Review

Mechanical disorders of the cervicocerebral circulation in children and young adults

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ABSTRACT

Mechanical disorders of the cervicocerebral circulation (MDCC) are conditions in which neurological symptoms result from a disturbance of cerebral blood flow attributable to external mechanical forces exerted on extracranial blood vessels by adjacent musculoskeletal structures during head movement that is presumably within a physiological range. The disease spectrum includes bow hunter's syndrome, carotid-type Eagle syndrome, and various dynamic venous compression syndromes. These conditions have distinct phenotypes in children which differ from those expressed in older adults. In contemporary practice, recognition and diagnostic evaluation is the domain of the neuroendovascular specialist. The diagnostic evaluation of MDCC involves significant technical nuance that can be critical to directing appropriate management, particularly in children. This report aims to provide a comprehensive overview of the pathophysiology, anatomical patterns, diagnosis, and treatment for the full spectrum of MDCC that is commonly encountered in clinical practice.

BOW HUNTER'S SYNDROME

'Bow hunter's stroke' was first introduced in a 1978 report describing a 39-year-old man with acute onset lateral medullary infarction during archery practice.[1] Bow hunter's syndrome (BHS) now refers to symptomatic vertebrobasilar ischemia secondary to dynamic external mechanical forces exerted on a vertebral artery (VA) by adjacent musculoskeletal structures during head movement. Head movements typically associated with BHS fall within the physiological range, but may include supraphysiologic movements achieved during chiropractic neck manipulation, athletic maneuvers, or surgical positioning.[2]

Anatomical classification

There are two anatomical types of BHS, each exhibiting a different phenotype with respect to age, VA pathology, level of VA injury, cerebral pathology, and musculoskeletal risk factors (table 1).[2–11] Subaxial BHS is characteristically found in older adults. Osteophytic and musculotendinous subtypes have been reported. Supra-axial BHS is more common in children and young adults. Musculoskeletal risk factors correspond to congenital osseous anomalies, fibrous bands, and hypermobility. Atlanto-axial and atlanto-occipital subtypes are reported. VA injury (often bilateral) is a consistent feature of supra-axial BHS. In supra-axial BHS, traction and/or impaction forces on vulnerable VA segments cause intimal tearing (dissection) due to stretching or intramural hemorrhage due to impaction. Vulnerability seems unique to children owing to ligamentous laxity and immaturity of osseous elements.

The location of VA injury differs according to supra-axial BHS subtype. In atlanto-axial BHS, which is more common, VA dissection occurs at the C1–C2 level just distal to the C2 foramen transversarium (figure 1A, B) (online supplemental file 1). In atlanto-occipital BHS, VA dissection is distal to the C1 foramen transversarium along the posterior neural arch of C1(figure 1C, D, E, F) (online supplemental file 2).

In both subtypes the VA is compromised during contralateral head rotation when the ring of C1 pivots around the axis of C2, moving the contralateral C1 foramen transversarium further away from the subjacent C2 foramen transversarium. Since the VA is variably fixed within the transverse foramina it is subjected to traction forces, the magnitude of which is influenced by local musculoskeletal architecture. Musculoskeletal risk factors such as bone spurs and fibrous adhesions act to shorten the length of the VA immobilized between spreading fixation points.[10] Other factors increase the rotational range of the cervical spine and vascular frailty. Heritable vulnerabilities influence ligamentous laxity and vascular frailty.[3 11]

Osseous factors associated with supra-axial BHS include congenital arcuate foramen, ponticulus posticus, Klippel–Feil anomaly with or without Sprengel deformity, odontoid dysplasia, and aberrant bony protrusions of the occipital condyle and occiput (figure 1D).[10] Iatrogenic factors contributing to supra-axial BHS include the dynamic effects of head positioning for craniospinal surgery as well as the artificial alterations of musculoskeletal anatomy or musculoskeletal relationships to cervical arteries created by surgical interventions.[12 13] For example, iatrogenic supra-axial BHS secondary to hardware failure after spinal fusion surgery has been reported.[12] Theoretically, a range of craniospinal surgeries could create adhesions involving the VA and promote the development of iatrogenic supra-axial BHS.

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Table 1 Characteristics of sub-axial and supra-axial bow hunter's syndrome (BHS)

	Sub-axial BHS	Supra-axial BHS
Patient age	Older adults	Children/young adults
Level of vertebral artery lesion	Lower C-spine (C4–5/C5–6)	Upper C-spine ► Type 1: Atlanto-occipital (0–C1) ► Type 2: Atlanto-axial (C1–2)
Associated vertebral artery dissection	Uncommon	Common
Ischemic pathology	Transient positional vertebrobasilar insufficiency	Acute ischemic stroke
Anatomical risk factors	Type 1: Osteophytic ► Degenerative disease and spondylosis ► Uncovertebral joint osteophytes Type 2: Musculotendinous hypertrophic anterior scalene	► Congenital osseous anomalies ► Hypertrophied soft tissue bands ► Hypermobility

A major cause of childhood posterior circulation stroke

Posterior circulation stroke represents a small fraction of non-perinatal arterial ischemic stroke (AIS) in children.[14] VA dissections are frequently unrecognized due to the underuse of cervical vascular imaging in pediatric clinical practice.[15] Nonetheless, studies still show that non-traumatic VA dissection is responsible for the majority of childhood posterior circulation stroke and that BHS accounts for the majority of childhood non-traumatic VA dissection.[2] BHS most commonly affects children between the ages of 2 and 16 years (mean 9–10 years), but children aged 1 year or younger can also be affected. There is a marked male predominance, which may be due to biological factors, behavioral factors, or both.

Diagnosis

Clues to BHS include a history of high-risk activities (chiropractic neck manipulation, exaggerated athletic maneuvers, and habitual self-adjustment of the cervical spine)[2 3 6 7 11] and torticollis or 'turtle-neck' posturing.[9]

Imaging evaluation begins with anatomical and diffusion-weighted brain MRI and MR angiography (MRA) of the intracranial and extracranial vasculature.[14 15] Posterior-inferior cerebellar or lateral medullary infarction is a helpful sign lateralizing the affected VA. In any child or young adult with posterior circulation stroke, a systematic investigation should be undertaken to exclude VA dissection. Fat-saturated T1-weighted sequences of the neck and black-blood vessel wall imaging may be useful.[16] CT angiography will show underlying anomalous bone that forms the basis of anatomical vulnerability. The authors have found surface-rendered three-dimensional reconstructed images to be particularly helpful as they increase the conspicuity of anomalous bony structures associated with supra-axial BHS (figure 1D). Catheter-directed DSA (CDDSA) is the most sensitive and specific study for the diagnosis of VA dissection. Given the dominant contribution of VA dissection to childhood posterior circulation stroke and the superior sensitivity of CDDSA in the detection of VA dissection, the authors of this review perform CDDSA in any child with unexplained posterior circulation stroke, particularly if the stroke is recurrent.[2]

Definitive diagnosis of BHS relies on vascular imaging with rotational head maneuvers (figure 1A, B, E and F). In the experience of the authors, these studies are best postponed beyond the acute/subacute phase of presentation. We have found that affected patients initially have painful spasm of the paraspinal muscles. Consequently, examiners will be unable to achieve the requisite degree of head rotation needed for diagnosis. Even with effective neuromuscular blockade, rotational head maneuvers may aggravate VA dissection, as reported by Rollins *et al*.[2] Consequently, we recommend delaying rotational head maneuvers until the index VA dissection heals and muscle spasm resolves. This can be assessed on MRA prior to performing CDDSA.

In cooperative patients, multiphase CT angiography with the head in neutral and rotated (dextro and levo) positions is possible. While this may be diagnostic in some cases, CT angiography lacks the temporal resolution of CDSSA and therefore does not permit dynamic adjustments of the head position in response to imaging feedback or provide information about the quality or direction of arterial flow across different anatomical segments. Consequently, interpretation can be difficult. Moreover, excess radiation is a particular concern in young children. Doppler ultrasound with head maneuvers avoids radiation, provides flow information and may be diagnostic, but it provides limited anatomical detail.[17] There is extensive literature describing the diagnosis of BHS by CDDSA in awake adults.[18] CDDSA is also the most widely reported approach to pediatric diagnosis.[5 8 10 11] In children, rotation of the head is performed by the angiographic team with the patient under general anesthesia and neuromuscular blockade. Safe manipulation of the cervical spine is possible because it is performed with live fluoroscopic guidance. The effect of head rotation on VA flow should be monitored in real time as the index VA is simultaneously injected with contrast media. Rotation of the head should be performed slowly, during continuous contrast injection of the VA (online supplemental file 1) (online supplemental file 2). Rotation should be stopped as soon as flow arrest is demonstrated to prevent VA injury, which has been reported.[2] In order to prevent a pseudo-occlusion misdiagnosis, rotation should not be initiated until the VA is filled with contrast. A critical goal is anatomical localization of the VA pathology and underlying musculoskeletal factors to facilitate surgical treatment planning.

Management

Initial management should be directed at treatment of AIS according to recommended guidelines.[14] If BHS is suspected, we initiate cervical spine immobilization with a hard collar. Secondary stroke prevention is achieved with antithrombotic or antiplatelet agents as soon as permitted. Depending on the extent of cerebellar infarction and clinical examination, decompressive surgery may be indicated.

Non-surgical management of supra-axial BHS in children has been associated with a high rate of recurrent stroke. In a recent series of 10 pediatric patients, Fox *et al* found that 80% had recurrent stroke despite appropriate medical therapy.[10] For comparison, in a large adult patient study only 1.5% of those with medically managed VA dissection had recurrent ischemic events during 12 months of follow-up.[19]

Although therapeutic VA occlusion has been reported,[10] we generally advise against this given the frequent occurrence of synchronous or asynchronous contralateral VA dissection in pediatric BHS.[2 4 8 10]

In older adult patients with intractable vertebrobasilar insufficiency due to unilateral supra-axial BHS and flow-restrictive atherosclerosis of the contralateral VA, stent angioplasty of the atherosclerotic lesion has been effectively used as an indirect

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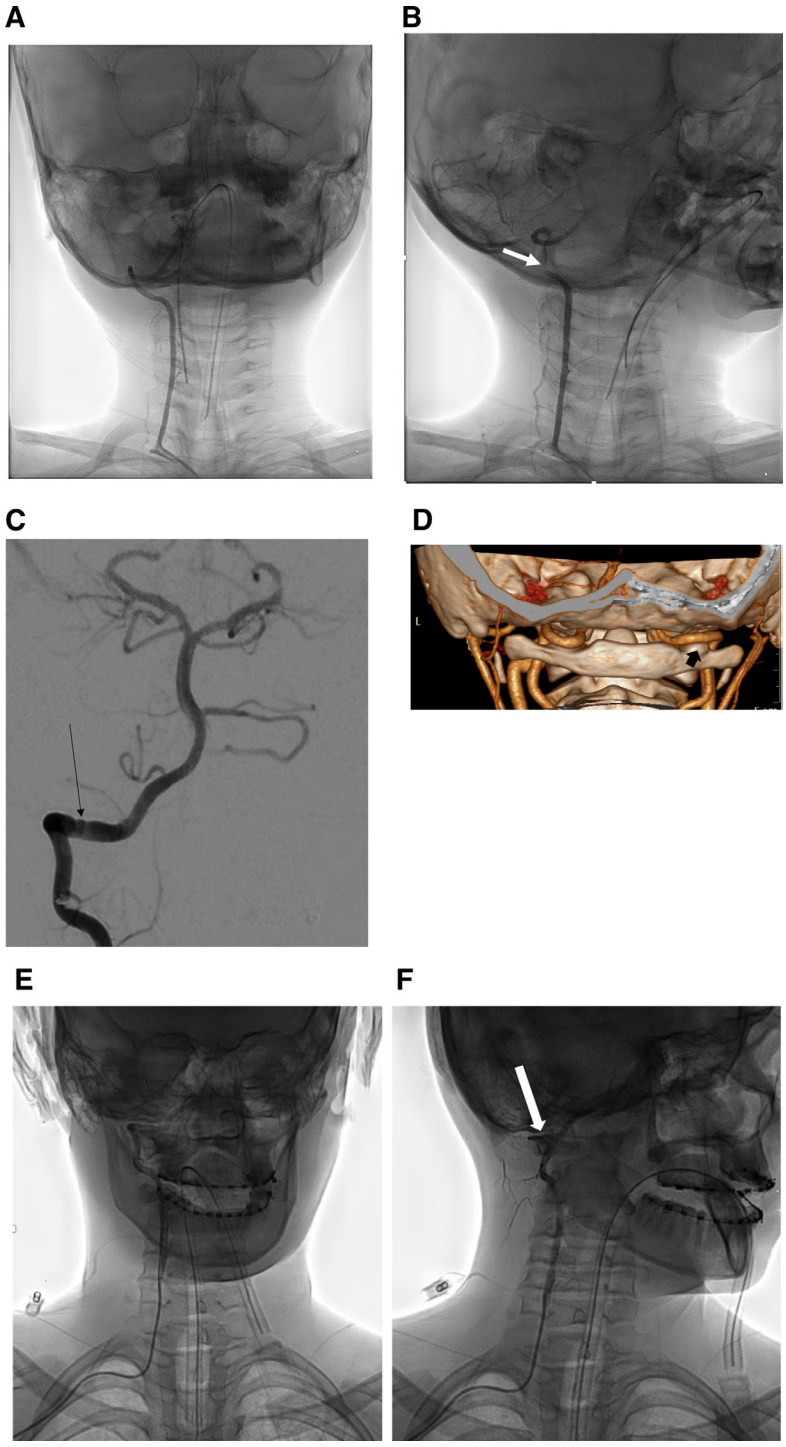

Figure 1 (A) Boy aged <5 years with prior right vertebral artery dissection at the C1–2 vertebral level. When the head is in the neutral position the right vertebral artery is angiographically normal. (B) When contralateral head rotation is performed by the angiographic team under general anesthesia with neuromuscular blockade there is reversible occlusion of the right vertebral artery (white arrow) which co-localizes to the site of prior right vertebral artery dissection at the C1–2 vertebral level consistent with the atlanto-axial subtype of supra-axial bow hunter's syndrome. (C) Teenage boy with prior right vertebral artery dissection distal to the C1 foramen transversarium. (D) Surface-rendered 3D image reconstructed from companion CT angiogram shows associated ponticulus posticus (short black arrow). (E) Right vertebral artery angiography with the head in the neutral position performed 3 months later shows interval healing of the dissection with normal flow. (F) When contralateral head rotation is performed by the angiographic team under general anesthesia with neuromuscular blockade there is reversible occlusion of the right vertebral artery (white arrow) which co-localizes to the site of prior right vertebral artery dissection consistent with the atlanto-occipital subtype of supra-axial bow hunter's syndrome.

approach to correct vertebrobasilar insufficiency.[20 21] Although some authors have reported direct endovascular stenting of the BHS lesion as a treatment for pediatric BHS,[6 7 10] we believe that technical and pathophysiological constraints limit stenting as an effective strategy for vessel traction and impaction injuries. Endovascular stent failure and vessel occlusion due to

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unresolved conflict of bony structures with stented cervical arteries has been reported.[22] Therefore, at present, most authors agree that patients with supra-axial BHS should receive definitive surgical treatment to prevent recurrent stroke. We favor delaying surgical treatment until the index VA dissection heals, diagnostic evaluation including dynamic CDDSA can be performed, and secondary stroke prevention with antithrombotic drug therapy can be safely discontinued.

Surgical treatment

Two surgical strategies are used in the definitive treatment of supra-axial BHS: VA decompression/transposition and spinal fusion.[18 23] The strategy and specific technique should be individualized to each patient based on critical analysis of clinical and imaging data. The VA pathology and associated musculoskeletal factors should guide selection of the optimal intervention.

VA decompression and transposition using a posterior-lateral or far-lateral approach has been widely reported as a treatment for atlanto-axial BHS.[2–5 10 18] Similar techniques have been reported for atlanto-occipital BHS management with good results.[3–5 9 10 18] Authors favoring VA decompression emphasize resection of anomalous bone, release of fibrous bands, C1 hemilaminectomy, and transposition of the VA from the sulcus arteriosus. Follow-up angiography with head rotation challenge is performed 8–12 weeks after the patient has fully recovered and muscle spasm has resolved. Some residual kinking of the VA is considered normal. Examination of both VAs is recommended as unilateral VA decompression and transposition may unmask contralateral BHS. Although some authors report excellent outcomes with VA decompression and transposition,[5 10 18] others have reported high stroke recurrence rates.[3–5 11 18 23] While proper patient selection plays a critical role, the relative success of VA decompression is proportional to the surgeon's experience with the technique.[4 18] We also believe that decompressive surgery is most efficacious when a focal target (eg, bone spur, arcuate foramen) is evident on pre-surgical imaging.[10]

C1–C2 fusion has been widely reported as a definitive treatment for atlanto-axial BHS.[2 4 5 7 10 11 18 23 24] Advocates cite superior efficacy and durability, particularly for patients with bilateral BHS. Moreover, advancements in technique allow for less invasive procedures with enhanced safety.[2 23 24] The most commonly reported fusion techniques include C1–C2 fixation using C1 lateral mass screws and C2 pars or pedicle screws. Critics of fusion cite limitations on cervical motion and the potential developmental and growth implications in skeletally immature patients. Notably, Braga *et al* reported no loss of mobility following C1–C2 fusion in their series of patients aged <10 years.[24] C1–C2 fusion appears to be efficacious particularly for atlanto-axial BHS when no focal target for decompression is found but dynamic occlusion of the VA is demonstrated by CDDSA with head rotation challenge.[24]

Less well understood is the role of C1–C2 fusion for atlanto-occipital BHS. Some authors suggest that VA dissection rostral to C1 usually involves a rotational mechanism of injury driven by C1–C2 mobility.[24] Consequently, if no focal target for decompression is identified in such cases, C1–C2 fusion may be beneficial. Our experience shows that contralateral head rotation produces dynamic VA occlusion that co-localizes to symptomatic atlanto-occipital dissection sites rostral to C1.[20] Accordingly, some authors have performed occipito-cervical fusion for atlanto-occipital BHS without a focal surgical target, but studies are limited.[9] Taking these considerations into account, we favor an anatomically and physiologically oriented treatment algorithm (figure 2).

STYLOCAROTID SYNDROME

Definition

The stylohyoid syndrome reported by Watt Eagle in 1937 is a symptom complex arising from an elongated styloid process.[25] Eagle described a classic pattern and a vascular pattern or stylocarotid syndrome (SCS), each related to styloid compression of adjacent structures. More recently, the role of the styloid process in venous compression has been recognized. According to clinicopathological studies, an elongated styloid process is >3 cm and is most often bilateral.[26] Although present in a minority of the population, the majority of affected individuals are asymptomatic.

Clinical manifestations

Most patients with symptomatic styloid process elongation present with chronic cervicofacial and/or cervicopharyngeal pain and dysphagia. In the classic pattern, intraoral palpation of the tonsillar fossa causes referred pain to the ear, face, or head. In SCS, an elongated or deviated (laterally or medially) styloid process compresses the internal carotid artery (ICA), or less commonly the external carotid artery. Iatrogenic factors such as radiotherapy have been associated with alterations of the styloid process and the development of acquired SCS.[27] Other artificially acquired alterations of cervical anatomy created by surgical dissection or radiotherapy could theoretically play an important role in the development of SCS. In cases of ICA compression, pain is referred to supraorbital or parietal regions and exacerbated by ipsilateral head rotation. Mechanical injury of the ICA resulting in dissection further complicated by AIS or aneurysm/pseudoaneurysm is a well-reported, although rare, manifestation in young adults.[28] In children, the condition is exceedingly rare. Of 34 cases reported in the literature since the millennium, only a single case of symptomatic SCS in a pediatric patient has been reported.[29]

Diagnosis

CT with three-dimensionally reconstructed images has supplanted older radiographic techniques for initial diagnosis and treatment planning due to widespread availability and superior anatomical detail.[30] MRI is indicated in suspected SCS. Non-contrast brain MRI to include diffusion-weighted and T2-FLAIR sequences will reveal new or prior ischemic insults. High-resolution contrast-enhanced black blood vessel wall imaging at 3 Tesla, in conjunction with MRA, will reveal ICA injury.[31]

The magnitude of ICA compression depends on the position and orientation of the styloid process, which in turn depends on head position. Consequently, vascular imaging with head maneuvers has improved diagnostic sensitivity and specificity.[32] While CT angiography has the benefit of being non-invasive, higher contrast and radiation partially offset the advantages. Additionally, CT imaging with head maneuvers is often non-diagnostic due to logistical challenges. Doppler ultrasonography with head maneuvers may reveal dynamic compression of the ICA and is less prone to logistical challenges.[33] CDDSA with head maneuvers is useful in equivocal cases. Angiograms of both common carotid arteries are best performed with the head in neutral, rotated (dextro and levo), and flexed positions.

Treatment

The only established treatment for SCS involves shortening of the styloid process through a transoral or transcervical incision.[34] Advantages of the transoral approach include the relatively straightforward dissection and cosmesis. The external

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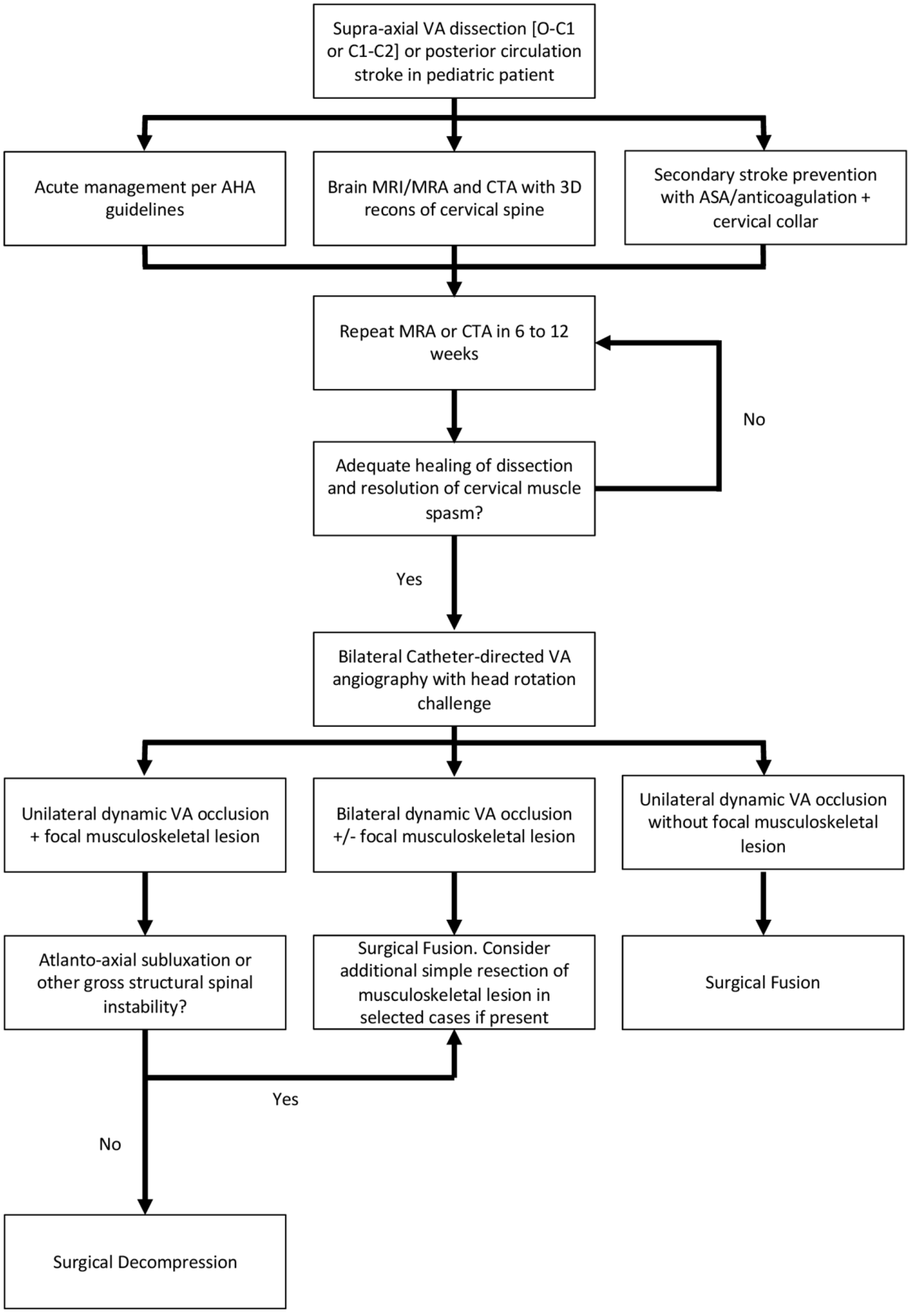

Figure 2 Proposed algorithm for the work-up and treatment of supra-axial pediatric bow hunter's syndrome. AHA, American Heart Association; ASA, acetylsalicylic acid; CTA, CT angiography; MRA, MR angiography; VA, vertebral artery.

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transcervical approach requires a skin incision behind the angle of the mandible, opening of the platysma and superficial fascia with exposure of the sternocleidomastoid and digastric muscles. The main advantage of an external route is the decreased risk of bacterial contamination and deep cervical infection by oral flora. Successful stent angioplasty treatment has been described in an adult patient presenting with rotational carotid insufficiency after laryngectomy and radiation for carcinoma, although only short-term clinical follow-up is reported.[35] In such cases, mechanical forces exerted on the carotid artery are attributable to fibrous scar tissue, which may be compliant relative to the elongated styloid process. Multiple examples of stent failure and carotid occlusion due to persistent conflict of the styloid process with stented carotid arteries have been reported with different stent devices.[22 36]

JUGULAR COMPRESSION SYNDROME

Clinical and anatomical features

There is growing evidence that dynamic mechanical obstruction of the cervical internal jugular veins by bone and muscle can express clinical neurological symptoms in young adults.[37] Osseous mediators of compression are more common and include the transverse process of the atlas and styloid process. In affected patients, alterations in head position which move the styloid and ipsilateral C1 transverse process closer pinch the internal jugular vein, aggravating symptoms. Conversely, alterations in head position which move the styloid further from the ipsilateral C1 transverse process relieve symptoms. Musculotendinous jugular compression occurs between the sternocleidomastoid muscle anteriorly and posterior musculotendinous structures in the lower neck (anterior scalene and omohyoid muscles). These compression zones are often located at the C6–C7 vertebral levels.

Although there is a paucity of literature concerning jugular compression syndrome in children, the accumulated clinical experience of the authors reveals patterns similar to those reported in adults. The clinical presentation and related anatomical mechanisms are illustrated by three cases (table 2 and figure 3). Presenting symptoms most often include headache, brain fog, and declining scholastic performance. Intracranial hypertension symptomatic with papilledema has also been reported. Symptoms often are positional and can be worsened or alleviated with head movement. Valsalva maneuvers characteristically aggravate symptoms. The diagnosis is supported by correlation with dynamic luminal narrowing and venous pressure gradients. Early experience in adults and children undergoing jugular decompression surgery suggests that improvements in venous drainage are associated with durable relief of symptoms.[37 38]

Diagnosis

Non-invasive imaging, ideally with MR venography or CT venography, will reveal internal jugular vein stenosis adjacent to musculoskeletal compressors within zones of vulnerability at C1 or C6–C7 vertebral levels. CT venography is less prone to flow artifacts and reveals the relationship of venous stenoses to adjacent bone in more detail. In isolation, the typical findings of MR and CT venography are non-specific because they are frequently incidental.[39] Diagnostic specificity is increased when a stenosis is made worse by provocative adjustment to a symptomatic head position. Dynamic engorgement of venous collaterals is common.

Catheter-directed transvenous angiography with measurement of venous pressure gradients between the superior sagittal sinus and right heart allow for definitive diagnosis. The examination is initially conducted with neutral head position and subsequently with provocative adjustments ranging from best to worst. In theory, the pressure gradient between the superior sagittal sinus and right atrium should not be altered by head position within the physiological range.

Treatment

At present there are approximately 100 reported cases of surgically treated jugular compression syndrome, including one case of a pediatric patient treated by C1 transverse process resection and subsequent internal jugular vein stenting.[38] Reported interventions are typically individualized and oriented to patient-specific anatomical mechanisms. Resection of compressive musculoskeletal structures, release of constrictive perivascular or mural fibrous bands and/or luminal dilatation may all play a therapeutic role in different patients or within the same patient.

Styloidectomy and venous angioplasty/stenting account for the majority of reported treatments, with both interventions achieving a moderate to high degree of clinical success in properly selected patients.[38] Although C1 lateral mass resections have been consistently associated with clinical success, reported experience is limited to just two cases performed in combination with other interventions. Similarly, there are a limited number of reported cases of myomectomies resulting in successful resolution of symptoms. Regardless of the treatment approach, reported clinical follow-up is very limited and the rate of symptom relapse is not yet known.[38]

CONCLUSIONS

Posterior circulation AIS in children and young adults should prompt a systematic neuroimaging investigation for VA dissection. When non-traumatic VA dissection is found, BHS should be suspected. Acute management should include cervical

Table 2 Clinical data for illustrative cases of pediatric jugular compression syndrome

Pt	Age/sex	Dominant venous system R/L	Compression mechanism	Symptoms	Diagnosis
1	Teenage/M	R	Mild to moderate bilateral osseous: transverse process of C1 and styloid process Severe bilateral musculotendinous: scalene/omohyoid	Headaches associated with declining scholastic and athletic performance, aggravated by head extension and left lateral head tilt	Provocative head maneuvers increase venous pressure gradient from 4 mmHg to 10 mmHg
2	Teenage/M	L	Severe bilateral osseous: hypertrophic transverse processes of C1 and styloid processes Mild to moderate right and severe left musculotendinous: scalene/omohyoid	Headaches and brain fog aggravated by rotation of head to right	Provocative head maneuvers increase venous pressure gradient from 3 mmHg to 10 mmHg
3	Teenage/M	B	Severe bilateral osseous: hypertrophic transverse process of C1 and styloid process	Headaches and vertigo aggravated by leftward head rotation and extension	Provocative head maneuvers increase venous pressure gradient from 3 mmHg to 10 mmHg

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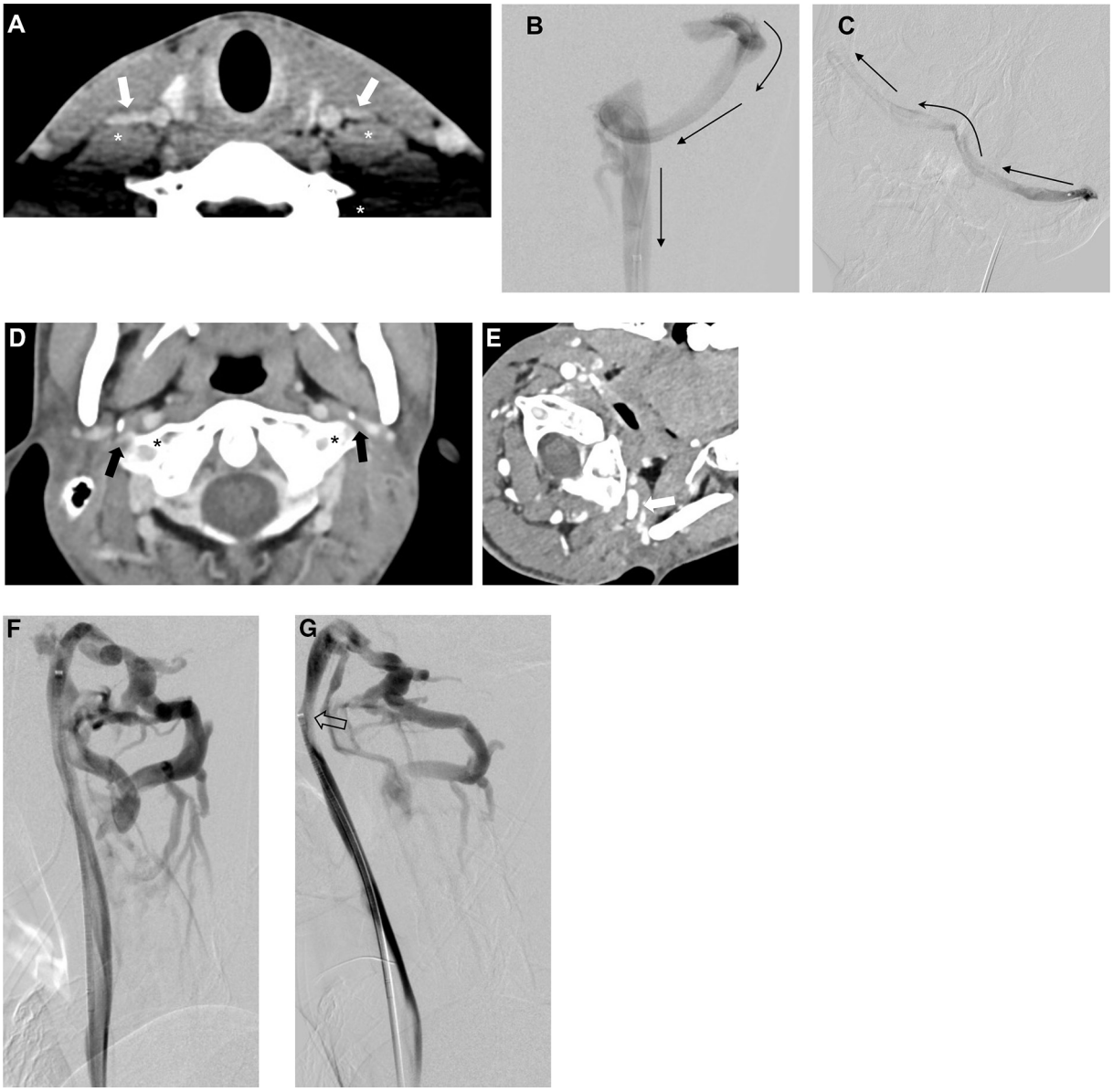

Figure 3 (A) Axial CT venogram image in a teenage student athlete with drastic reduction in grades and athletic performance shows severe left worse than right bilateral internal jugular vein stenosis (white arrows) in the inferior neck predominantly resulting from compression posteriorly by the anterior scalene muscles (*). (B) Frontal projection DSA with left transverse sinus injection at the neutral position in the same patient shows antegrade venous flow with brain–heart pressure gradient of 4 mmHg (black arrows show direction of blood flow). (C) On provocation (head extension with left head tilt), the same injection shows flow reversal via the torcula to the right transverse sinus with brain–heart pressure gradient of 10 mmHg (black arrows show direction of blood flow). (D) At neutral position, axial CT venogram image in a teenage boy complaining of head pressure and brain fog shows large C1 transverse processes bilaterally (*) causing severe stenosis of bilateral internal jugular veins (arrows). The patient reports that his symptoms are worse with far left and far right head rotation (worst position), and least severe with moderate left head rotation (best position). (E) At best head position (moderate left head rotation), axial CT venography images show decreased left internal jugular stenosis at the C1 level (white arrows). (F) Lateral projection catheter-directed DSA with right internal jugular vein injection shows extensive collateral drainage via the anterior condylar system at the neutral head position. (G) Same injection at worst head position shows worsened right internal jugular vein stenosis at the C1 level (black open arrow) with worsened collateral drainage via the venous plexus of the vertebral foramen transversaria. Venous pressure measurements at worst head position show brain–heart gradient of 10 mmHg with most significant reduction at the skull base, likely representing outflow relief via the anterior condylar system.

spine immobilization and secondary stroke prevention with antithrombotic or antiplatelet therapy. Definitive diagnosis comprising CDDSA with head rotation challenge should be postponed until the VA dissection is healed and skeletal muscle spasm has resolved. Definitive treatment options including VA decompression and spinal fusion should be tailored to address the anatomical level of the VA lesion and underlying musculoskeletal factors revealed by diagnostic evaluation.

Elongated styloid process is most often incidental, although it is occasionally a cause of symptomatic carotid impingement during head rotation. Symptom manifestations include positional cranial pain and carotid dissection. Vascular imaging with

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dynamic maneuvers may be used to confirm the diagnosis of suspected SCS. Surgical removal of the styloid process is curative.

Osseous and musculotendinous patterns of symptomatic jugular compression syndrome, aggravated by changes in head position, are increasingly recognized as a reversible cause of headache, neurologic dysfunction, and vision loss. Catheter-directed measurements of venous pressure gradients with provocative head maneuvers support the diagnosis. Surgical decompression of affected venous segments may produce durable symptom relief.

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