable scarring and minimise re-coarctation (re-CoA) during the vessel's remodelling process. Surgical reports have shown variable degrees of re-CoA or aneurysm formation, particularly with older patch material like Dacron, which have been replaced by polytetrafluorethylene (PTFE) for the better outcome. For CoA cases involving aortic arch

hypoplasia, arch augmentation and extended repair should be considered during surgical repair[24,25].

Surgical intervention in adults is typically considered in: complex CoA, when associated with other significant lesions requiring intervention, such as aortic valve (AV) disease necessitating AV replacement; presence of significant aortopathy such as aortic aneurysm measuring over 50mm or rapid progression in the aortic diameter[26,27].

4.3 Percutaneous interventional repair in CoA

Percutaneous intervention particularly aortic stenting is currently the favoured approach of treating CoA in adults. It is less invasive and offers a good post interventional recovery. It is beneficial for treating re-coarctation or aneurysm formation following balloon angioplasty or surgical repair. However, careful consideration is required for patients with concomitant transverse arch hypoplasia or aortic tortuosity. The decision to perform stenting should be based on center's expertise, stent availability and multidisciplinary discussion of the case.

4.3.1 Balloon angioplasty

Balloon angioplasty (BA) can be effective in short term and provide immediate relief of CoA at any age, as in high-risk neonates as a bridge to surgery. BA creates a 'controlled tear' of the intima and part of the medial layer of the vessel wall at the stenotic segment. However, initial series reported higher rates of re-coarctation (15-30%) and aneurysm formation (up to 20%), as well as dissection due to weakening of the dilated vessel wall[28]. In post-surgical re-coarctation, BA can alleviate the obstruction with lower rate of aneurysm formation. BA had a comparable immediate post-treatment gradient with stented group, but the high risk of reintervention or vascular re-coil makes it unsuitable for adult patients, especially in cases of hypoplastic aortic isthmus or tubular CoA [29]. Thus, BA for adults with native CoA or recurrent CoA may be considered if stent placement is not feasible and surgical intervention is not an option[30].

4.3.2 Stenting

Compared to BA, stenting avoids overdistension of the aortic wall and strengthens the vessel walls, reducing the rates of aneurysm formation, aortic wall injury, or dissection. The application of stent implantation in aortic CoA is increasing with growing experience and advancements in stent technology, leading to expansion of its utilization to include more complex CoA anatomy such as atretic CoA (acquired atresia), aortic arch tortuosity, treatment of CoA aneurysms and stent related complications such as fracture, endoleak or aortic wall injury[31,32].

4.3.3 Stent Technology

The use of intraluminal stainless-steel stents was first developed and tested in dogs by Palmaz et al. This was followed by studies in swine models. The first clinical applications in an infant and later in a small adult series were in 1990s. Over time, stent technology has evolved to achieve an improved stent characteristic, including better flexibility, lower profile, minimal foreshortening, maximal expansion diameter, high radial strength, non-sharp edges, and most importantly, the ability to further dilate the stent to an adult size with somatic growth. The open cell strut configurations are particularly useful when overlapping stents at brachiocephalic vessel is required[33].

Choosing the appropriate stent is essential in managing CoA. Various stent types from several manufacturers have been employed for CoA treatment, though most are used off-label. Example of the only United States approved stent is the Cheatham Platinum

(CP) stent (NuMED Inc., Hopkinton, NY, USA). Other stents used off-label include: Palmaz series (Johnson & Johnson Interventional Systems Co., Warren, NJ, USA), Genesis XD (Cordis Corp., Miami, FL, USA), Atrium Advanta V12 (Maquet, Rastatt, Germany), IntraStent (Medtronic, Minneapolis, MN, USA), Formula (Cook Medical, Bloomington, IN, USA), AndraStent (Andramed GmbH, Reutlingen, Germany), Valeo stent (BD, NJ, USA) and the Optimus CoCr (Andra Tec, Germany).

4.3.4 Covered stent (CS) vs bare metal stent (BMS)

The bare metal stent (BMS) provides endovascular prosthesis, maintaining less vascular recoil compared to BA alone. The covered stent (CS), equipped with PTFE sleeve, optimizes endoleak and excludes aneurysm formation, and addresses BMS complication such as fractures or acute wall injury. The use of CS in the treatment of CoA was first described in 1999. The Cheatham-Platinum (CP) covered stents (NuMED Inc., Hopkinton, New York) were introduced in 2001. These CP stents are balloon-expandable and needed to be mounted on the appropriate balloon diameter. Newer CP stent with gold soldering, pre-mounted bare and covered stents are now available. The Bentley BeGraft Aortic stent (BeGraft Aortic, Bentley InnoMed, Hechingen, Germany) is available in Europe, parts of Asia, Latin America and Africa. The PTFE tube of the BeGraft Aortic stent is folded inside at both ends of the stent and clamped between the first and the last stent struts to ensure stability during implantation. These stents are premounted, requiring sheaths up to 14Fr for 18-24mm diameter[34,35]. The Optimus-CVS (AndraTec, Koblenz, Germany) is a cobalt–chromium stent with hybrid design and PTFE-covering. It offers a secure sandwich covering with thermally bonded inner and outer Nano-PTFE layers and a unique end-free technology, with half a row of cells being bare-metal at both extremities. It has low profile delivery system and can be deployed through sheathes that are 1-Fr to 3-Fr sizes smaller than those required for similar stents[36].

The use of CS can be particularly beneficial in preventing acute wall injury in a very tight or atretic CoA or complex tortuous CoA, which carry a high risk of aneurysm or dissection. The COAST II (Covered Cheatham-Platinum™ Stents for Prevention or Treatment of Aortic Wall Injury Associated with isolated CoA Trial) study showed that covered stents are preferred over bare metal stents for treatment of high-risk CoA[37]. Despite the increasing commercial availability of CS treatment for CoA, especially in adults, long-term longitudinal studies are needed to determine if it is superior to bare metal stent (BMS) and to evaluate its long-term benefits[38].

In rare cases of adults with CoA and patent ductus arteriosus, covered stents can be used to close the ductal shunt. In COAST II trial, which evaluated the use of covered CP stent in 158 patients, reported a success rate of 92% with no incidence of aortic wall injury or death[37]. An endovascular covered stent with ready built side-branch of subclavian artery (example of thoracic branch endoprosthesis), might be a future solution for treatment of recurrent CoA with closed proximity to subclavian artery branch[13]. The small risk of spinal cord blood supply is a consideration in covered stents in general when deployed in T9-T12 level.

Premounted covered stents can be extremely valuable in significant complications such as aortic rupture, allowing for rapid deployment. A recent

ready-to-use preloaded all-in-one stent delivery system was introduced, the NuDEL (NuMED Inc) which includes a premounted covered CP stent on a BIB balloon, preloaded into a kink-resistance long sheath that allows for quick actions in emergency situations.

Use of covered stents in patients with coarctation close to vital branches should be carefully exercised with meticulous attention to such side branches.

4.4 Procedure steps of endovascular stenting [14]

The procedure is generally performed under general anaesthesia, even in adults, to manage discomfort or pain during balloon inflation or stent deployment. In some centers, conscious sedation is used for adults, with deep sedation administered at the time of stent implantation under the supervision of cardiac anaesthesiologist[39]. Retrograde femoral arterial access is obtained, unless anticipating difficulty crossing severely narrow area or atretic CoA segment from descending aorta, in which case left radial or left brachial artery access is used to access from above the area of coarctation[40]. Arterial haemostasis suture device (e.g. Perclose, Abbott Vascular) can be inserted after femoral arterial access is obtained for securing haemostasis once the procedure is completed, especially when a larger sheath is expected. Femoral venous access is required for hemodynamic studies or sometimes used for rapid right ventricular pacing to ensure more controlled deployment and less movement of the stent, especially if coarctation is mild and stent positioning is crucial.

Intravenous heparin is administered to achieve activated clotting time (ACT) greater than 200 seconds. A straight-tip catheter (multipurpose) is preferred, especially when crossing a tortuous CoA or tight stenosis. Another option is advancing a Judkins right catheter with a soft tipped wire (eg; hydrophilic wire) into the ascending aorta. Following crossing of the CoA by the catheter, the wire is exchanged with an extra stiff 0.035 inch guidewire (e.g Amplatz extra stiff wire). Pressure measurement can then be done using either a Multitrack (NuMed) monorail catheter over the wire or an end-hole catheter to obtain an accurate pressure gradient.

Pigtail catheters are used for angiography. Biplane angiography is performed in shallow left anterior oblique (LAO) and straight lateral (90 degrees LAO). The diameters of transverse arch, aortic isthmus, the site of CoA, the descending aorta at the diaphragm are measured after appropriate calibration. The maximum diameter of the balloon for stent placement is based on the diameter of the isthmus (just above coarctation), ensuring it does not exceed the diameter of the aorta at the diaphragm level. Pre or post stenotic dilatation should not be used as a reference when determining balloon size. A long sheath, (e.g Mullins, Cook Medical, 75cm length) is passed over the extra stiff guidewire and positioned across the CoA segment. The wire is positioned either in the ascending aorta or preferably in the right subclavian artery

The stent is hand-crimped onto a balloon catheter, with umbilical tape used for crimping if available. Commonly used balloon catheters are the BiB Balloon from 14 to 20mm of outer diameter balloon requiring sheaths from 8 to 14Fr sheath. The shorter inner balloon ensures uniform stent expansion without flaring the ends, thus reducing the risk of balloon perforation or wall injury. It also provides a better anchoring system, resulting in more controlled outer balloon inflation and potential space to adjust the stent position after inner balloon expansion. The steps of CoA procedure are

demonstrated in Figure 1.

After stent deployment, the balloon is deflated fully and carefully withdrawn to avoid stent dislodgment. The gradient across the stent is remeasured, and a final aortogram is repeated to assess the result and to exclude dissection or aneurysm formation. A successful procedure is considered when the peak-to-peak gradient (PPG) is less than 10mmHg and there is an improvement in vessel caliber of more than 80% of reference aorta just above the coarctation. In some patients, careful additional post-dilation of stent can be considered, but caution against full expansion at the site of CoA, especially in tight stenosis. Stent apposition to the aortic wall can be achieved if needed using a medium or high-pressure balloons (e.g TYSHAK or Z-MED^{TM} II balloon; NuMED). In adults with tight stenosis, diameter of 12-14mm is considered satisfactory with option of staged balloon dilations in the future to decrease the risk of aortic wall injury. Usually, the balloon compliance testing prior to stent placement is not required, and may even pose a higher risk of aortic injury compared to primary stent techniques[39].

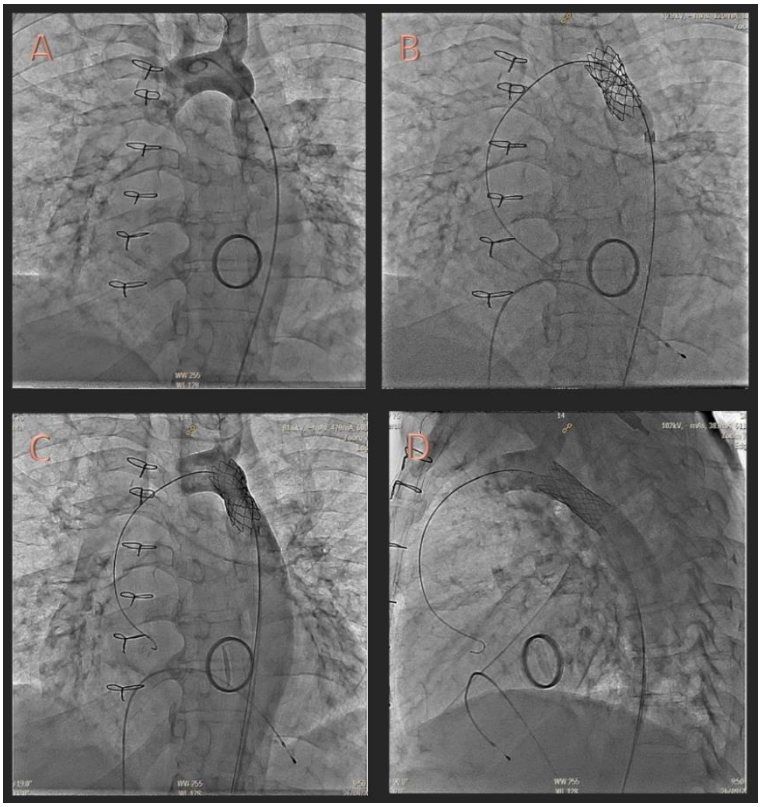

Figure 1: A 48-year-old gentleman with underlying hypertension and diabetes, who had previously undergone mechanical prosthetic mitral valve replacement, and had concomitant bicuspid aortic valve. CT cardiac screening revealed discrete native CoA. A frontal aortogram (A) with pigtail catheter showed discrete CoA at the aortic isthmus with calcifications, with an invasive peak-to-peak gradient of 24mmHg. He underwent stent implantation with 8-zig covered CP mounted on 16mm BiB balloon (B) with rapid ventricular pacing. Post-stenting angiogram (C and D) showed improved area of CoA and hemodynamic measurement showed no residual gradient.

4.5 Complications of stenting

In general, stent therapy is considered safe and preferred option for adults with CoA, especially in discrete native CoA. Stenting has a high procedural success rate over 90% and can reduce the hemodynamic gradient at CoA more effectively than BA alone. Acute adverse events are uncommon, occurring in 2-2.5% of patients aged 1 to 18 years and 6.6% in adults. Complications such as bleeding (3.4%), acute aortic wall injury (0.6-2.4%) and femoral artery injury or

thrombosis can occur. Acute aortic rupture or dissection can be managed with the rapid deployment of a covered stent[39,41].

In 5-year follow-up of the Coarctation of Aorta Stent Trial (COAST) and Covered Cheatham-Platinum Stents for Prevention or Treatment of Aortic Wall Injury Associated with Coarctation of the Aorta (COAST II) trials, the cumulative incidence of aortic aneurysms was 6.3%, and stent fractures were seen in up to 25% of patients (without clinical significance except in 1.3% of patients) [42].

The weight and age at which a better candidate for stent placement has not yet been established. The use of an adult-size stents in small patients less than 20 kg is controversial, due to the larger sheath sizes needed to implant a stent that is expandable to adult size diameter in the future (at least 18mm), which can be challenging and need to outweigh the risk of vascular injury and reintervention for the somatic growth. Studies on transcatheter treatment with adult-size stent in small patients are very few and limited, primarily conducted in re-CoA[43,44]. A study involving 39 patients weighing less than 20 kg, implanted adult-size stents for both native and recurrent CoA, with a 97% success rate, demonstrating both safety and efficacy[45]. Additionally, data from Improving Pediatric and Adult Congenital Treatment (IMPACT) registry, which is focusing on native CoA stenting, the patients weighing 10 to 20 kg, showed low incidence of major adverse events, potentially representing a shift in practice toward increased use of stenting in smaller patients in the future by development of lower profile stents[38].

5. LONG TERM OUTCOME

Patients with unrepaired CoA have mean survival age of 35 years with aortic rupture in 21% of cases[22]. However, repaired CoA has shown a 20-year survival rate of 84%[46]. Repaired CoA, whether by surgical or percutaneous intervention (especially stenting) has demonstrated significant short-term outcome in immediate alleviation of obstruction with improved hemodynamic results[47,48]. Surgical repair of CoA in adult population showed cumulative survival of 82% after 30 years[49]. An observational study by Congenital Cardiovascular Interventional Study Consortium (CCISC) in 2011, showed stenting had lower acute complications compared to surgery or balloon angioplasty[33]. Following that, a meta-analysis review in 2023 indicated that stenting might have superior efficacy not only in re-CoA, but in native CoA with higher success rate compared to balloon angioplasty[50]. A long-term follow-up study after stenting of CoA showed a survival rate of 98.1% after 5 years, and 95.6% after 10 and 15 years. Causes of late death in this cohort included myocardial infarction, progressive coronary artery disease, or congestive heart failure[31,51].

5.1 Re-coarctation and reintervention

Re-CoA can occur due to intimal proliferations following any type of repair. The re-CoA rates have been reported to be between 30-59% [52], while the rates of re-intervention vary across studies due to differences in thresholds of re-intervention. A recent study showed re-intervention needed rate was about 11% in older population[53]. In contrast, 25% of patients in extended COAST study required reintervention, majority at late follow up. Comparable results were also reported in other studies (27.8% reintervention rate) of stented patients[31,37].

There is no expert consensus on when the re-intervention should be performed especially in post stent CoA. As to differentiate between staged stent expansions, re-interventions for somatic growth, re-interventions for restenosis, and re-intervention for other indications is

challenging[37]. However, most studies suggest similar criteria for re-intervention as for the initial intervention. An increase in the non-invasive systolic blood pressure gradient between the upper and lower limbs > 20mmHg should prompt further investigation with imaging or invasive assessment, and followed by catheter intervention in patients with a coarctation to diaphragm diameters ratio of ≤0.5. One study demonstrated a positive correlation between exercise capacity and CoA isthmus diameter, with those having a ratio of ≤ 0.7 exhibiting significantly reduced functional capacity. Cardiopulmonary exercise testing may be helpful in identifying asymptomatic or non-severe CoA cases[54].

While catheter intervention is often preferred over surgery in recurrent CoA, surgical repair as re-intervention is usually considered in patients with persistent endoleak or prosthetic infection. The relative higher mortality of surgical re-intervention compared to native CoA repair, ranging from 1-3% to 5-10% [6], is related to adhesions, bleeding, and trauma to surrounding structure, which may lead to spinal cord trauma or thoracic duct injury. Thus, the treatment strategy in re-intervention is moving toward percutaneous treatment especially post-surgical CoA. Figure 2 demonstrates an example of stenting intervention in a patient with re-CoA.

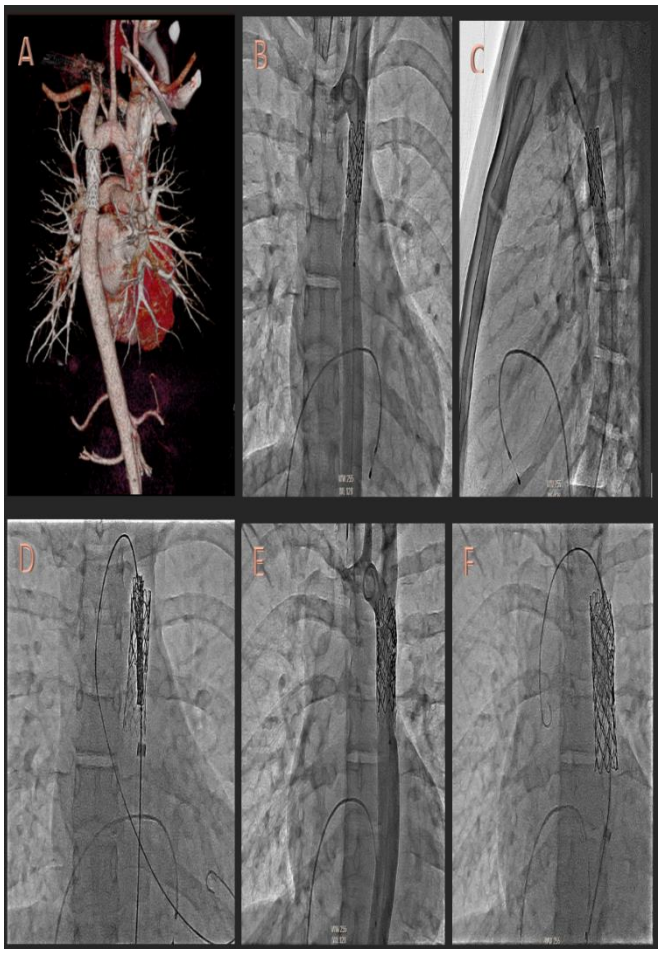

Figure 2: A 17-year-old athletic girl with a history of discrete CoA, who underwent CoA Stenting at the age of 10 with a 28mm long bare metal CP stent on a 10mm balloon, and a second stent placement two years later with 30mm long AndraStent over 12mm balloon, post-dilated to 15mm. After six years of follow-up, she was noted to have a non-invasive blood pressure discrepancy of more than 20mmHg, with an Echo gradient of 26mmHg. CT scan revealed re-CoA within the stent (A). Catheter angiography showed reCoA with minimum diameter of 9mm (B and C), with descending aorta of 16mm at the level of diaphragm. The invasive peak-to-peak gradient was 25mmHg. She underwent two covered CP stent implantation, (C, D) one 45mm mounted on a 16mm BiB balloon and another 34mm mounted on 16mm BiB balloon (F) with rapid ventricular pacing and required a long sheath of 12 Fr. Both stents were Post-dilated with Atlas Balloon (16mm). Post-stenting showed peak-to-peak gradient of 2mmHg.

5.2 Aortic Aneurysm formation

Aortic aneurysm is defined as secondary expansion of aortic diameter beyond the stent or native aortic diameter, which was not present before or immediately after the intervention as proposed by the Congenital Cardiovascular Interventional Study Consortium[33,55]. Pedra et all classified aneurysm as 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[56].

Severe aortic aneurysm may pose a risk of aortic dissection, rupture and death in 7% after CoA repair. True aneurysm can be discrete at the level of previous intervention which contained all 3 layers of aortic wall. While the false aneurysm or pseudoaneurysm is outpouching of defect that contained part of adventitia layer of vessel wall. False aneurysm can occur along the aorta and can present as a late complication. They can occur with both bare metal and covered stents[55]. Stenting may prevent the 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. However, there remains risk of injury at the ends of the stent in both BMS and covered stents or in case with unopposed two stent implantation[57]. (Figure 3 demonstrates an aneurysm formation in previously stented individual).

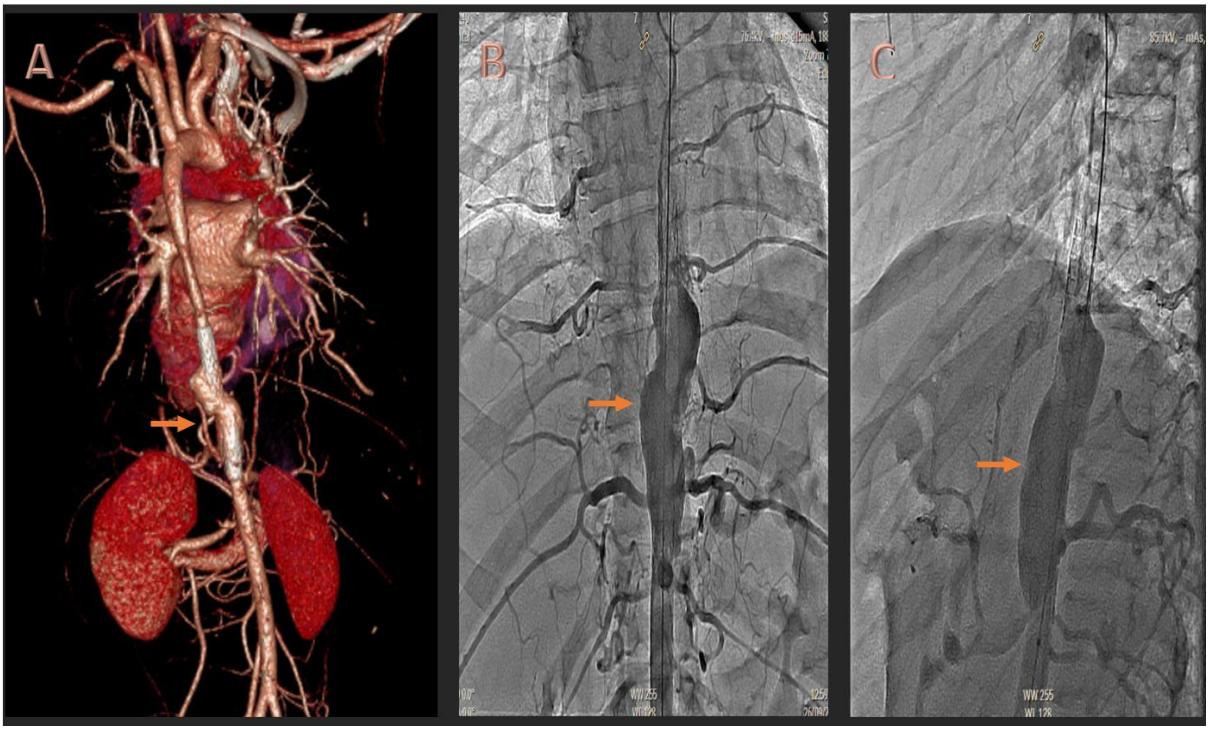

Figure 3: A 19-year-old man with oculo-neuro-ectodermal syndrome and previous history of thoraco-abdominal CoA, underwent placement of two thoracic-abdominal stents at age 13 (Andrastent XL 39mm dilated to 10mm) for significant abdominal CoA. CT surveillance showed non-progressive aortopathy changes with two saccular aneurysm formations (arrows) at the previously placed stents (A) measuring 21mm and 19mm in diameter, respectively. He is otherwise asymptomatic. A cardiac angiogram revealed re-CoA with thoracic narrowing measuring of 7mm and a transverse arch diameter of 14mm with multiple collateral vessels (B and C)

The previous surgical technique using patch repair using "Dacron" carries a risk of repaired-site aneurysm, which is no longer used in the current era. Other risk factors for developing aortic aneurysm include the use of balloon angioplasty due to unpredictable therapeutic tear of narrow segment of CoA, and co-existing aortopathy disease such as bicuspid aortic valve or Marfan syndrome[58]. Aneurysm can be diagnosed by CT or CMR or during cardiac angiography during catheterization[59].

The decision and timing of intervention in asymptomatic patients with an aortic aneurysm after CoA repair is difficult. Surgical management of progressive aneurysm or pseudoaneurysm carries significant mortality and morbidity.

Alternatively, endovascular stent grafts have been used to repair aortic aneurysms at the site of prior CoA repair. In one case series of six adult patients (age 31 to 68 years) with aortic aneurysm following CoA repair, placement of a stent graft was successful in all cases with no related morbidity or mortality at one-year follow-up after intervention[60,61].

6. FOLLOW UP

Despite good long-term survival, patients with repaired CoA require lifelong follow-up ideally with Adult Congenital Heart Disease (ACHD) cardiologists as the morbidity rate after intervention is still high[20,22]. Recommended post-repair monitoring includes annual measurement of upper and lower limb blood pressure and echocardiography. Cross sectional imaging preferably CMR is recommended at least 3 to 5 years post intervention to detect anatomical complication such as re-CoA, or aortic aneurysm[62].

6.1 Hypertension

With successful repair of CoA, hypertension remains a common long-term complication even after a complete relief of obstruction. Patients with isolated CoA repaired early in life may have lower rate of developing hypertension[14,30]. The increased hypertension burden is associated with poor survival in adult post-CoA repair, as it becomes a risk factor for developing premature coronary artery disease, ventricular dysfunction as well as aortic or cerebral aneurysm. Despite the elimination of the pressure gradient across the CoA, systemic hypertension persists in 25-40% of repaired CoA patients, necessitating long-term pharmacological treatment[63,64].

The mechanism of hypertension can be due to functional or structural causes. In patients with hypoplastic arch or a smaller stented size compared to somatic growth of aorta, right arm 24-hour ambulatory blood pressure monitoring is a better detection of hypertension, as masked hypertension may occur. Treating an isolated exercise-induce hypertension (Systolic blood pressure >190mmHg in adult women and > 210 mmHg in adult men) remains a matter of debate [17]. Medical management of arterial hypertension should follow standard guidelines of 2018 European Society of Hypertension.

Routine attention should be given to patients with conditions such as bicuspid aortic valve, premature CAD, or intracranial berry aneurysm[58]. Modifiable risk factors such as hyperlipidemia, should be aggressively managed to prevent premature atherosclerotic plaque formation in the diseased aorta, which could complicate matters further. Encouraging a healthy lifestyle, including appropriate physical activity, avoiding a sedentary lifestyle or obesity, and non-smoking, is essential for all patients with repaired CoA.

7. MANAGEMENT IN SPECIAL GROUP

7.1 CoA and pregnancy

Many women who had undergone successful CoA repair can tolerate pregnancy without major issues, even though they are considered moderate risk (WHO class II-III) for maternal morbidity and mortality. Historically, it was believed that women with repaired CoA faced a significant risk of preeclampsia; however recent studies suggest that the risk of serious complications may be lower than previously thought[7]. Nonetheless, careful blood pressure monitoring throughout pregnancy is recommended to promptly initiate therapy and avoid complications such as pre-eclampsia or aortic dissection. A multidisciplinary team management is ideal during follow up of pregnant patients with repaired CoA. Fetal echocardiography should be offered during their pregnancy[65].

Women with unrepaired CoA are considered high risk (WHO class IV) and should be advised against pregnancy until the CoA is repaired. Those with significant residual CoA, should undergo intervention prior to pregnancy. Preconception counselling should be offered to all women with congenital heart disease in childbearing age for risk stratification and to discuss the heritability risk to the offspring. Cardiopulmonary exercise testing can be considered during pre-conception counselling to detect exercise induced hypertension.

7.2 Sport participation

Patients without residual obstruction, and who are normotensive at rest or during exercise can lead normally active lives without restrictions. However, patients with arterial hypertension, residual obstruction, or other complications should avoid heavy isometric exercise according to severity of their complications. Isometric exercise is discouraged in cases of aortic dilation, especially in the presence of resting or exercise induced HTN as risk of dissection can be high[14,30].

8. CONCLUSION

Managing coarctation of the aorta (CoA) in adults, whether it is native or recurrent, remains a significant challenge requiring long-term, holistic management and includes collaboration with adult congenital heart disease program to improve the morbidity and survival outcome. Careful preplanning for intervention is crucial to stratify the risk of complication. Endovascular treatment has evolved, and stent therapy has become a widely acceptable alternative to surgery, especially for adolescents and adults. Increased operator experience and advancements in stent technology have extended applications in CoA management with improved safety and success rate. Successful treatment of adult patients with CoA results in improved quality of life, and allows them to engage in normal activities, including childbirth, and sports.

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الإدارة الشاملة لضيق الشريان الأورطي؛ الأساليب الحديثة والاستراتيجيات المحدثة للمراهقين والبالغين

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الملخص

في المراهقين والبالغين الذين يعانون من تضيق الشريان الأورطي (CoA)، سواء كان يظهر على شكل CoA أصلي أو CoA متكرر، ظهر تدخل CoA عبر القسطرة كبديل مقبول على نطاق واسع للجراحة. أدت التطورات السريعة في التقنيات وصقل تكنولوجيا الدعامات إلى إحداث نقلة نوعية كبيرة في الممارسة السريرية. تقدم هذه المخطوطة مراجعة مفصلة لاستراتيجيات الإدارة الحالية ل CoA، مع التركيز بشكل خاص على المراهقين والبالغين. يوفر رؤى محدثة حول المؤشرات الحالية للتدخل، والنصائح والتقنيات الإجرائية، ويعالج النتائج الفورية والطويلة الأجل لإصلاح CoA لدى البالغين. من خلال هذه المراجعة الشاملة، نهدف إلى تسليط الضوء على المشهد المتطور لإدارة CoA وفوائد الأساليب التدخلية الحديثة.

الكلمات الدالة: تضيق الشريان الأورطي، أمراض القلب الخلقية، انسداد مجرى تدفق البطين الأيسر، زرع دعامة للتضيق.