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Osteovascular Conflicts in the Neck Region and Cerebrovascular Events: Illustrative Cases and Literature Review

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Abstract

Study design: Literature Review.

Objective: Abnormal bone structures in the neck can cause headache, neck pain, and difficulty swallowing, but also cerebrovascular events. We introduce the term "osteovascular conflicts" to describe this phenomenon. The objective of this study was to conduct a literature review of such conflicts involving the anterior and posterior cerebral circulation. Furthermore, we aimed at presenting additional illustrative cases from our institution both for increasing awareness for unusual osteovascular conflicts, and for assessing the practice and care of such patients.

Methods: We focused on osteovascular conflicts in the neck leading to cerebrovascular events related to an abnormal bone structure causing arterial or venous compression, dissection, and/or occlusion. We excluded pure vascular forms without cerebrovascular repercussions. Our PubMed/MEDLINE search for articles published in any language and for which an English abstract was available (from 1966 to 2022) included Eagle's neurovascular, bow hunter's syndrome, and golfer's stroke, excluding trauma-induced artery dissections or compressions and those concerning systemic bone disorders. We also provided illustrative cases collected by the authors.

Results: All studies were either case reports or small case series. We found 82 cases of Eagle's neurovascular, 258 of bow hunter's syndrome, and 17 golfer's stroke cases. Mean ages were 52, 48, and 47 years, respectively. Male predominance was evident: 81% for Eagle's, 74% for bow hunter's, and 93% for golfer's.

Conclusion: Osteovascular conflicts are rare but important causes of cerebrovascular events and often go unrecognised. A greater awareness of cerebrovascular symptoms related to these conflicts can facilitate early diagnosis and treatment.

Keywords

stroke, osteovascular, bone disorders, transient ischemic attack, neck, bow hunter, Eagle's syndrome, golfer's stroke

Introduction

Elongated or misshapen bone structures can rarely cause mechanical conflicts with blood vessels, which can lead to vascular events. We propose the term osteovascular conflict to describe such phenomena concisely and unambiguously. The Greek prefix osteo- and its Latin equivalent osseo-both convey the meaning "bony." Although it would be correct to say "osseovascular," since the word vascular is derived from the Latin noun vāsculum (small vessel), we suggest the

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Greek-Latin hybrid "osteovascular" in line with terminological precedents, such as "hypertension" instead of "supertension."[1] Although infrequent, osteovascular conflicts are an important cause of cerebrovascular events and often go undetected. Specific syndromes resulting from the direct contact of a bone structure with a brain-supplying vessel have been described, but no umbrella term has been used in the literature so far: Eagle's neurovascular syndrome[2,3] (Figure 1), also known as stylocarotid syndrome, bow hunter's syndrome,[4] also known as rotational vertebral artery syndrome (Figure 2), and the often related golfer's stroke,[5,6] as well as stroke, amaurosis fugax or transient ischemic attack (TIA) after strenuous coughing associated with bone structures.[7] This article discusses cerebrovascular events related to osteovascular conflicts in the neck. Cases of the jugular variant of Eagle's syndrome are briefly reviewed, although they are not the focus of this paper. Neck artery dissections related to clear trauma such as vertebral fractures or arterial disease caused by systemic bone disorders are beyond the scope of this review.

The epidemiology of these events is only documented by reviewing case reports and short case series because of their rarity and the absence of systematic studies. Eagle[2] himself mostly concentrated on painful conditions from conflicts between an elongated styloid process and adjacent soft tissue structures. In his second paper on the subject, he highlighted a carotid variant of his eponymous syndrome, observing its presence in approximately 12 of 211 cases (5.7%). He wrote that "[…] an elongated styloid process which fits so tightly against [the external or internal] carotid artery as to impair its circulation may cause the carotid artery syndrome," but only reported on "vascular pain" as a consequence, not cerebrovascular events.[8] In his research, he documented an overall prevalence of elongated styloid process of 4% in the general population and estimated a lifetime incidence of any form of Eagle's syndrome of .16%.[9,10]

Since then, other osteovascular syndromes have been documented (bow hunter's syndrome first by Sorensen et al in 1978,[11] golfer's stroke first by Taniguchi et al in 1993[12]), each associated with cerebrovascular repercussions and symptoms related to the specific anatomical location of the osteovascular conflict. We have attempted to comprehensively review some of the most prevalent syndromes to provide a clearer understanding of these events in terms of age, sex, laterality, mechanisms, clinical presentation, and potential treatment options. We also have added exemplary cases from our own experience for illustrative purposes and for assessing the practice and care of such patients in our own institution.

Methodology

We compiled a list of well-known osteovascular conflicts and conducted a literature review of PubMed/MEDLINE articles published in any language and for which an English abstract was available (from 1966 to December 2022) using PRISMA guidelines. We focused on the three main entities: Eagle's neurovascular syndrome, bow hunter's syndrome, and golf-related stroke. We also collected cases related to the jugular vein variant of Eagle's syndrome but discussed these separately, given that only a small fraction had signs of cerebral venous ischemia. The specific keywords we used are detailed in the supplemental material (Supplemental Table 1). We included case reports, case series, and review articles (if new cases were reported). We also provided a flow-chart illustrating which studies were included or excluded (Supplemental Figure 1). From these, we extracted epidemiological data about age, sex, and the affected side. We also selected the articles we deemed crucial for our discussion on

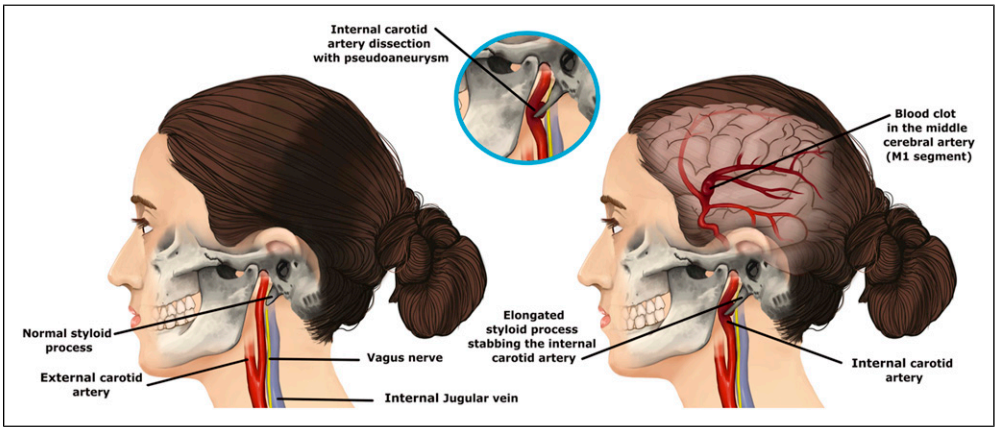

Figure 1. Illustration of Eagle's neurovascular syndrome (one possible mechanism). The image on the left shows a normal styloid process, while the image on the right an elongated styloid process characteristic of most variants of Eagle's syndrome (alternatively, a calcified stylohyoid ligament can cause the same symptoms). The elongated styloid process impinges on the internal carotid artery, leading in this case to an internal carotid artery dissection with pseudoaneurysm and thrombus formation with displacement of the thrombus to the middle cerebral artery. Notice the anatomical proximity to the internal jugular artery and vagus nerve that may also be affected in some variants. The image is only intended for illustrative purposes and should not be used for neurosurgical guidance.

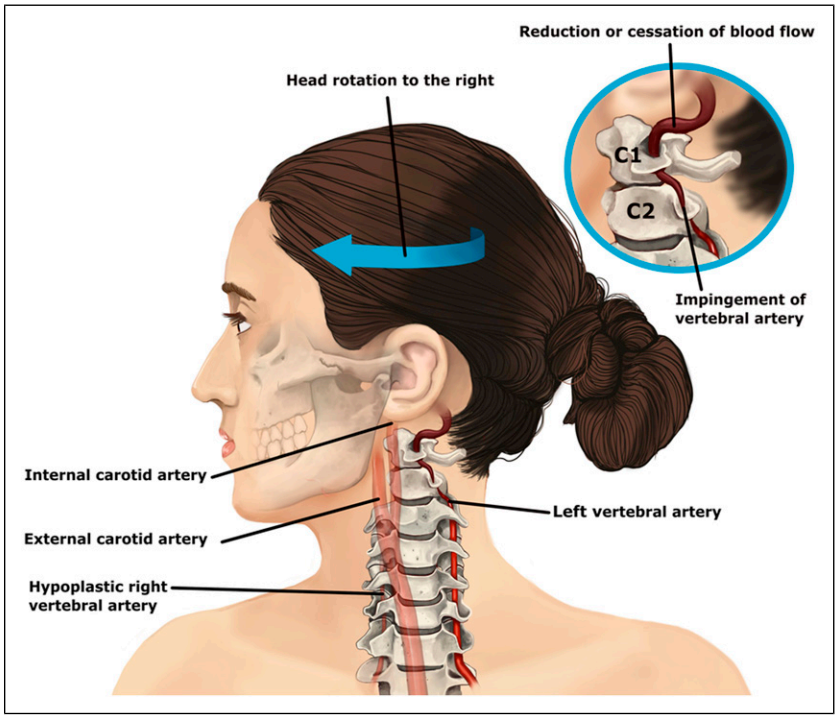

Figure 2. Illustration of bow hunter's syndrome (one possible mechanism). This image depicts a patient with her head fully rotated to the right, with tethering to a bony structure, such as an osteophyte or a hypertrophic ligament, of the contralateral vertebral artery at the atlanto-axial level (C1-C2). The resulting mechanical compression can lead to temporary reduction or cessation of blood flow to the brain, or to the formation of blood clots. The underlying mechanism in golfer's stroke is similar. The image is only intended for illustrative purposes and should not be used for neurosurgical guidance.

the clinical presentation, pathophysiology, and management of these syndromes. In addition, we added our experience and management of other specific osteovascular conflicts encountered over 20 years by the last author (P.M.) at the Lausanne University Hospital, rarely described in the literature and therefore not suitable for a review according to the PRISMA guidelines.

Results

Regarding Eagle's syndrome, we found a large variability in both the definition and applicability, with results yielding a wide variety of clinical presentations ranging from orofacial pain, headache, vertigo to cerebrovascular symptomatology. We identified a total of 82 cases of carotid variant of neurovascular Eagle's syndrome (see Supplemental Figure 1). Of these, 30 might have experienced TIAs or a minor stroke (the exact number was not determinable due to inconsistent MRI use) and a minimum of 52 suffered strokes. Central to these cases was the presence of an elongated styloid process and/or a calcified stylohyoid ligament. The mean age based on 78 patients was 52 years, with an age distribution resembling a bell curve spanning from the second to the eighth decade and centering around 40-50 years (see age distribution in Supplemental Figure 2). Fifty-two patients out of 79 were male (81%). There was a left-sided predominance with 37 cases involving the left carotid artery, and almost a third of the patients (24 cases) had bilateral carotid artery involvement. Additionally, we identified 13 patients with the jugular variant of Eagle's syndrome.

Additionally, we identified 86 patients with confirmed or presumed jugular vein stenosis caused by an elongated styloid process (details in Supplemental Figure 1). Of these, only eight patients (9%) had venous thrombosis, but clinical details were available for only three cases. An MRI was performed in one of these cases, which did not show cerebral venous infarction. Symptoms similar to idiopathic intracranial hypertension were far more common, reported in 83% of cases. Out of 85 patients, 48 were male (56%). The age ranged from 15 to 87 years, with a mean age of 45 years calculated from 20 cases (individual data were not available from larger case series). Out of 50 cases, 68% had bilateral jugular vein stenosis, 18% had stenosis on the right side, and 14% on the left.

Regarding bow hunter's syndrome, we identified 257 cases (Supplemental Figure 1). Of these, 172 likely developed no infarction (ie, TIA), and 82 had a stroke. Patient ages, based on 233 documented cases, spanned from 2 to 85 years, with two age peaks: a primary around 50-60 years and a secondary, less pronounced, between 10 and 20 years (see age distribution in Supplemental Figure 2). Out of all patients, 187, or 74%, were male. There was a left-sided predominance, with 142 out of 246 cases (56%) involving the left side, while 24 cases, or 9%,

demonstrated bilateral involvement. In most of the 244 cases with a documented compression site (62%), the affected area was at the C1 to C2 level (mostly caused by a contralateral head rotation), while 34% had a subaxial compression (mostly caused by an ipsilateral head rotation). In 10 cases (4%), the compression resulted from an abnormality, typically a bone spur, of the occipital bone (C0).

Golfer's stroke was identified in 17 cases, with a mean age of 47 years and only one documented female case. Of note, most cases (15 patients) affected the vertebrobasilar circulation (10 on the right, 3 on the left, 2 bilateral) and likely arose from a mechanism identical to that of bow hunter's syndrome. Two other cases occurred in the anterior circulation, following dissections of the right carotid artery.

In addition to the results of the review of these three well-described osteovascular conflicts, we have presented a case of Eagle's neurovascular syndrome from our own institution and identified four additional illustrative cases of other such conflicts which are presented in the discussion section, specifically strokes resulting from bilateral carotid artery compression by the hyoid bone during intense coughing, from a vertebral artery compression due to an intraosseous ganglion cyst, and from a vertebral compression caused by an ossified thyroid cartilage. Furthermore, we have detailed a case of vertebral artery compression by an intervertebral disc.

Discussion

Literature Review on the Main Osteovascular Conflicts

For the main osteovascular conflict in the anterior circulation, the Eagle[10]'s syndrome or stylocarotid syndrome, our literature review found this typically to be a middle-aged (30-50 years old) woman, with a 3:1 female to male ratio. Still, it has been identified in individuals over a wide age range.[13,14] A systematic study of extracranial cervical carotid artery dissections suggests that the proximity to the styloid process or the hyoid bone may play an important causal role, given that a direct osteovascular mechanical contact could be established in 23% of cases and only in 7% in the nondissection control group.[15]

In the posterior circulation, a literature review of patients with bow hunter's syndrome suggested a male predominance with an overall mean age of 53 years.[16] The incidence of anomalies of atlanto-occipital fusion that potentially could cause compression of the vertebral artery varies between .5% and 1.0%.[17] However, the incidence of cerebrovascular events related to this condition remains unknown. Golf-related cervical artery dissections have been described in 8.4% of cases in a review of 190 patients.[18] Interestingly, in golfer's stroke we observed a right predominance, which might be related to the stereotypical head movement needed to perform a golf swing.[19]

Causative Mechanisms for Cerebrovascular Events in Osteovascular Conflicts

Contact Injury With or Without Demonstrated Dissection. Repeated contact between the vessel and the bone or bone-like anomaly (eg, elongated styloid process, stylohyoid ligament calcification, hyoid or occipital bone abnormality) causing arterial dissection and cerebrovascular events appears to be the most common mechanism. Both macrodissections and microdissections of the arterial wall may occur. Macrodissections, also known as subintimal dissections, show characteristic radiological signs of arterial dissection (ie, a double lumen, intimal flap, or mural haematomas). Microdissections, also described as subadventitial dissections, may show pseudoaneurysm formation without the aforementioned radiological signs[20,21] or only thrombus formation[7] (see case no. 2). The latter may be hypothetical in many cases, as it is hard to prove with current radiological methods. Such contact injuries and dissections may then lead to local thrombus formation, resulting in embolic rather than hemodynamic cerebrovascular events downstream.[22]

Transient Compression. Impingement of the vessel by the adjacent bony structures leading to hemodynamic compromise is another explanation for ischemia, which is mostly transient. This vessel compression may occur during rotational postures of the neck, for example during golfing, and may affect vessels of both the anterior and posterior circulation.[4,5,23,24] However, the posterior circulation seems to be disproportionately affected, with 88% of golf-related strokes involving the vertebro-basilar territories.[18]

Sustained Compression. Vascular structures can be chronically compressed by bony structures, even in a neutral head position, leading to hemodynamic compromise. This can result in hemodynamic cerebrovascular events (see case no. 3) or, less commonly, clot formation due to arterial blood stasis. Various structures can lead to this compression, including cervical cartilages and bones that are at the extreme end of the longitudinal and morphological distribution spectrum, as well as focal osteoarticular pathologies like atraumatic vertebral compression fractures, bone cysts, or herniated intervertebral discs. The latter might also result in the invasion of fibrocartilaginous material into the vertebral arteries leading to embolic cerebrovascular events.[25]

Discussion of Anterior Circulation Conflicts

Eagle's Neurovascular Syndrome. Eagle's vascular syndrome, also referred to as Eagle's neurovascular syndrome when cerebrovascular symptoms are present, or alternatively as stylocarotid syndrome or "self-stabbing phenomenon," is a well-known cause of cerebrovascular events due to an osteovascular conflict. The non-vascular variant of the syndrome

was described by Eagle in 1937[2] in a patient in whom an elongated styloid process was the probable cause of pharyngeal pain radiating into the middle ear. In another case, it was thought to cause mild swallowing problems. In his first publication,[2] Eagle also mentioned several older publications already describing the syndrome that would eventually bear his name. Such styloid processes are now considered to be an occasional cause of both unilateral and bilateral facial and/or neck pain, with or without underlying carotid artery impingement. Other less frequent symptoms include dysphagia, tinnitus and otalgia. In his subsequent paper, Eagle[8] reported on a total of 254 patients with this condition, separating them into two categories, namely one accompanied by throat pain irradiating into the ear and mastoid process, and a "carotid artery syndrome […] causing pressure and some obstruction of the [internal or external] carotid artery," in which "pain follows the distribution of the artery in probably 12 patients" (out of 211 patients, because he only started considering the difference between these two forms in 1940). Although he documented occasional tinnitus and inferior cranial nerve palsies in the carotid syndrome, he did not describe cerebrovascular or retinal ischemic events. He operated on more than 40 patients with the syndrome named after him (only one of them for the "carotid artery syndrome"), and did not observe a carotid pathology, stating that "it is impossible to conclude too decisively […] [an] obstruction to the arterial function and symptoms of pressure of the sympathetic nerves of the arterial walls." The first descriptions of cerebrovascular events (post-traumatic aneurysms, dissections) related to styloid-carotid conflict were published several decades later.[26,27]

In several studies exploring the characteristics of the styloid process in cervical carotid artery dissection, the length of the styloid process of affected cases was significantly increased when compared to controls.[28,29] The length of the styloid process is normally between 2 to 2.5 cm, and elongation beyond 2.5 cm may be associated with the syndrome.[21] Furthermore, a shortened distance between the styloid process and the dissected arteries was found in several case-control series,[15,29] supporting the theory that the styloid process is involved in certain patients with extracranial dissections of the internal carotid artery (see case no. 1). A jugular variant of Eagle's syndrome has also been described, causing secondary compression of the internal jugular veins, typically presenting with symptoms such as headache and tinnitus.[30,31]

Case no. 1

A male patient in his fifties with hypertension presented with weakness and sensory loss in the left upper limb lasting for about 12 h. The symptoms began after yawning and a sensation of 'something popping' in the right jaw area. These were paired with right-sided pulsatile tinnitus lasting approximatively 30 min. He reported several recurrent episodes of numbness in the left side of the neck lasting for a few minutes over the subsequent 6 months. Simultaneously, there was a progressive worsening of voluntary control of the left arm. MRI brain at an external clinic showed hyperdense lesions in the right subcortical parietal lobe. The patient was later diagnosed with left hand apraxia and suspected corticobasal ganglionic degeneration at a university hospital's movement disorder clinic. A follow-up MRI one year later showed extension of the right subcortical hemispheric lesion into the frontal and occipital regions (Figure 3A). At that time, the neuroradiologist discovered a low flow in the right carotid siphon, and additional imaging now showed a chronic right internal carotid dissection and a long styloid process impinging on the artery (Figure 3B and C). The diagnosis was recurrent ischemic stroke in the right hemisphere caused by repeated right internal carotid dissections due to Eagle's neurovascular syndrome was made. During 6 months of oral anticoagulation and 4 months of combined aspirin 100 mg and clopidogrel 75 mg treatment, the patient had several other clinical and radiological recurrences. Internal carotid stenting was performed under double antiplatelet treatment followed by an intracerebral hemorrhage on the 10th postinterventional day in the preexisting chronic ischemic lesion territory. Thereafter, progressive clinical improvement was observed through neurorehabilitation and no further cerebrovascular events or restenosis of the stented artery were reported at 2-year follow-up.

Hyoid-Carotid Conflict. Carotid artery compression by the hyoid bone has been implicated as a cause of both carotid artery stenosis and dissection. Hyoid-ICA distances have been found to be significantly shorter on the side of the carotid artery dissection as compared with non-dissection patients, and the greater horn of the hyoid bone is usually implicated.[15,32] Possible mechanisms of carotid injury mirror those discussed above. Among these, transient compression causing TIA during golf-associated rapid neck rotation has been reported. In these cases, a repetitive impingement of the ICA between the sternocleidomastoid muscle and the hyoid bone has been hypothesized.[24] Finally, strenuous coughing causing hyoid vibrations may also cause ICA injury,[7] as described in case no. 2.

Case no. 2

As described in this case report by our group,[7] "a previously healthy adult was admitted after acute onset of aphasia and right hemiplegia. The week prior to admission, strenuous cough and asthenia were reported. Neurological examination revealed global aphasia, conjugate eye deviation to the left, right homonymous hemianopsia, and flaccid right hemiplegia. Head CT on admission showed early signs of ischemia in the left middle cerebral artery (MCA) territory. CT angiography revealed a carotid T-occlusion in the left internal carotid artery, indicating a blockage at the point where the carotid artery bifurcates into the middle and anterior cerebral arteries. Additionally, non-occlusive endoluminal thrombi were observed along the posterior-medial aspect of both common carotid artery (CCA), adjacent to the hyoid bone (Figure 4A). These findings were confirmed by catheter cerebral angiography

(Figure 4B) and Duplex sonography (Figure 4C). Mycoplasma pneumonia was diagnosed based on a typical pulmonary infiltrate on chest radiography and elevation of total serum antibodies (Fc Ig and agglutinins) for *Mycoplasma pneumoniae*. Despite several attempts at endovascular aspiration of the thrombus in the left distal ICA, the occlusion could not be recanalized. Intra-arterial thrombolysis was not performed because more than 6 h had passed since the onset of symptoms. The endoluminal lesions of the CCA were no longer detected by Duplex sonography on day 12, and by MR angiography performed on day 22. MRI of the brain showed extensive infarction in the left MCA territory. The patient was still globally aphasic and hemiplegic when transferred to a rehabilitation unit 1 month after the event. In this case, vigorous coughing might have induced repeated injury of the CCA from contact with the hyoid bone leading to subsequent thrombus formation."

Discussion of Posterior Circulation Conflicts

Bow Hunter's Syndrome. The anatomical course of the vertebral artery in the intracranial-extracranial (mainly

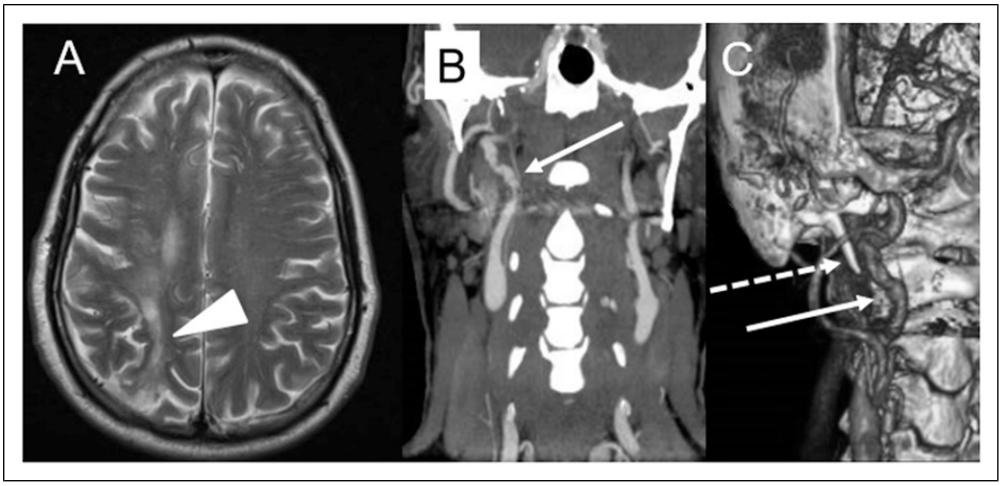

Figure 3. Case no. 1. (A) T2-weighted images 4/2013: right posterior hemispheric juxta-cortical gliosis (arrowhead), interpreted as corticobasal degeneration. (B) 7/2013 cervical CT-angiography with multiple stenoses and dilatations of the right internal carotid artery (arrow), compatible with chronic or recurrent arterial dissection. (C) Impingement of the styloid process (dotted arrow) on the internal carotid artery (arrow).

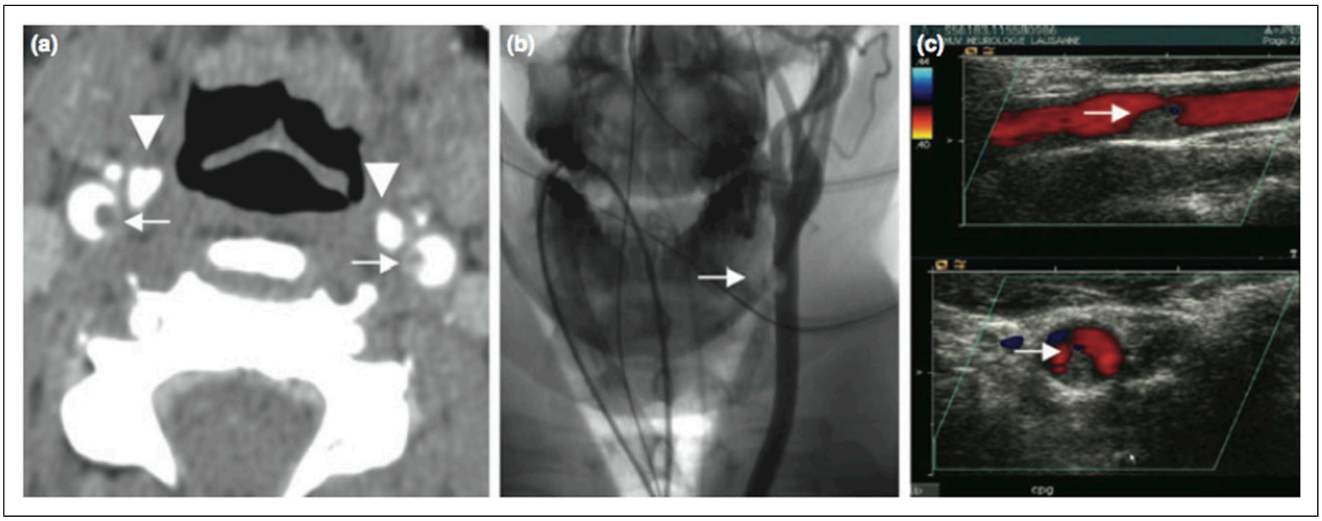

Figure 4. Case no. 2. Bilateral hyoid-carotid conflict, with the publisher's authorisation: "(A) CT angiography, showing two symmetrical endoluminal defects along the posterior-medial aspect of the common carotid arteries (CCA) (arrows), at the level of the hyoid bone (arrowheads). (B) Digital subtraction angiography, left CCA injection, anteroposterior view, confirming the presence of a spherical filling defect within the distal CCA (white arrow). (C) Duplex sonography, longitudinal (top) and axial (bottom) views, depicting the endoluminal lesions (white arrows)".

V3 segment) and extracranial-intracranial transition zone (V4 segment) renders it particularly vulnerable to compression and arterial dissections following rotational movements of the neck. A typical presentation of an osteovascular conflict is known as the "rotational vertebral artery syndrome" (or occlusion) or "bow hunter's syndrome" (BHS). Presenting features include reversible symptoms of ischemia (TIAs) of the posterior circulation during head rotation such as vertigo, syncope, diplopia, blurred vision, dizziness, tinnitus, nausea/vomiting and focal neurological deficits like ataxia, focal numbness, weakness, or paresthesia.[16] BHS was first described with this name in 1978 by Sorensen[11] as transient vertebrobasilar insufficiency due to mechanical compression or stretching of the dominant vertebral artery during contralateral neck rotation, against various bony structures in the region of the atlas or axis (C1/C2 level, Figure 2). However, the connection between head rotation and vertebrobasilar insufficiency was described as far back as 1933.[33] Given the bilaterality of vertebral arteries and the good retrograde supply of the distal vertebral artery by the other vertebral artery or the basilar artery, a unilateral compression of the vertebral artery (VA) during head rotation only rarely causes symptoms. Therefore, occlusive pathology (atheromatosis) of one VA, a clearly hypoplastic VA, or a VA ending in posterior inferior cerebellar artery is usually required to cause vertebrobasilar ischemia, as described in a recent comprehensive review of the BHS.[16] Their review of 153 cases with BHS revealed a significantly higher prevalence of BHS when compressing the left VA, possibly due to a higher incidence of left vertebral artery dominance in the general population. It should be noted that the term "bow hunter's syndrome" is not consistently used in the literature. While traditionally reserved to describe compression at the atlantoaxial level, its usage has expanded to encompass osteovascular conflicts at lower vertebral levels. The insufficient blood flow in one VA, allowing for the rotator compression of the other one to become symptomatic, may be facilitated by osteophytes in the zygoapophyseal joints or degenerative intervertebral disc disease, fibrous bands, cervical disc herniation[34] (see case no. 3), C1 or C2 instability, or even an unstable uncovertebral joint (C3-C4).[35] Another mechanism how BHS occurs is by rotation-induced VA dissection, which only concerns a minority of patients with BHS.[16]

Case no. 3

This female patient in her forties presented with acute left neck pain and headaches, followed by severe paresthesias in the left C4 distribution, but no cerebral symptoms. Angio CT showed compression and lateral displacement of the left vertebral artery on a C3-C4 level (Figure 5). Subsequent MRI showed a C3-C4 lateral disc herniation with loss of the VA lumen. The patient was treated with laser assisted microdiscectomy and had a complete recovery of symptoms.

Conflicts of the Vertebral Artery with Other Osseous Structures

Intraosseous Ganglion Cysts in the Vertebral Body. These cysts commonly occur in long bones, flat bones, small bones of hands or feet or lumbar spine around the 4th or 5th decade, without any sex preponderance. They are benign, nonneoplastic bone lesions. The pathogenesis is not known, but these lesions do not appear to be associated with osteoarthritis or

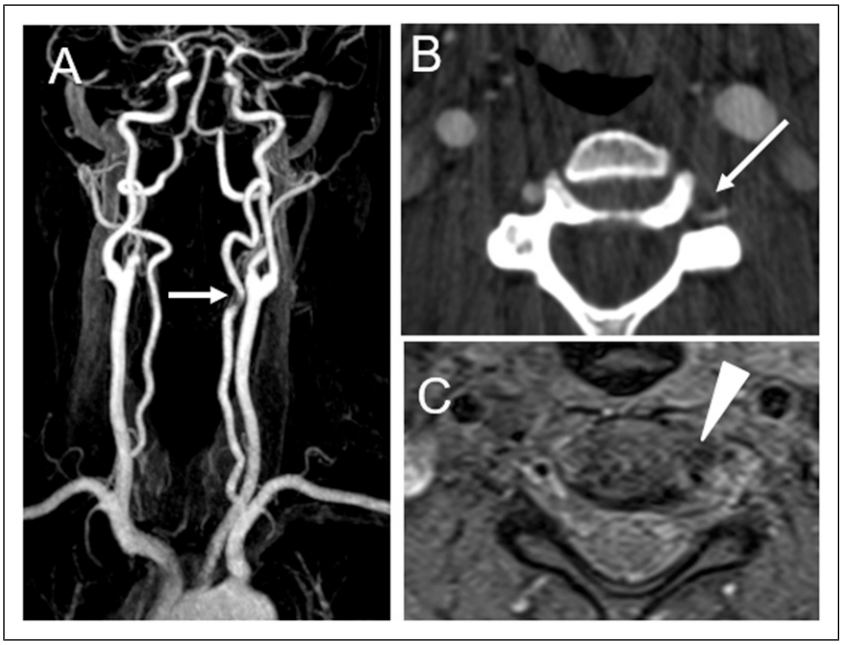

Figure 5. Case no. 3. Patient with acute left neck pain and left headaches, followed by paresthesias in the left C4 distribution, but no cerebral symptoms. (A, B) CTA showing compression and lateral displacement of the left vertebral artery on a C3-C4 level (white arrow). (C) Axial MRI T2W images showing a C3-C4 lateral disc herniation (white triangle) with loss of the VA lumen.

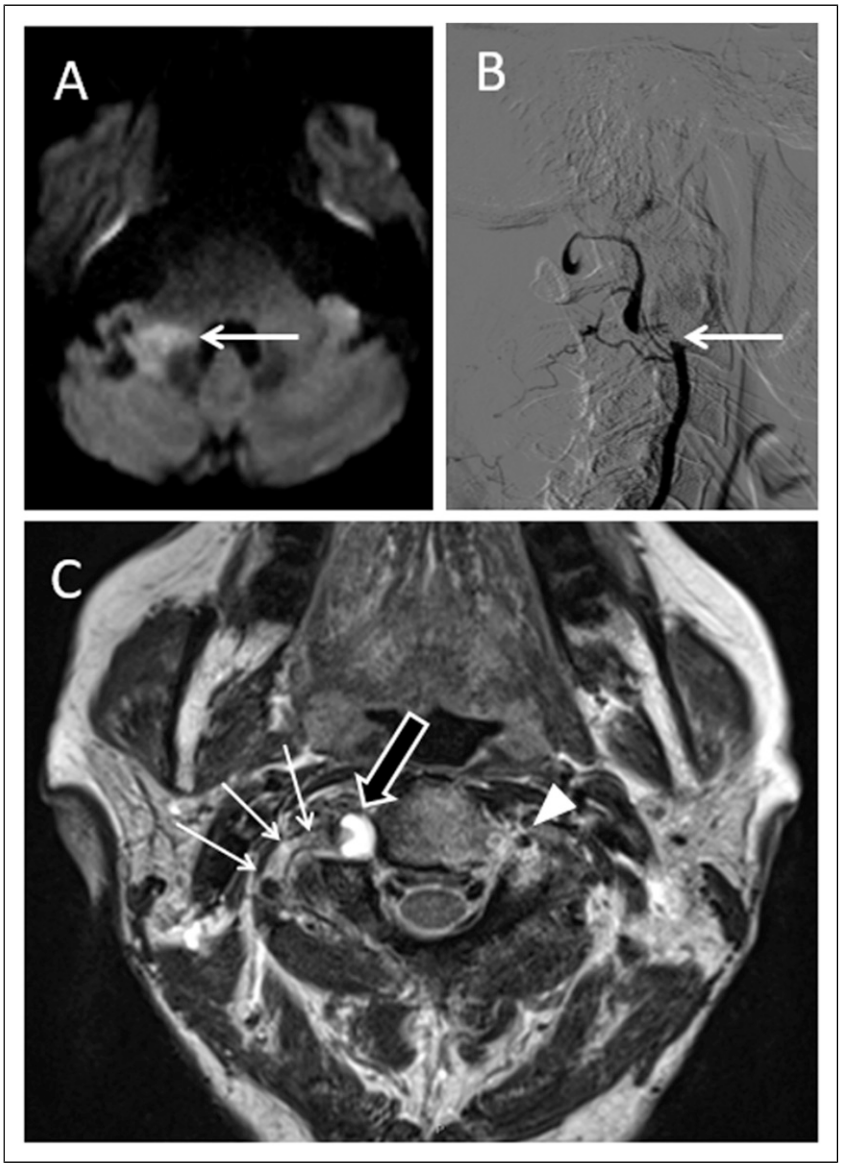

Figure 6. Case no. 4. (A) DWI-MRI showing a subacute right middle cerebellar peduncle stroke. (B) Digital subtraction angiography with V3 subocclusion. (C) T2-weighted MRI with C2 vertebral body liquid-filled ganglion cyst (black arrow), thrombus in adjacent right vertebral artery (white arrows), and flow void in the normal left vertebral artery (white arrowhead).

soft tissue ganglion.[36] Case no. 4 below describes a case of stroke from arterial compression by intraosseous ganglion cysts (Figure 6).

Case no. 4

A male patient in his seventies presented with a sudden onset dysarthria and right-sided hemiataxia. Investigations including a digital subtractions angiography revealed an intraosseous ganglion cyst arising from C2-C3 facet joints, extending into C3 vertebral body, and compressing the V3 segment of the right vertebral artery. This compression might have lead to a thrombo-embolic event explaining the observed ischemia in the right cerebellum. An MRI of the brain, conducted 3 months post-stroke, showed a preexisting intraosseous ganglion cyst with a focal right vertebral artery occlusion. The patient was treated conservatively with oral antiplatelet and did not report any recurrence of symptoms at subsequent follow-ups.

There are several case reports of posterior circulation strokes from sustained vertebral artery compression due to other osteovascular compressions, such as anomalous occipital bone process[17] or cervical spondylosis,[37] a hypertrophied uncovertebral joint (C5-C6)[38] or ossified posterior horn of the thyroid cartilage, as described in case no. 5.

Case no. 5

A female patient in her eighties experienced sudden rotatory vertigo and formed visual hallucination. MRI (Figure 7) revealed bilateral embolic inferior cerebellar strokes, which were attributed to the compression of the

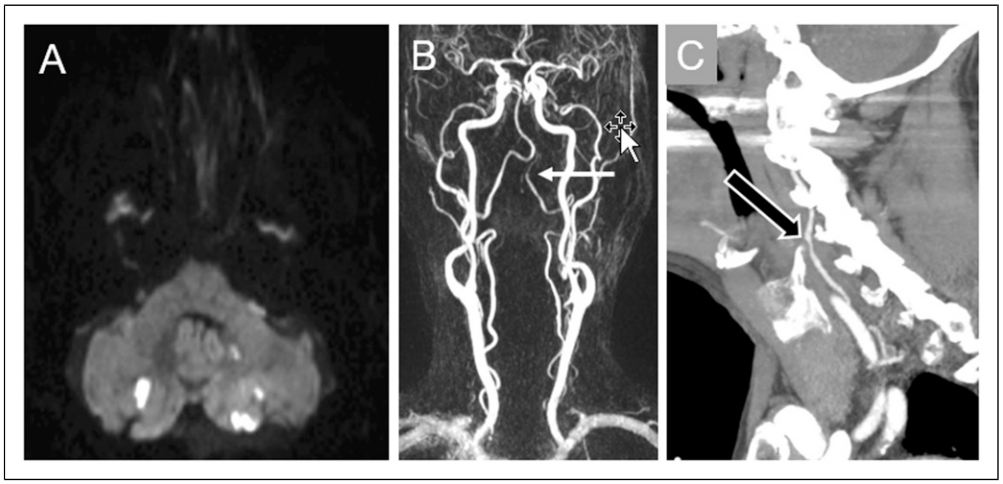

Figure 7. Case no. 5. (A) DWI-MRI showing acute bilateral strokes in the posterior inferior cerebellar artery territory. (B) Compression of the fourth segment of the left vertebral artery (arrow). (C) Direct contact between the ossified posterior horn of the thyroid cartilage (black arrow) with the left vertebral artery.

left VA by the thyroid cartilage. The patient underwent dual antiplatelet treatment for 3 months (aspirin 100 mg and clopidogrel 75 mg). Thereafter, she was prescribed lifelong aspirin. Concerning the thyroid cartilage, discussion with otolaryngology specialists led to the decision against surgical intervention due to the high risk of procedural complications. The patient remained free neurological events during the subsequent 2-year follow-up.

Osteovascular Conflicts Involving the Spinal Artery Causing Ischemic Myelopathy. Atraumatic vertebral compression fractures, usually resulting from osteoporosis and most commonly occuring in the thoraco-lumbar region, can lead to spinal strokes from sustained compression.[39] Rarely, fibrocartilaginous emboli derived from the nucleus pulposus following an acute disc herniation can also cause spinal ischemia.[40]

Treatment Approaches

Conservative management is advocated in most cases and includes mainly antiplatelet therapy. There is insufficient evidence for use of oral anticoagulation. Medical therapy of cervicofacial pain associated with Eagle's syndrome may include analgesics, anticonvulsants, antidepressants, local infiltration with steroids or long-acting anesthetic agents, and stellate ganglion block.[41]

In extracranial arterial dissections with recurrent cerebrovascular events on medical management, endovascular stenting remains an option with a Class IIb recommendation level from the American Heart Association (AHA).[42] Surgical procedures like partial styloidectomy, which can be performed by internal (intraoral) or external (extraoral) approaches, are usually considered in cases with recurrent episodes despite medical therapy. The external approach is preferred as it permits a better visualization of the styloid process and its adjacent structures and has a lesser incidence of serious complications.[43]

For patients with golfer's stroke and bow hunter's syndrome, cervical collars and certain precautions, such as the avoidance of prolonged head rotation, could be helpful.[44,45] Surgical options for bow hunter's syndrome include cervical decompression, cervical spine fusion, and endovascular stent placement within the unaffected VA to increase blood flow during head turning.[46] In cases of persistent vertebral artery compression by anomalous bony structures, when surgical removal is not possible, endovascular occlusion of the vertebral artery may also be performed. However, this can be attempted only after confirming the absence of neurological deficits or flow impairment in the posterior circulation upon temporary occlusion of the vessel by an intraprocedure balloon occlusion test.[17]

Conclusion

Osteovascular conflicts are an umbrella term we propose to encompass a group of conditions that involve conflicts between blood vessels and elongated or misshapen bones, especially but not exclusively in the head and neck region, such as Eagle's neurovascular syndrome, bow hunter's syndrome, and golfer's stroke. These conflicts can cause a range of symptoms, including headache, neck pain, and difficulty swallowing, and can also be a cause of cerebrovascular events. Increasing awareness of these conditions will help to ensure that patients receive timely and appropriate care, improving patient outcomes and quality of life.

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Author Contributions

P.C.: Conceived the manuscript, analyzed the data, drafted the initial manuscript, agreement to be accountable for all aspects of the work. I.A.M.: Acquisition and analysis of literature data, major revision of manuscript, conception of figures, creation of supplementary material, agreement to be accountable for all aspects of the work. P.M.: Acquisition and interpretation of clinical data, critically revised the manuscript, agreement to be accountable for all aspects of the work.

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