



Role of the Cervical Anterior Spinal Artery in the Endovascular Treatment of Vascular Diseases: Bystander, Accomplice, Victim, or Friend?

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The cervical anterior spinal artery (ASA) is a very important artery arising from the intracranial vertebral artery (VA). It can play different roles in endovascular treatment (EVT) of spinal vascular diseases. The current understanding of these roles is incomplete; therefore, we performed this review. We found that cervical ASA can be involved in many spinal vascular diseases, such as arteriovenous fistula (AVF), arteriovenous malformation (AVM), and aneurysm, and can serve as a collateral channel in proximal VA occlusion. In AVF and AVM, when the cervical ASA is involved, it often plays the role of an accomplice or victim because it acts as the feeder or as a bystander that does not provide blood flow to the AVF and AVM. In cervical ASA aneurysm, the ASA is a victim. During EVT of VA aneurysms or stenoses, the cervical ASA ostia can be covered or occluded, resulting in ASA ischemia. In this situation, the ASA is a victim. In VA occlusion or the subclavian steal phenomenon, the cervical ASA can serve as a collateral channel to provide blood flow to the posterior circulation. In this case, the ASA plays the role of a friend. According to the role of the cervical ASA in spinal vascular diseases, EVT should be determined "case by case." Most importantly, when EVT is performed to treat these diseases, the cervical ASA axis must be preserved. Therefore, understanding the role of the cervical ASA in spinal vascular diseases is crucial.

Keywords: cervical anterior spinal artery, vascular disease, endovascular treatment, arteriovenous malformation, arteriovenous fistula

INTRODUCTION

The cervical anterior spinal artery (ASA) arises from the intracranial vertebral artery (VA) and provides blood to the anterior two-thirds of the spinal cord (1). It may be involved in many cervical spinal vascular diseases, such as arteriovenous fistula (AVF), arteriovenous malformation (AVM), and aneurysm (2–4). In addition, in VA occlusion or the subclavian steal phenomenon, the cervical ASA can serve as a collateral channel (5).

Currently, endovascular treatment (EVT) has become an effective method for cervical spinal vascular diseases (6). However, the EVT can damage the cervical ASA. For instance, during flow diversion (FD) deployment, VA stent angioplasty, or balloon angioplasty, the cervical ASA ostia can be covered or occluded (7). Therefore, the role of the cervical ASA is very complex. It can be an accomplice, victim, bystander, or friend.

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According to the role of the cervical ASA in spinal vascular diseases, EVT should be determined "case by case." It is of most importance that the cervical ASA axis be preserved (8). The current understanding of the role of the cervical ASA in EVT of spinal vascular diseases is insufficient. Therefore, we performed this important review.

CERVICAL ASA ANATOMY

The typical ASA originates as a common trunk from paired VAs, and its origin is 5–17 mm proximal to the vertebrobasilar junction, angiographically presenting with a characteristic midline hairpin (5, 9, 10). High anatomical variability of the ASA origin exists, and it often has either a predominance of one ramus over the other or a sole unilateral ramus of origin (11, 12). The Santos-Franco et al. study reported a less typical bilateral origin of the ASA (13).

The ASA is not a single artery but a series of anastomotic vascular loops. In the cervical region, the ASA is continuous, unlike that in the thoracic region (12, 14, 15). The cervical ASA requires segmental radiculomedullary arteries from the VA (C1–C6), the ascending cervical artery (C3–C4), and the deep cervical artery (C3–C7) (10, 16, 17). In the craniocervical junction, the ascending pharyngeal artery (C2–C4) and the occipital artery (C1–C2) can be involved in the blood supply to the ASA (18, 19). In addition, the supreme intercostal artery can be involved as a feeder to the ASA in the cervical region (C7) (20).

In the cervical region, the radiculomedullary artery from C4–C7 is usually the most predominant and is called the artery of cervical enlargement (16, 21). In addition to the VA, the artery of cervical enlargement can arise as a segmental branch of the ascending or deep cervical arteries (22–24). The angiographic anatomy of the cervical ASA is shown in **Figure 1**.

CERVICAL SPINAL AVFS

Cervical spinal AVFs vary and can be divided into dural AVFs (DAVFs), radicular AVFs, epidural AVFs, and perimedullary AVFs (PAVFs); they are located on the inner or outer surface of the dura, on the spinal nerves, or on the spinal cord (6, 25–28). In cervical AVFs, the ASA can be an active accomplice. In the Hiramatsu et al., study, half of high cervical AVFs were fed by the ASA (6).

Cervical DAVFs

Spinal DAVFs are located near or within the dura of the nerve sleeve, connecting radiculomeningeal arteries with the radicular vein that drains into the perimedullary vein (29). Cervical DAVFs occur in <6% of spinal DAVFs (30–33). In most cervical DAVFs, the ASA acts as a bystander (34–36). Rarely, the ASA can be an accomplice. In the report of Adrianto et al., 2.4% of cervical DAVFs had a concomitant origin of the ASA with the feeder (37).

Currently, EVT is feasible (31, 32). However, when cervical DAVFs originate from the radicular branch that supplies both the fistula and ASA, the EVT must be chosen carefully (31). During EVT, liquid embolic materials can be chosen, and they should penetrate the vein beyond the fistula without disturbing

the ASA. Therefore, the microcatheter should be in a wedged position to ensure no contrast reflux into the ASA (36). In EVTs for other AVFs and AVMs, the microcatheter should be in the wedged position.

For cervical DAVFs, the liquid material N-butyl-2cyanoacrylate (NBCA) and Onyx (Medtronic, Irvine, California, USA) can be used, and NBCA with a lipiodol mixture at a 20–30% concentration is preferred (31). Onyx is limited by the difficulty in achieving venous penetration; at this time, using a balloon-occlusion catheter to assist Onyx casting is helpful, which can promote Onyx penetration (38).

Radicular AVFs

Radicular AVFs are located on the intradural nerve root and are fed by radicular and/or radiculomeningeal arteries that drain into the radicular vein (39, 40). Cervical radicular AVFs have an angioarchitecture similar to that of spinal DAVFs. However, they are different. First, the radicular AVF site is on the nerve root, and the C1 or C2 level is the preferred location (39, 40). Second, in radicular AVFs, the ASA often joins the same radicular artery (6, 40). Third, in radicular AVFs, a long-distance perimedullary draining vein is uncommon (6, 39, 40). Because radicular AVFs often have an ASA blood supply, EVT has a higher risk.

Cervical Epidural AVFs

Spinal epidural AVFs feature a dilated epidural venous pouch that is supplied by paraspinal or paravertebral arteries and drains into epidural plexuses (41–43). Cervical epidural AVFs are not uncommon (**Figure 2**). In the report of Asai et al., 30.8% of epidural AVFs were located in the cervical region and often in the lateral spinal canal, even with bone involvement (44–46). They are divided into types A and B: type A has a small venous pouch with intradural venous drainage, often with congestive myelopathy, and type B has a large venous lake without intradural drainage, often with compressive myelopathy (47–50).

In cervical epidural AVFs, especially in high locations, spinal pial arteries often have a common origin with the feeder; therefore, the ASA can be involved as an accomplice (50, 51). In the Hiramatsu et al., study, 57% of high cervical epidural AVFs were fed by the ASA (6).

EVT is appropriate for cervical epidural AVFs. Transarterial EVT is mostly applied, and liquid embolic material must be avoided in the ASA (48, 52, 53). Sometimes, transarterial EVT of high/middle cervical epidural AVFs is difficult, as these AVFs are usually fed by small and short VA branches (48, 54). If there is a fistulous connection, transvenous EVT is a good choice, and the use of a proximal balloon to control high blood flow followed by coiling or liquid embolic material embolization is helpful (51, 55).

Cervical PAVFs

PAVFs result from direct communication between feeding arteries and enlarged draining veins without the intervening nidus; they are intradural but extramedullary, and in the cervical region, they are usually located on the anterior or lateral surface of the spinal cord (56). The shunting points can be single or multiple (57). Cervical PAVFs are not uncommon; in the Mizutani et al., study, PAVFs in the cervical region accounted for



FIGURE 1 | Angiographic cervical ASA anatomy. (A) VA angiogram showing a long-distance ASA arising from intracranial VA termination (asterisk). (B) VA angiogram showing that the ASA is relayed by the continuous segmental radiculomedullary arteries (asterisks). (C) VA angiogram showing the artery of cervical enlargement (asterisk). ASA, anterior spinal artery; VA, vertebral artery.



FIGURE 2 | Cervical epidural arteriovenous fistula. (A–C) Right VA anteroposterior view (A), lateral view (B), and three-dimensional (C) angiograms showed an epidural arteriovenous fistula (asterisk), the arrow shows the feeding artery and the ASA were involved, with draining to the suboccipital venous plexus (triangle). ASA, anterior spinal artery; R, right; VA, vertebral artery.

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FIGURE 4 | EVT of a cervical intramedullary glomus arteriovenous malformation. (A) MRI showed an intramedullary glomus AVM near the C7 level (circle). (B) Left angiogram of the supreme intercostal artery showed that the AVM was mainly supplied by the dilated radiculomeningeal artery (arrow). (C) The aneurysm in the AVM was coiled first *via* the radiculomeningeal artery (arrow), and then the NBCA was cast. (D–F) Six-month follow-up angiograms showed that the AVM had decreased. Radiotherapy was recommended. (D) shows an angiogram via the right costocervical trunk revealing that the ASA was involved as the feeder. (E,F) show angiograms via the left and right supreme intercostal arteries, revealing that the PSA was the feeder. AVM, arteriovenous malformation; ASA, anterior spinal artery; EVT, endovascular treatment; L, left; MRI, magnetic resonance imaging; NBCA, N-butyl cyanoacrylate; PSA, posterior spinal artery; R, right.

22.4% of all PAVFs (2). In the study of Mizutani et al., of pediatric cases, the rate was 11.8% (2).

The ASA is an active accomplice in cervical PAVFs. In the Endo et al. study of 22 cervical PAVFs, the ASA contributed to shunts in 72.7% of patients (58). Cervical PAVFs can be divided into three types. Type A PAVFs are small, single-vessel fistulas supplied by a single ASA that mostly occur in adult patients. Types B and C PAVFs are giant, multiple-vessel fistulas supplied by the ASA and posterolateral spinal arteries, with high-flow, enlarged, and tortuous draining veins that occur more often in children (59, 60).

Cervical PAVFs can be managed with EVT, and the key aim is ASA preservation as long as possible during fistula obliteration (58, 61, 62). Not all PAVFs are appropriate for EVT. For Type A PAVFs, EVT is difficult because ASA catheterization is problematic; for Types B and C PAVFs, transarterial EVT via the ASA may be a safe and effective choice (57, 63). Certainly, if the ASA is a bystander in PAVFs, EVT is easy (**Figure 3**). In addition, the transvenous path can be used (58). Coils and NBCA are preferred for cervical PAVFs because of the short course of the ASA extending from the VA (64, 65).

CERVICAL INTRAMEDULLARY GLOMUS AVM

Spinal glomus AVM is an intramedullary arteriovenous shunt with an intervening nidus (60, 66). A cervical location accounts for \sim 30% of intramedullary glomus AVMs with multiple feeding

vessels arising from the ASA and posterolateral spinal arteries (67–69). The vast majority of intramedullary glomus AVMs are diffuse, and they are comparatively smaller than juvenile-type AVMs (68). The ASA is often an active accomplice. In the Mizutani et al., study including 69 glomus intramedullary AVMs, 100% of the ASAs were involved (2).

Currently, EVT is a good option for cervical glomus AVMs (70). For ideal EVT, only the nidus is eliminated, while the ASA axis is maintained (71). If the ASA is not chosen as the pathway to perform EVT, then EVT is easy (**Figure 4**). If EVT is performed via the ASA, the embolic agent used is NBCA or Onyx, provided that the microcatheter tip can be placed within the nidus (72–74). If the microcatheter tip can be placed close to the nidus but beyond the angiographically visible normal ASA, NBCA is still a good choice (75).

However, the goal of complete EVT is difficult because the main feeding vessel of the cervical glomus AVM, a perforating artery from the ASA, also serves as a feeding vessel to the spinal cord (76). EVT can be accompanied by inadvertent proximal reflux of the embolic material (72, 77). Therefore, many glomus AVMs cannot be cured by EVT; more often, EVT is adopted extensively as a palliative treatment (78).

CERVICAL ASA ANEURYSM

ASA aneurysms are rare and are mainly located in the upper cervical segment (4, 79–82). They are typically dissections and can be divided into isolated and flow-related types (83, 84). The





flow-related type is common. In the Madhugiri et al., study, 20.8% of spinal aneurysms were associated with AVM, and 7.8% of aneurysms were isolated (80). Flow-related aneurysms are found in 29% of glomus AVMs and 10% of spinal PAVFs (85, 86). Even cervical epidural AVFs can be associated with ASA aneurysms (50).

EVT for isolated ASA aneurysms remains difficult because the catheter system must be constructed via the small-caliber ASA (3). For flow-related aneurysms of the ASA, because the ASA is often dilated, EVT may be feasible (87). When the aneurysm is located at the bifurcation between a large artery supplying the AVM and the ASA, the aneurysm may be suited for coiling

(88). If liquid embolic material is used, the ASA axis needs to be preserved, and only superselective embolization of the branches harboring the aneurysm can be allowed (87, 89).

In addition, conservative management is a reasonable option for ASA aneurysms (90, 91). Even flow-related ASA aneurysms can be managed conservatively. In an Ichiro et al. report, a ruptured aneurysm of the ASA regressed after feeding to an AVF after EVT eliminated the AVF and reduced the hemodynamic stress on the aneurysm (50). The reduction of hemodynamic stress is effective, and even ruptured ASA aneurysms can be cured by hemodynamic remodeling with FD placed in the ipsilateral VA (92).



FIGURE 6 | Flow diversion deployment to treat bilateral VA aneurysms. (A,B) CTA (A) and VA angiogram (B) showing bilateral VA dissecting aneurysms (asterisks in (A)); in (B), the ASA was not observed. (C,D) Angiogram of the bilateral VA showing the flow diversions deployed to treat the aneurysms. ASA, anterior spinal artery; CTA, computed tomography angiography; L, left; R, right; VA, vertebral artery.



FIGURE 7 | Medulla oblongata infarction from ASA ischemia after stent angioplasty. (A) MRI showed a normal medulla oblongata. (B) A right VA angiogram showed stenosis (arrow) of the VA beyond the posterior inferior cerebellar artery. (C) Balloon angioplasty was performed. (D) A balloon-expandable stent was deployed. (E) After stent angioplasty, the stenosis was relieved (arrow). (F) Postoperative MRI showed acute medulla oblongata infarction (arrow), indicating ASA ischemia and resulting in locked-in syndrome. ASA, anterior spinal artery; MRI, magnetic resonance imaging; R, right; VA, vertebral artery.







FIGURE 9 | Cervical ASA as a collateral channel in VA occlusion. (A) Angiogram of the VA shows that the intracranial VA is not continuous, and the ASA connects the proximal and distal segments of VA. (B) Angiogram of the VA shows that there are double branches of ASA, and the ASA connects to the PICA. (C) Angiogram of the VA shows the VA rete mirabile; the ASA is tortuous and dilated to provide blood to the posterior circulation, and an aneurysm is found (arrow). ASA, anterior spinal artery; PICA, posterior inferior cerebellar artery; VA, vertebral artery.

CERVICAL ASA ISCHEMIA FROM ASA OCCLUSION OR COVERAGE BY EVT

During EVT, the ASA ostia can be occluded or covered, resulting in medullary ischemia. The procedures include conventional stent-assisted coiling, VA trapping, FD deployment, VA stent angioplasty, etc. In such cases, the ASA is a victim.

Stent-Assisted Embolization of VA Aneurysms and VA Trapping

According to Wang et al., VA aneurysms can be classified into three types: type I aneurysms, located distal to the posterior inferior cerebellar artery (PICA); type II aneurysms, located at the PICA origin; and type III aneurysms, located proximal to the PICA (93). In theory, EVT for type III aneurysms can result in occlusion of the ASA ostia. However, multiple segmental arterial supplies of the cervical spinal cord make the ASA relatively resistant to ischemia (94–96).

The metal coverage of conventional intracranial stents is low; with their assistance, coiling for VA aneurysms is safe for the

ASA (**Figure 5**) (97). However, when the ASA originates from the aneurysm, coiling with sacrifice of the ASA should be conducted with caution (11). If the collateral circulation is insufficient, occlusion of an unpaired ASA can result in bilateral medial medullary syndrome (11, 98, 99).

Is VA trapping safe? In an Aihara et al., study on the predictive factors of medullary infarction after VA trapping for aneurysms, 30% of the patients suffered medullary infarction. The study showed that the risk was not the length but the anatomical location of VA trapping; therefore, preservation of the ASA origin can reduce the risk of medullary infarction (100). In addition, it is worth noting that, sometimes, despite preserved flow of the ASA, spinal cord hemodynamic infarction can occur due to hypoperfusion (11).

FD Deployment

The VA has fewer critical perforators, and FD deployment in the VA is always considered a safe choice. However, in theory, because the occlusion of an unpaired ASA can result in medial medullary syndrome, FD deployment in the VA still causes



FIGURE 10 | Cervical ASA as a collateral channel in steno-occlusive disease of the subclavian artery. (A) Angiogram of the right VA in arterial phase shows that the compensation of collateral circulation from the right segmental radiculomedullary arteries (red arrows) to the left radiculomedullary arteries (blue arrows) through ASA (asterisk). (B) Angiogram of the right VA in late arterial phase shows the capillary dyeing sign in ASA region (frame). ASA, anterior spinal artery; L, left; R, right; VA, vertebral artery.

concern regarding medullary ischemia from ASA ostia coverage (101, 102).

What is the consequence in the real world? In a recent multicenter study by Adam et al., VA aneurysms were treated by FD, ASAs were identified in 80.9% of aneurysms, 55.6% of ASAs were covered by the FD, and patency after FD coverage at the last follow-up was 89.2% for ASAs. The study showed that FD deployment with coverage of the ASA was not associated with higher rates of occlusion of the ASA or any instances of cord infarction (7).

Considering the potential risk factor, FD deployment should likely be avoided in cases of an unpaired ASA, and FD with coverage of the sole ASA is likely not a favorable treatment option (11). In cases of paired ASAs, FD use could be more liberal; however, FD deployment in bilateral VAs necessitates more caution (**Figure 6**).

VA Stent Angioplasty and Balloon Angioplasty

Acute VA dissection can result in occlusion of the ASA (94, 103). Therefore, there is a danger that intracranial VA stent angioplasty or balloon angioplasty of the VA may injure the ASA ostia, where the dissection formed. In the study of Wang et al. of 55 cases with intracranial VA stenosis, balloon angioplasty and stent angioplasty resulted in 9.1% of cases with perforator injuries of the VA, resulting in ischemic complications, most of which resulted from ASA injury (104). Therefore, the ASA should be

identified. If the ASA is not seen, dissection of its region of origin should be avoided (**Figure 7**).

Injury From a Supporting Catheter

In general, the occlusion of an artery feeding the cervical regions rarely results in an infarction of the spinal cord, as these areas have well-vascularized networks (105). However, similar to occlusion of the artery of Adamkiewicz, which can result in spinal cord infarction, insufficiency of the artery of cervical enlargement is a dangerous situation (**Figure 8**) (106, 107).

During EVT *via* the VA, a larger-sized guiding catheter is often necessary; it can restrict blood flow and produce catheterinduced vasospasm, which can rarely result in thromboemboli and/or hemodynamic insufficiency of the artery of cervical enlargement, resulting in ASA ischemia (24). Therefore, excise VA angiography is recommended, and the catheter should be placed away from the ostia of the artery of cervical enlargement. The guiding catheter should also be continuously flushed with heparinized saline (108).

CERVICAL ASA AS A COLLATERAL CHANNEL

Proximal VA occlusion at the neck is usually compensated *via* the thyrocervical, deep cervical, occipital, and ascending pharyngeal arteries (109). Rarely, retrograde flow through the ASA can serve as a collateral channel, especially in chronic bilateral VA



FIGURE 11 | Coiling of a ruptured aneurysm on the ASA as a collateral channel. (A) Angiogram of the T5 intercostal artery showing an aneurysm on the ASA (arrow), the ASA served as an upward collateral channel. (B) Under the roadmap, the aneurysm was coiled (arrow). (C) Overview of the angiogram showing that the aneurysm was coiled (arrow) and that the ASA was connected to the intracranial VA. ASA, anterior spinal artery; T, thoracic; VA, vertebral artery.

occlusion or a single VA occlusion with other severe stenoses (**Figures 9A,B**) (5). In addition, in VA rete mirabile, the ASA may also serve as the collateral channel (**Figure 9C**) (110).

The degree of the ASA collateral channel varies depending on the presence of other collateral routes (109). Due to the small diameter of the ASA, collateral flow through the ASA may be unable to compensate for critical hypoperfusion, resulting in recurrent strokes (111). VA stenting can markedly improve the flow of the posterior circulation; after successful recanalization of the occluded VA, the collateral channel of the ASA may disappear (5, 112).

Other than in cases of VA occlusion and rete mirabile, the ASA as a collateral channel can occur in the subclavian steal phenomenon, in which the artery of cervical enlargement may stem from the normal VA to connect to a spinal branch of the contralateral VA or costocervical trunk, resulting in ASA syndrome presenting as cervical myelopathy (**Figure 10**) (113, 114). At this time, the subclavian artery should be reconstructed, and the steal path should be occluded (114).

When the ASA acts as a collateral channel, aneurysms can occur on the ASA due to hemodynamic stress (**Figure 9C**) (115). ASA aneurysms can be coiled in selected cases (**Figure 11**) (116, 117). However, due to the tortuous path and remote location, coiling is often difficult and impossible because EVT

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has a low likelihood of parent artery preservation with the latter option (118).

SUMMARY

The cervical ASA is a very important artery. It can be involved in many cervical vascular diseases and has many different roles. In AVF and AVM, the cervical ASA often acts as an accomplice or victim because it acts as the feeder. In ASA aneurysm, the cervical ASA is a victim. During EVT for VA diseases, the cervical ASA ostia can be covered or occluded. In such cases, the ASA is a victim. In VA occlusion, the cervical ASA can serve as a collateral channel to provide blood flow to the posterior circulation. In this situation, the cervical ASA plays the role of a friend. In summary, EVT for cervical spinal vascular diseases should be determined "case by case," and damage to the ASA axis should be avoided.

AUTHOR CONTRIBUTIONS

JY contributed to the conception, design of the manuscript, and critically revised the manuscript. KZ and CL wrote the manuscript. KH and CL collected the medical records of the patients. All authors approved the final version of this manuscript.

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