ASDIN white paper: Management of cephalic arch stenosis endorsed by the American Society of Diagnostic and Interventional Nephrology*

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Abstract
Brachiocephalic arteriovenous fistulas (AVF) makeup approximately one third of prevalent dialysis vascular accesses. The most common cause of malfunction with this access is cephalic arch stenosis (CAS). The accepted requirement for treatment of a venous stenosis lesion is \( \geq 50\% \) stenosis associated with hemodynamically abnormalities. However, the correlation between percentage stenosis and a clinically significant decrease in access blood flow (Qa) is low. The critical parameter is the absolute minimal luminal diameter (MLD) of the lesion. This is the parameter that exerts the key restrictive effect on Qa and results in hemodynamic and functional implications for the access. CAS is the result of low wall shear stress (WSS) resulting from the effects of increased blood flow and the unique anatomical configuration of the CAS. Decrease in WSS has a linear relationship to increased blood flow velocity and neointimal hyperplasia exhibits an inverse relationship with WSS. The result is a stenotic lesion. The presence of downstream venous stenosis causes an inflow-outflow mismatch resulting in increased pressure within the access. Qa in this situation may be decreased, increased, or within a normal range. Over time, the increased intraluminal pressure can result in marked aneurysmal changes within the AVF, difficulties with cannulation and the dialysis treatment, and ultimately, increasing risk of access thrombosis. Complete characterization of the lesion both hemodynamically and anatomically should be the first step in developing a strategy for management. This requires both access flow measurement and angiographic imaging. Patients with CAS present a relatively broad spectrum as relates to both of these parameters. These data should be used to determine whether primary treatment of CAS should be directed toward the anatomical lesion (small MLD and low Qa) or the pathophysiology (large MLD and high Qa).

Keywords
Cephalic arch, cephalic arch stenosis, management of cephalic arch stenosis, AV fistula < dialysis access, techniques and procedures

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Introduction

A distal radial-cephalic arteriovenous fistula (RC-AVF) remains the first choice for dialysis vascular access. If this is not possible or fails, a proximal AVF using the cephalic vein (BC-AVF) with brachial artery inflow is often the next access created. The cephalic vein’s anatomy results in simple AVF construction and easy cannulation for dialysis. BC-AVFs make up 25%–34% of prevalent vascular accesses. One of the most common lesions resulting in malfunction of BC-AVFs is cephalic arch stenosis (CAS). CAS has a reported incidence ranging from 15% to 77%. and is reported to account for 50%–70% of stenoses in upper arm AVFs in addition to 15%–18% of cases of AVF failure (all types).

Due to its prevalence, resistance to treatment, and high recurrence rate, CAS is a frequent cause of dialysis vascular access failure. Nevertheless, a standardized approach to management has not been generally accepted. Although cases presenting with evidence of the inflow-outflow imbalance that characterizes CAS represent a broad spectrum anatomically and hemodynamically, the primary approach for the treatment of this lesion has been anatomical, designed only toward addressing the pathological anatomy. The role of excessive Qa in the development and recurrence of the lesion and its adverse cardiovascular effects if left untreated have generally been ignored. The purpose of this paper is to highlight the importance of performing a complete characterization of the CAS lesion both hemodynamically and anatomically in developing a strategy for its management. Based upon these data, an approach to the treatment of CAS should be adopted which encompasses both parameters.

Clinical features of CAS

The presence of downstream venous stenosis causes an inflow-outflow mismatch resulting in increased pressure within the access. Qa in this situation may be decreased, increased, or within normal range depending upon the relationship between the patient’s baseline Qa, the degree of luminal narrowing produced by the lesion, and available collateral outflow. Increased intraluminal pressure is manifest clinically by a progressive dilatation of the access which can eventually become aneurysmal and over time develop into a “mega-fistula.” The AVF becomes hyperpulsatile, has a high venous pressure on dialysis and a tendency for excessive bleeding post-dialysis needle removal. In some cases, progression of the stenotic lesion eventually causes a decrease in Qa resulting in ineffective dialysis and if left untreated eventually culminates in thrombosis of the access.

An upper arm cephalic AVF can be created using inflow from either the brachial artery (BC-AVF) or the proximal radial artery (PRC-AVF). CAS can be seen with either of these, however, it is more frequently associated with the former. Blood flow through the cephalic arch is generally higher with a brachial artery associated AVF than a proximal radial artery based AVF. In addition, almost all of the blood flow from a BC-AVF passes through the cephalic arch. A PRC-AVF usually has three major outflow channels: the median-cephalic vein, the median-cubital vein and, importantly, the perforator vein into brachial veins. The relative resistance in each of these can result in different flow patterns with much or even all of the blood flow drainage from a PRC-AVF missing the cephalic arch. Long-term patency of a BC-AVF is significantly less than that of a RC-AVF. This has been attributed at least in part to the higher incidence of CAS associated with this access. The incidence of CAS is also lower with a PRC-AVF, a difference attributed to a lower blood flow occurring through the proximal radial artery than through the brachial artery.

There is significant variability in the exact location of stenotic lesions that occur in the cephalic arch. The most common site for CAS is the point of confluence of the cephalic arch and axillary vein to form the subclavian vein (Figure 1(a)). However, it may also develop more peripherally (Figure 1(b)), and in some cases the lesion may extend into the subclavian vein (Figure 1(c)).

Pathogenesis

In the upper arm, the cephalic vein is located within the superficial fascia on the anterior lateral surface of the biceps muscle. Just below the clavicle it turns to run deeper as it passes from the deltopectoral groove. The vein then assumes a curved configuration and pierces the clavipectoral fascia to join the axillary vein. The term cephalic arch has been coined to refer to this curved anatomical configuration (Figure 2).

When the cephalic vein becomes the outflow for an arteriovenous (AV) access it becomes susceptible to the development of CAS. This problem has been attributed to the combined effects of three factors—(1) the effect of increased blood flow velocity on this unique anatomical configuration; (2) the presence of venous valves, 96% of people having at least one valve within 3 mm of the cephalic arch confluence with the axillary vein; and (3) restriction to dilatation by the limited opening in the dense clavipectoral fascia and accompanying lateral pectoral nerve and thoracoacromial artery. Although these other putative mechanisms exist, published reports support the concept that the primary culprit ultimately resulting in venous stenosis in this region is result of the unique curved configuration combined with increased blood flow adversely affecting wall shear stress (WSS).

Based primarily upon data derived from arterial studies, it has been shown that WSS plays an important role in regulating the function of endothelial cells. High blood flow velocity in the vein beginning with access creation, results in increased WSS which promotes vessel

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remodeling. Laminar blood flow associated with high WSS promotes normal endothelial function and inhibits the development of neointimal hyperplasia. However, because of the curved configuration of the cephalic arch, low blood flow pockets can develop in association with low WSS. Decrease in WSS has a linear relationship to increased blood flow velocity. Neointimal hyperplasia develops and demonstrates an inverse relationship with WSS. This process eventually leads to the development of a stenotic lesion. It has been suggested that blood flow in the zone immediately downstream from a stenotic lesion is subjected to turbulent blood flow, leading to further development of neointimal hyperplasia as an extension of the original stenosis a process referred to as “propagating intimal hyperplasia.”

Although studies demonstrating the relationship between the hemodynamics of blood flow in the cephalic arch and CAS have dealt with blood flow velocity, blood flow volume is more commonly evaluated in connection with the dialysis vascular access. Since blood flow volume is directly proportional to velocity \( Q = AV \) (\( Q \)—flow volume, \( A \)—cross-sectional area of vessel lumen, \( V \)—velocity) this pathogenic relationship is also relevant to that parameter.

**Figure 1.** Three variations in lesion location (arrows): (a) lesion at the junction with axillary vein, (b) lesion peripheral to junction with axillary vein, and (c) lesion extending into subclavian.

**Figure 2.** Cephalic arch relationships: (a) cephalic arch, (b) thoracoacromial artery, (c) lateral pectoral nerve, and (d) axillary/subclavian vein.

**Prevention of CAS**

Efforts directed toward prevention of CAS are worthwhile and should be incorporated into access planning. Since there is a direct linear relationship between blood flow velocity and the development of CAS, avoiding high blood flow is important to prevention. Because of the size and pressure of the brachial artery, the BC-AVF created using this artery as inflow is associated with a high \( Q_a \) creating a higher risk for CAS. Conversely, use of the proximal radial artery for inflow results in a lower \( Q_a \) and a lower risk for developing the lesion. In a prospective study comparing 56 patients with a BC-AVF and 50 patients with a PRC-AVF, the mean \( Q_a \) was 1060 ± 578 mL/min versus 735 ± 344 mL/min, respectively. No differences in AVF maturation were noted and patency rates showed no significant difference between the two groups. However, the incidence of clinically significant CAS in the BC-AVF group (\( n = 13 \)) was twice that noted in the PRC-AVF group (\( n = 5 \)).

Once it has been treated, CAS is likely to recur and require re-intervention. In a study involving 123 patients with CAS, there was a statistically significant correlation in the need for re-intervention with the severity of stenosis at index visit, access flow, vessel diameter, and prolonged bleeding for >30 min as a reason for referral.

**Indications for treatment**

Practice guidelines state that the general requirement for treatment venous stenosis is that there be \( \geq 50\% \) decrease...
in the luminal diameter of the vessel and must be shown to be hemodynamically significant.\textsuperscript{22,23} Percent stenosis is determined by comparing the lesion to the adjacent “normal” vessel. Identifying an adjacent normal vessel can be challenging. The vein can vary considerably in diameter along its length and segments may be aneurysmal, particularly the vessel adjacent to a stenotic lesion.\textsuperscript{24,25} The major concern in most instances is a decrease in $Q_a$ leading to inadequate dialysis and eventually thrombosis. However, the correlation between percentage stenosis and a clinically significant decrease in $Q_a$ is low. While a 50% decrease in luminal diameter in a 6 mm vein may produce a marked decrease in $Q_a$, a 50% decrease in luminal diameter for a 12 mm vein may be associated with an abnormally high $Q_a$. The critical parameter is the absolute minimal luminal diameter (MLD) of the lesion.\textsuperscript{24,25} This is the parameter that exerts a restrictive effect on $Q_a$ and results in hemodynamic and functional implications for the access. Unfortunately, it is not practical to accurately measure MLD in the routine clinical setting. Nevertheless, recognition of this principle is important, and no lesion should be treated based only upon an anatomic criterion using percentage stenosis. “Critical stenosis” is defined by the functional effect rather than the anatomical appearance. Clinically, this is primarily dependent upon the detection of clinical signs and symptoms (Table 1).

**Table 1.** Criteria for judging clinical-hemodynamic significance.

<table>
<thead>
<tr>
<th>Clinical parameters</th>
<th>Physical examination</th>
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<tbody>
<tr>
<td>Inability to achieve the target dialysis blood flow ($Q_b$)</td>
<td>Hyperpulsatility of fistula</td>
</tr>
<tr>
<td>Prolonged bleeding from needle puncture sites for three consecutive dialysis sessions</td>
<td>Abnormal thrill palpated in ipsilateral infraclavicular fossa and/or anastomosis</td>
</tr>
<tr>
<td>Unexplained (&gt;0.2 units) decrease in the delivered dialysis dose ($K_t/V$) with a constant dialysis prescription and without prolongation of dialysis duration</td>
<td>Abnormal bruit heard in ipsilateral infraclavicular fossa and/or anastomosis</td>
</tr>
<tr>
<td>Elevated venous pressure recorded during hemodialysis for three consecutive dialysis sessions</td>
<td>Failure of fistula collapse when the arm is elevated</td>
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**Table 2.** Management options.

<table>
<thead>
<tr>
<th>Angioplasty plus stent</th>
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<tr>
<td>Flow reduction</td>
</tr>
<tr>
<td>Endovascular</td>
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<tr>
<td>Surgical</td>
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<tr>
<td>Surgical intervention</td>
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<tr>
<td>Central transposition</td>
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<tr>
<td>Bypass</td>
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**Treatment modalities**

Multiple modalities have been used to treat CAS. These include percutaneous balloon angioplasty (PTA), inflow reduction, endovascular stents (bare-metal stents and stent-grafts), and surgical interventions (Table 2). Each of these modalities has been used both as primary and secondary treatment for recurrences.

**Percutaneous angioplasty**

Treatment of CAS with PTA has been judged to be successful although the results obtained have varied considerably\textsuperscript{1,6,26-31} (Table 3). In a meta-analysis involving seven studies (years 2003–2018),\textsuperscript{31} primary patency at 6 and 12 months was 23.3% and 9.5%, respectively. Cumulative patency at 12 months was 67.5%. In more recent years with the advent of the ultrahigh pressure angioplasty balloon, technical success and patency rates have improved.

When compared to treatment of venous stenosis in general, technical success with PTA for CAS has been lower; the lesions have been more resistant to dilatation and venous rupture has been higher than other lesion sites.\textsuperscript{1,4,28,29} Long-term patency has been less. The reported requirement for reintervention has ranged from 1.5/year\textsuperscript{3} to 3.5/year.\textsuperscript{32} In addition, an association has been reported between the number of PTAs performed for recurrent CAS and a decreasing number of days between subsequent recurrences.\textsuperscript{33} Residual stenosis following PTA has been found to be an independent risk factor for lesion recurrence.\textsuperscript{21} Therefore, care should be taken to assure that optimal results are obtained with the procedure. Proper balloon sizing, use of an ultrahigh pressure balloon if necessary and avoiding any residual stenosis are important.\textsuperscript{28}

Although no studies specifically directed toward CAS have been reported, drug-eluting balloon angioplasty (DEA) has been compared with standard high-pressure balloon angioplasty (HPA) for venous stenosis in general. In a large multi-center prospective randomized clinical trial of DEA that was independently adjudicated, cephalic arch cases were included.\textsuperscript{34} Primary patency at 6-months, 6-month dialysis circuit primary patency, and the number of repeat interventions during the 6-months following the index procedure were statistically superior for DEA.
versus HPA. While this data supports the use of DEA for treatment of venous stenosis in general, in the absence of a study specifically focused on CAS, conclusions concerning its place in a treatment strategy for this lesion are not possible.

**Stent placement**

The indications for stent placement in cases with CAS are not well defined. In general, stents have been used in two situations: (1) cases of PTA technical failure characterized by marked elastic recoil or vein rupture and (2) recurrence of the lesion in <3 months. When placing a stent in the cephalic arch, care must be taken to avoid advancing into the subclavian vein. Obstruction of the axillary vein will seriously affect use of it and the basilic vein for future access creation. Because of the curved configuration of the vessel, a flexible stent is required.

Both bare-metal stents (BMS) and stent grafts (SG) have been used. Only a limited number of studies relating specifically to stent placement for the treatment of CAS are available (Table 3). In a meta-analysis of three reports dealing with BMS and seven with SG, primary patency for BMS was 23.3% and 9.5% at 6 and 12 months, respectively. Primary patency for SGs at the same time periods was 82.7% and 44% (both statistically superior to that for BMS and for PTA alone). The 12-month comparison with BMS showed a RR of 0.31 (95% CI = 0.19–0.41, p < 0.0001) and with PTA an RR of 0.34 (95% CI = 0.25–0.44, p < 0.001). BMS compared with PTA showed statistical superiority at 6 months, but no significant difference at 12 months.

A major problem with the use of a BMS has been the development of in-stent stenosis. In one report, restenosis developed in 70% of BMS cases within 3 months. When placing a stent in the cephalic arch, care must be taken to avoid advancing into the subclavian vein. Obstruction of the axillary vein will seriously affect use of it and the basilic vein for future access creation. Because of the curved configuration of the vessel, a flexible stent is required.

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A major problem with the use of a BMS has been the development of in-stent stenosis. In one report, restenosis developed in 70% of BMS cases within 3 months of the index intervention. SGs have an advantage in that they can prevent neointimal hyperplasia from progressing into the lumen of the vessel although stenosis can occur at either end of the device. The number of interventions per patient-year have also been reported to be less with SG use.

Commonly with venous stenosis, there is the development of collateral outflow vessels. In some instances, the total combined cross-sectional area of these collateral vessels is greater than the area of the stenosed vessel itself. Such vessels can also act as a conduit for continued blood flow to distal sites and can facilitate the delivery of therapeutic agents. In addition, the presence of collateral vessels can provide an excellent opportunity for intervention, particularly in cases of recurrent stenosis or occlusion. Therefore, it is crucial to identify and preserve these vessels during the intervention, whenever possible.

<table>
<thead>
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<th>Table 3. Post-intervention patency rates for CAS treatment.</th>
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<tbody>
<tr>
<td>6 months</td>
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<tr>
<td># Cases</td>
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<tr>
<td>Primary (%)</td>
</tr>
<tr>
<td>Angioplasty</td>
</tr>
<tr>
<td>Rajan ('03)</td>
</tr>
<tr>
<td>Kian ('08)</td>
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<tr>
<td>Aitken ('14)</td>
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<tr>
<td>Davies ('17)</td>
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<tr>
<td>Neves ('18)</td>
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<tr>
<td>Miller ('18)</td>
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<tr>
<td>Kim ('19)</td>
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<tr>
<td>Bare-metal stent</td>
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<td>Shamesh ('08)</td>
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<td>Davies ('17)</td>
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<td>Miller ('18)</td>
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<tr>
<td>Stent-Graft</td>
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<td>Shamesh ('08)</td>
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<td>Sawyer ('13)</td>
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<tr>
<td>Rajan ('15)</td>
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<tr>
<td>Jones ('17)</td>
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<tr>
<td>Miller ('18)</td>
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<tr>
<td>Outflow relocation</td>
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<tr>
<td>Chen ('05)</td>
</tr>
<tr>
<td>Kian ('08)</td>
</tr>
<tr>
<td>Wang ('13)</td>
</tr>
<tr>
<td>Sigala ('14)</td>
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<tr>
<td>Jang ('14)</td>
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<tr>
<td>Davies ('17)</td>
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<tr>
<td>Henry ('17)</td>
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<tr>
<td>Kim ('18)</td>
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individual patient characteristics. Most studies dealing with flow reduction have been for the treatment of an existing condition rather than its prevention. Based upon these considerations and with the goal of treating high Qa prospectively, we suggest a threshold of 1.2 L/min for considering access blood flow reduction.

According to a survey conducted by the US DOPPS Practice Monitor 2014, the Qa prescribed by surveyed facilities had a mean of 417 mL/min (median 400 mL/min). For effective dialysis, the Qa should exceed this level by at least 100 mL/min. When Qa has been reduced to levels no lower than 500–600 mL/min for AVFs and 600–700 mL/min for AVGs, post-banding access patency rates have been reasonably good. These levels should be adequate to allow for effective dialysis and high enough to avoid thrombosis.

In selecting an intra-operative post-banding Qa, the relationship between blood flow and blood pressure must be taken into consideration. The values quoted in these recommendations are based upon a normalized blood pressure maintained during the procedure for the final Qa measurement. Since the effect of a stenotic lesion on Qa is directly proportional to the mean inflow pressure, clinical judgment is necessary in dealing with patients with a systemic blood pressure that is either high or low.

Migration of the suture used for endovascular banding has been reported to erode through the wall of a markedly dilated AVF into the lumen and some cases have resulted in pseudoaneurysm formation. As a result, it has been recommended that if the AVF diameter exceeds 2 cm in the postanastomotic segment, surgical revision of the anastomosis to a size smaller than the feeding artery be considered.

**Surgical intervention**

Although it has been used as primary treatment for CAS, surgical intervention has not been widely employed and has generally been used late in the course of the patient with a recurrent problem. Commonly employed interventions include outflow relocation and outflow bypass (Table 2).

**Outflow relocation (OR):** Outflow relocation is a surgical procedure in which the cephalic vein is divided proximally and rotated down to the axillary, basilic, or proximal brachial vein. Ultrasound examination by the surgeon clarifies which option offers the greatest opportunity for success. Fortunately, CAS lesions are generally short and focal and it is unusual to have a problem with available vein length necessary for the transposition. In most cases the procedure can be performed even if a long segment of the cephalic vein is stenotic. Once freed, the vein is transposed by passing it through a subcutaneous tunnel to the upper target vein where an end-to-side venovenostomy is created to restore centrally-directed blood flow.
Although most reports have involved small cohorts of cases, the success rate for these procedures has been relatively good with 6- and 12-month primary patency rates of 57%–95% and 25%–100%, respectively (Table 3). Cumulative patency at 6 and 12 months have been reported at 71%–100% and 92%–100%, respectively. The majority of these re-interventions are required for stenosis that develops at the venovenostomy site.

Bypass: Surgical bypass has generally been performed by tunneling graft material, either saphenous vein or expanded polytetrafluoroethylene (ePTFE), from the proximal uninvolved end of the vein to the internal jugular vein. Using the external jugular vein as a bypass by rotating it down to the cephalic vein has also been reported.

Basics of management

The goal of CAS management should be to restore and maintain access function for the longest possible period of time with the fewest interventions. Complete characterization of the lesion both hemodynamically and anatomically is the first step. This requires both flow measurement and angiographic imaging. Patients with CAS present a relatively broad spectrum as relates to both of these parameters. It is recommended that the brachial artery be used as an indirect measure of access flow, imaged at least 5 cm proximal to the anastomosis. Because the incidence of high bifurcation of the brachial artery has been reported to be in the range of 12%–19%, care must be taken to assure that the measurement is being made from the brachial artery.

There is inadequate evidence available to construct a single CAS management strategy. However, we find two general approaches to the primary treatment of CAS useful: (1) treatment of the anatomical lesion or (2) treatment of the pathophysiology. Although there is a direct correlation between Qa and both the development and recurrence of the CAS lesion, primary treatment directed toward the pathophysiology of the lesion has been largely ignored. The primary approach for managing this lesion has been anatomical, designed toward addressing the pathological anatomy of CAS with PTA as the recognized standard of practice. This has the advantage of being a relatively simple procedure. However, the results obtained with angioplasty are not optimal and the complication rate has been high including evidence that PTA can lead to vessel injury promoting the development of intimal hyperplasia, increasing the risk of recurrent stenosis.

As examples: PTA as primary therapy for symptomatic CAS with an MLD of 2 mm and Qa of 200 mL/min is appropriate; however, a symptomatic CAS patient with an MLD of 8 mm and a large pulsatile vein with a pretreatment Qa >1200 mL/min should have blood flow/pressure reduction. PTA in this second patient may result in a problematic high flow access.

Because of the wide spectrum that characterizes cases of CAS, individualization is critical to appropriate management. Based upon measured Qa, primary (initial) treatment for patients should be categorized as either: (1) PTA-primary treatment—Qa of ≤800 mL/min, or (2) flow reduction-primary treatment—Qa of ≥1200 mL/min for either an AVF or an AVG in a patient with a normal blood pressure. Clinical judgment is required for intermediate patient presentations and adjusting these levels for a case with either low or high blood pressure. For PTA primary cases, initial treatment for the case should be PTA. Qa should be assessed following PTA and if found to be >1200 mL/min, flow reduction should be considered as an ancillary procedure using a Qa of 600–800 mL/min as a target level with normalized blood pressure. Close follow-up should be considered for asymptomatic patients with marginally elevated Qa values. For flow reduction-primary treatment cases, initial therapy should be precision banding using this same Qa level as the target value.

While the measured Qa is used as the defining feature for these two categories, there is a typical clinical picture that corresponds to each (Figure 3). In the PTA primary category, the AVF tends to be less dilated and very hyperpulsatile. A strong bruit and thrill are evident at the arterial anastomosis and the ipsilateral subclavicular area which are predominantly or only systolic. Marked narrowing of the vessel lumen is evident radiographically. With the flow reduction primary category, the AVF tends to be dilated and often aneurysmal, but may be less hyperpulsatile. A thrill and bruit are evident at the arterial anastomosis and the ipsilateral subclavicular area but is not as prominent and may retain its diastolic component. The vessel adjacent to the stenotic lesion is often large resulting in the case being classified as ≥50% stenosis even though the MLD is not abnormal.

Secondary treatment modalities such as stenting and surgery should be reserved for angioplasty failures and early recurrence of the lesion after primary treatment. Which of these two modalities is used should be based upon the treating team’s clinical judgment and local management expertise in selecting an appropriate alternative for each individual patient, considering the patient’s clinical status.

Recommendations for the management of CAS

Management of CAS should be individualized consistent with the goal of maintaining access function for the
longest possible period of time with the least number of interventions. Clinical judgment of the treating team is critical in achieving this goal. Recommendations are offered for each phase in the planning and long-term management of CAS. An algorithm is offered which represents the recommended progression for the management of this lesion (Figure 4). Recommendations made herein reflect a consideration of the published studies such as they are, their clinical interpretation, application, and relevance.

Prevention
- It is recommended that in those cases in which a distal radial-cephalic AVF cannot be created, an upper arm proximal radial-cephalic fistula (prRCF) be given priority over a brachial-cephalic fistula as a second-choice vascular access.
- For patients where anatomy requires a BC-AVF, care should be taken to limit the anastomosis to 4mm or 75% of the diameter of the brachial artery, whichever is less.
- It is recommended that regular maintenance examinations be in place for all patients with a vascular access and particularly for those with a BC-AVF in order to detect excessive blood flow and/or early clinical-hemodynamic evidence suggesting the presence of CAS.

Primary treatment
- Primary treatment of symptomatic CAS (see Indications for Treatment) should be individualized based upon measured Qa and imaging of the lesion.
- Based upon Qa, CAS patients should be categorized as either: (1) PTA-primary treatment—Qa of \(\leq 800\) mL/min, or (2) flow reduction-primary treatment—Qa of \(\geq 1200\) mL/min for either an AVF or an AVG in a patient with normalized blood pressure. Clinical judgment is required for patients with intermediate Qa values and imaging. Individualized treatment plans are necessary depending on clinical status.
- PTA with either a standard (high-pressure) or an ultrahigh-pressure balloon should be used as primary treatment for PTA-primary cases. Care should be taken to avoid any residual stenosis following PTA since it has been shown to have a positive correlation with recurrence of CAS.
- Following a successful PTA, Qa should be measured to assess the result. If \(\geq 1200\) mL/min, flow

![Figure 3](image-url)

**Figure 3.** Clinical features of typical CAS treatment categories, patients with intermediate Qa values and imaging require individualized treatment plans depending on clinical status. (a) clinical appearance of PTA primary CAS (b) angiographic appearance of PTA primary lesion (c) clinical appearance of flow reduction primary CAS (d) angiographic appearance of flow reduction primary lesion.
reduction should be considered as an ancillary procedure using a Qa of 600–800 mL/min as a target. Close follow-up should be considered for asymptomatic patients with marginal Qa values.

- Flow reduction should be used as the primary treatment for flow reduction-primary treatment cases using a Qa of 600–800 mL/min as a target value.
- Precision endovascular flow reduction and minimal invasive surgical flow reduction both offer reliable outcomes.
- If the AVF diameter exceeds 2 cm in the post-anastomotic segment, surgical revision of the anastomosis to a size smaller than the feeding artery should be performed, guided by intraoperative post-revision access flow measurement.

Stent placement

- Consideration should be given to stent placement in the event of failure to obtain optimal results with PTA.
- Unless the individual clinical situation dictates otherwise, a stent should be placed only in cases with two or more recurrences of the same lesion within a 4-month period or less.
- A stent-graft be used in preference to a bare-metal stent.
- In cases with two or more recurrences of the same lesion within a 4-month period or less following stent placement, surgical treatment focusing on establishing a new outflow conduit and bypass options should be considered, based upon the individual situation.

Surgical intervention

- Depending upon the clinical situation, surgical intervention focusing on establishing a new outflow conduit and bypass options should be considered a reasonable alternative to stent placement or stent failure.
- Unless the individual clinical situation dictates otherwise, a surgical modality should be used in cases with two or more recurrences of the same lesion within a 4-month period or less following stent placement.
• The management team should use their clinical judgment in selecting the appropriate modality for the individual patient consistent with the goal of maintaining access function for the longest possible period of time with the least number of interventions.

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