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What you need to know about: arterial cerebrovascular syndromes caused by static or dynamic musculoskeletal compression

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Abstract

Compressive syndromes of the cervical arteries caused by musculoskeletal structures include bow hunter's syndrome, beauty parlour stroke syndrome, carotid compression by the hyoid bone, carotid compression by the digastric muscle and Eagle syndrome. They are a rare but increasingly recognised group of syndromes, so a high level of suspicion is needed so the diagnosis is not missed. The diagnosis is typically based on a combination of clinical history and advanced imaging investigations. Compression of the arteries may be static (only provoked by compression) or dynamic (exaggerated by movement), and this should be considered when selecting imaging studies. Symptoms resulting from vertebrobasilar insufficiency or ischaemia of areas supplied by the internal carotid artery are caused by compression of the vertebral artery and the internal carotid artery respectively. Surgical procedures are the preferred treatment for most of these syndromes.

Key words: Carotid stenosis; Cerebral infarction; Eagle syndrome; Stroke; Vertebrobasilar insufficiency

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Introduction

Vertebral artery and vertebrobasilar insufficiency

The vertebral artery arises from the subclavian arteries, enters the transverse foramen of C6 and ascends through the transverse foramina of the upper cervical vertebrae up to C2. After that, it deviates laterally to enter the laterally placed transverse foramen of C1. It then turns behind the lateral mass and above the posterior arch of C1 to course medially and superiorly to pierce the dura at the foramen magnum. The extracranial vertebral artery is divided into three segments: V1, V2, and V3. This designation is helpful from a clinical point of view as different pathologies are associated with different segments (Standring, 2016).

When flow through basilar or vertebral arteries is disrupted, vertebrobasilar insufficiency arises. It causes ischaemia to the occipital lobes, thalamus, cerebellum and brainstem. The most common signs and symptoms of vertebrobasilar insufficiency are nausea, vomiting, vertigo, syncope, ataxia, dysarthria and visual field deficits.

The internal carotid artery and arterial compression

The internal carotid artery is one of two pairs of cervical arteries that form the circle of Willis. From its origin at the carotid bifurcation, it ascends in front of the transverse processes of the upper three cervical vertebrae to the inferior aperture of the carotid canal at the skull base. The internal carotid artery is initially superficial in the carotid triangle and then courses deeper, medial to the posterior belly of the digastric muscle. At the level of the digastric muscle, the internal carotid is crossed by the stylohyoid ligament. Above the digastric muscle, the internal carotid artery is separated from the external carotid artery by the styloid process.

One of the most important causes of ipsilateral stroke is extracranial stenosis of the internal carotid artery. Symptoms are usually caused by rupture of an atherosclerotic plaque in the internal carotid artery, leading to ischaemia in the region normally supplied by the internal carotid artery. Clinical manifestations include muscle weakness and numbness in the limbs and face, difficulty in vocalisation, vision loss and dysphasia.

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Mechanical compression of the internal carotid artery and vertebral artery by musculoskeletal neck structures constitutes a heterogenous but increasingly established group of pathological conditions. The compression is either static or dynamic, with dynamic compression significantly associated with movement (Morimoto et al, 1996; Kuether et al, 1997). Diagnosis can be problematic and is typically based clinical history with advanced imaging investigations, often supplemented by provocative manoeuvres.

This article summarises the clinical findings, imaging characteristics and treatment options for various compressive syndromes of the cervical arteries. Table 1 summarises the options for diagnosis and treatment of each syndrome.

Bow hunter's syndrome

Bow hunter's syndrome, also known as rotational vertebrobasilar insufficiency, is a vertebrobasilar insufficiency syndrome caused by mechanical dynamic compression of the vertebral artery by the surrounding structures with the head turned more than 45° to the right or left (Figure 1). The term 'bow hunter's syndrome' was proposed by Sorensen (1978), following a patient who had experienced a Wallenberg stroke during archery.

The occluded areas can be seen throughout the entire length of the vertebral artery, with the most common sites being the V2 and V3 segments (Jost and Dailey, 2015). Proximal to the skull base, temporary stenosis or obstruction of the vertebral artery is at the C1 or C2 level.

A review of the literature showed that osteophytes, fibrous bands, cervical disk herniation, C1 or C2 dysfunction, chiropractic manipulation, surgical positioning and a multitude of

Table 1. Compressive syndromes of the cervical arteries caused by musculoskeletal structures

Syndrome or disorder	Aetiology	Signs and symptoms	Suggested imaging modalities	Treatment	Outcome
Bow hunter's syndrome	Mechanical dynamic compression of the vertebral artery by the surrounding structures with the head turned more than 45° to the right or left	Dizziness, ataxia, syncope/ presyncope, nausea, headache, sensory disturbances, weakness, hemiparesis	Angiography and digital subtraction angiography	Surgical	Surgery yields good results
Beauty parlour stroke syndrome	Mechanical impingement of the vertebral artery at the atlanto-occipital junction with the hyperextension of the neck		Magnetic resonance imaging, computed tomography or magnetic resonance angiography	Surgical or conservative	Limited evidence regarding the outcome of surgical or conservative treatment
Eagle syndrome	Compression of the internal carotid artery by the elongated styloid process	Extremity weakness, aphasia/dysphasia, dizziness, sensory disturbances, visual impairment, headache, neck pain, dysphagia/ odynophagia, globus sensation, dizziness, tinnitus, ataxia	Computed tomography angiography	Surgical or conservative	Both surgical and medical treatments are reported to have good results
Carotid compression by hyoid bone	Kinking of the internal carotid artery by the hyoid bone with the presence of elongated greater horns and a low carotid bifurcation		Three-dimensional reconstructions of computed tomography angiograms	Surgical	Surgical treatment has achieved perfect outcomes
Carotid compression by digastric muscle	Mechanical stress on the internal carotid artery by the hypertrophied posterior belly of the digastric muscle		Angiographic studies	Surgical	Surgical treatment has achieved perfect outcomes

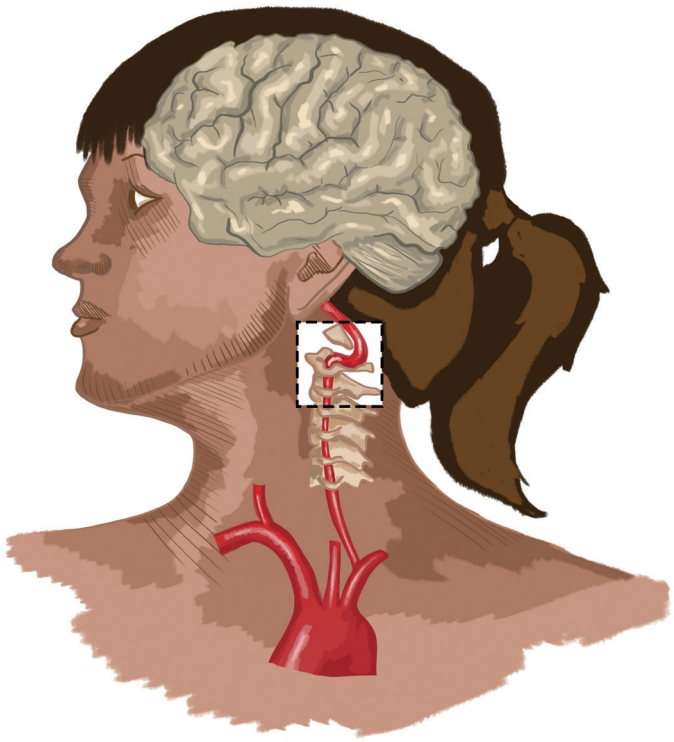

Figure 1. Bow hunter's syndrome.

physical activities have been involved in causing bow hunter's syndrome (Netuka et al, 2005; Bruneau et al, 2011).

The risk factors for bow hunter's syndrome were having a hypoplastic or an aplastic vertebral artery on one side, and a vertebral artery ending in the posterior inferior cerebellar artery. Under those circumstances, posterior circulation depends on the blood supply on one side. Compression on the dominant side causes the patient to become symptomatic more easily.

The syndrome classically presents with transient symptoms and may be relieved when the head is returned to the neutral position. From more common to less common, these symptoms include dizziness, near syncope attacks or syncopes, vertigo, visual disturbances, ataxia, weakness, hemiparesis, sensory disturbances, nausea and headaches (Darkhabani et al, 2012; Hernandez et al, 2019).

Imaging findings

Most of the diagnostic findings in imaging studies were obtained during provocative manoeuvres that reproduced patients' initial symptoms, referred to as 'dynamic' studies (Anaizi et al, 2014).

In general, computed tomography helps to visualise the bony anatomy surrounding the vertebral arteries. The most common diagnostic finding was osseous structures at various points in the course of the vertebral artery, more specifically through the V2 and V3 segments. Congenital bony anomalies were seen in paediatric patients (Jost and Dailey, 2015).

Computed tomography studies were found to be non-pathological in some reported cases, although most studies were done in the neutral position, so failed to show possible compression of the vertebral artery by bony structures with rotation. Therefore, it is worth re-evaluating these patients using other imaging modalities.

Computed tomography angiography was conducted in most cases and had diagnostic value because it could demonstrate the relationship between the vertebral arteries and surrounding structures. Bony anomalies were also detected when no prior computed tomography scans were available. Most studies were conducted in comparatively neutral and symptom-provoking positions. Compression or stenosis of the vertebral arteries was

shown in relatively mild cases, while complete occlusion was shown in more severe cases. Anatomical risk factors such as a hypoplastic vertebral artery or a vertebral artery terminating in the posterior inferior cerebellar artery or other arteries and not contributing to the basilar artery could be detected in some patients.

In the radiological diagnosis of bow hunter's syndrome, either conventional cerebral angiography or digital subtraction angiography is the gold standard (Anaizi et al, 2014; Buch et al, 2017). During either sort of angiography, imaging findings correlated with patients' experience of symptoms. In some cases, the vertebral arteries were patent in the neutral position. They showed different degrees of extrinsic compression with head rotation, including complete occlusion. In contrast, in other cases there was a degree of pre-existing stenosis, which increased or resulted in a complete obstruction.

Treatment

Surgery is the treatment of choice in terms of results and safety, with the primary objective being the reduction of the mechanical compression of the vertebral artery on neck rotation. Imaging demonstrates restored vascular systems postoperatively.

Medical management with antithrombotic therapy, cervical collars and restriction of excessive neck movement were included in conservative care. While no studies specifically compared conservative approach and surgical treatment, most authors have observed that conservative therapy is typically unsuccessful (Safain et al, 2014).

Beauty parlour stroke syndrome

Beauty parlour stroke syndrome was first described by Weintraub (1993), named after cases of vertebrobasilar insufficiency symptoms observed after patients had visited a hairdresser's salon. Mechanical impingement with the neck in hyperextension causes a reduction in the blood flow of the vertebral artery at the atlanto-occipital junction (Figure 2).

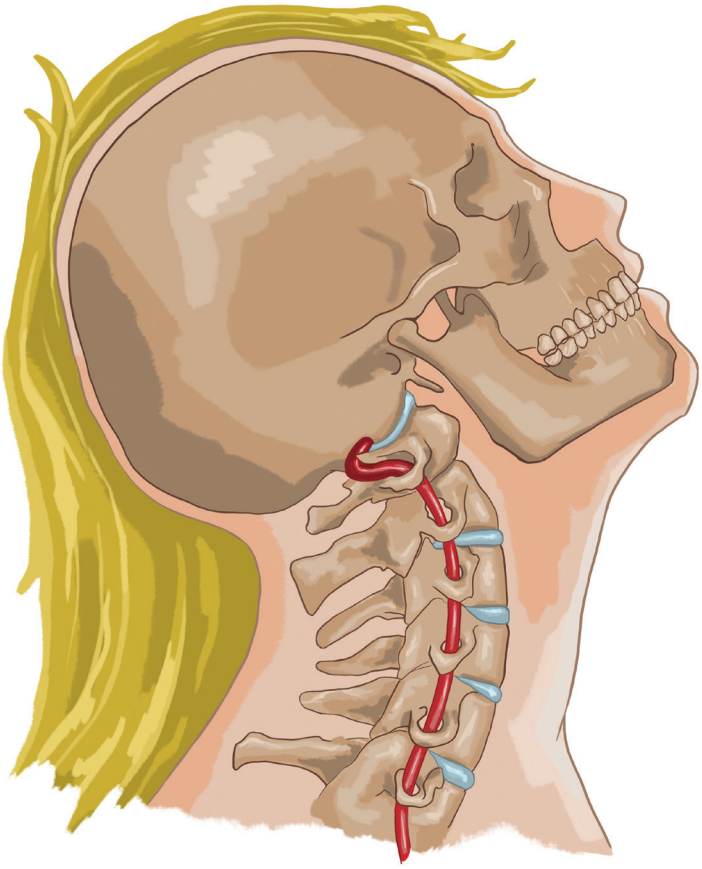

Figure 2. Beauty parlour stroke syndrome.

Hypoplastic or aplastic vertebral artery on one side and hypoplastic or aplastic posterior communicating arteries either unilaterally or bilaterally could be anatomical predispositions for developing beauty parlour stroke syndrome.

The syndrome presents with symptoms of stroke in patients following a visit to a hairdresser's salon or other situations that place the neck in a hyperextended position. One reported case included a patient with beauty parlour stroke syndrome as a result of a perioperative and intraoperative neck extension posture (Zangbar et al, 2015).

From more common to less common, symptoms include dizziness, near syncope attacks or syncopes, sensory disturbances, extremity weakness, nausea, vomiting, tinnitus, vertigo, and hemiparesis (Kameda et al, 2018).

Imaging findings

Magnetic resonance imaging and computed tomography showed cerebellar or posterior cerebral infarctions caused by compression of the vertebral artery, while magnetic resonance imaging studies have shown earlier infarctions related to the condition.

Magnetic resonance angiography has shown either narrowing of the vertebral artery lumen or compression at the atlanto-occipital junction with neck extension.

Treatment

There is limited evidence regarding the outcome of surgical or conservative treatment. Kameda et al (2018) reported that a patient had undergone surgery involving removal of the osteophyte and inferior rim osteotomy of the C1 groove without any perioperative complications. The patient was symptom-free during the 48-month follow-up period. Endo et al (2000) treated their patient conservatively which improved the patient's experience of symptoms.

Eagle syndrome

Piertro Marchetti was the first to associate clinical symptoms with an elongated styloid process in 1652, although the syndrome was defined by Watt W Eagle in the 1940s (Eagle, 1958). Eagle described the characteristic symptoms in patients with an elongated styloid process, calcified stylohyoid ligament or both.

The syndrome is divided into two subtypes: classic and carotid. Neck pain, foreign body sensation in the neck, odynophagia and dysphagia are more commonly seen in the classic subtype, while the carotid subtype is associated with symptoms related to internal carotid artery compression.

The styloid process emerges from the skull base anteromedially to the mastoid process, lateral to the jugular foramen and posterolaterally to the carotid canal orifice. This relationship to the vascular openings is important in the pathology of the syndrome. The longer the styloid process, the greater the chance of having a close relationship with the carotid artery (Figure 3). The same mechanism applies to the stylohyoid ligament with its increased calcification (Fusco et al, 2012).

From clinically more to less common, symptoms include aphasia/dysphasia, extremity weakness, dizziness, sensory disturbances, visual impairment, hemiparesis, headache, neck pain, facial droop, vertigo, dysphagia/odynophagia, foreign body sensation in the neck, tinnitus and ataxia.

Imaging findings

Computed tomography angiography has been the gold standard diagnostic radiological method for Eagle syndrome – this has typically been able to identify elongated styloid processes, and has also less commonly revealed calcified stylohyoid ligaments. These pathologies have been identified simultaneously and bilaterally. Since the elongated styloid processes were in close apposition to the lateral portion of the internal carotid artery, kinking of the artery more commonly resulted in dissections and pseudoaneurysms, fusiform aneurysms and saccular aneurysms. When imaging studies were done with positional changes, movement of the neck was associated with decreased diameter of the arterial lumen in patients who had suggested symptomatology.

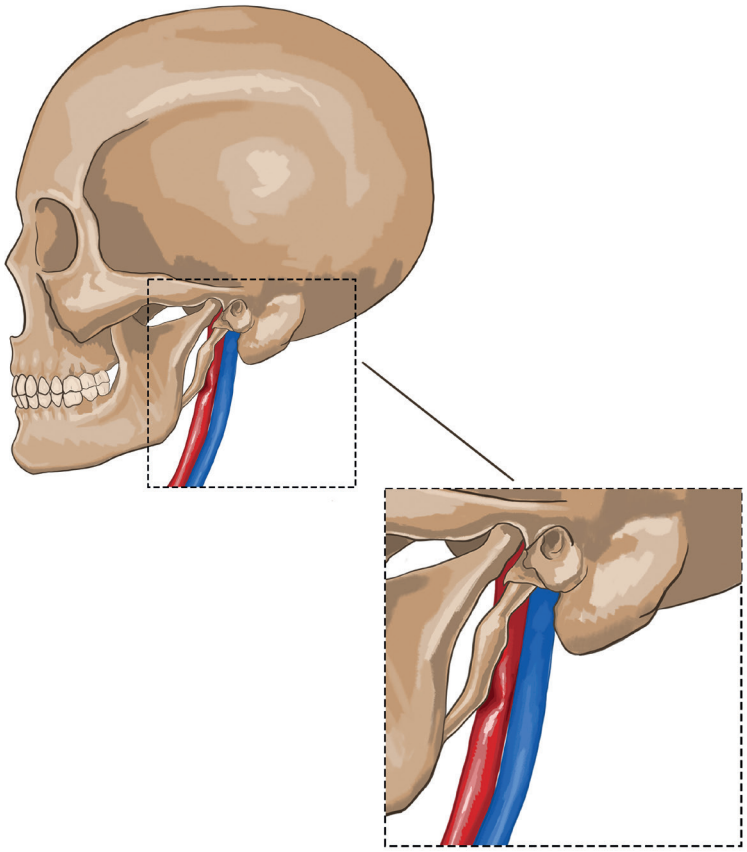

Figure 3. Eagle syndrome.

Magnetic resonance imaging demonstrated cerebral ischaemic signals and infarctions in a majority of patients when the arterial disease caused transient or chronic ischaemia.

A close relationship between localisation of the indentation of the internal carotid artery and elongated styloid process or crossing of the stylohyoid ligament has been demonstrated by arteriographic studies such as angiography and digital subtraction angiography.

Treatment

Evidence from several studies indicates that endovascular interventions (stent placement), surgery (resection of elongated styloid processes or stylohyoid ligaments) and medical care can all be performed. More patients were treated medically than for other syndromes discussed in this article.

Carotid stenosis or compression by the hyoid bone

Hyoid bone compression is a rare cause of compression of the internal carotid artery (Figure 4), because it requires both an elongated greater horn of the hyoid bone and a low carotid bifurcation (McMurtry and Yahr, 1966; Martinelli et al, 2019).

The greater horn causes repetitive traumas with subsequent vessel wall injury, stenosis, pseudoaneurysm, dissection, thrombosis or mechanical stress resulting in endothelial damage and subsequent atheromatic plaque.

Symptoms that occur after repeated trauma include extremity weakness, sensory disturbances, hemiparesis, dysphasia and dizziness.

Imaging findings

Three-dimensional reconstruction of computed tomography angiograms is reported as the best way to visualise the anatomopathological correlation, demonstrating the relationship between the greater horn of the hyoid bone and the compressed internal carotid artery.

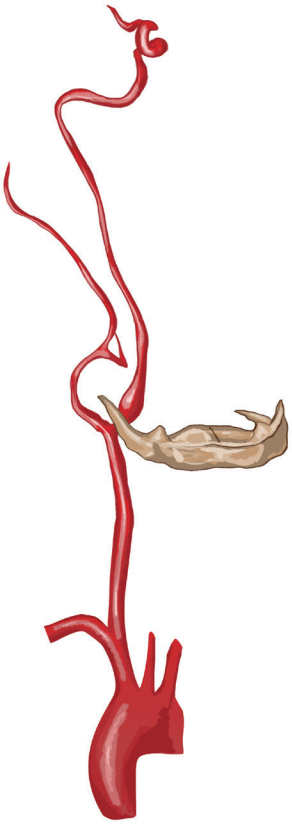

Figure 4. Carotid compression by the hyoid bone.

Treatment

Surgical approaches are mainly carotid endarterectomy and resection of the greater horn of the hyoid bone. Surgical treatment has achieved perfect outcomes from both safety and outcome perspectives.

Carotid stenosis or compression by the digastric muscle

Hypertrophied posterior belly of the digastric muscle puts the internal carotid artery under mechanical stress and patients present with hemiparetic symptoms.

Imaging findings

Angiographic studies show complete occlusion of the internal carotid arteries. Dynamic compressions were reported by Etheredge et al (1984) and static compressions were reported by McMurtry and Yahr (1966).

Treatment

Surgical treatment of patients with resection of the hypertrophic posterior belly of the digastric muscle has resulted in the resolution of symptoms without any complications.

Discussion

Thromboembolism, large artery atherosclerosis, penetrating small artery disease and arterial dissection are the most common causes of vertebrobasilar insufficiency (Moulin et al, 2000; Caplan et al, 2004), although extrinsic compression by musculoskeletal structures has also been described. Drug abuse, coagulopathies, fibromuscular dysplasia and migraine have also been reported as rarer causes. Internal carotid artery stenosis accounts for about half of large vessel cerebrovascular disease, which accounts for about 15–20% of ischaemic stroke (Flaherty et al, 2013). The most common underlying cause is atherosclerosis (Chaturvedi

Key points

- Vertebrobasilar insufficiency is defined as transitory ischaemia of the posterior circulation.
- Even if static imaging does not show any significant compression, dynamic imaging studies should be performed when the clinical findings and patient history suggest dynamic compression syndromes.
- Advanced imaging investigations are supplemented by provocative manoeuvres to diagnose these syndromes.
- Surgery has been the preferred treatment option in most of the reported cases.
- Although the underlying causes are variable, clinical findings in each syndrome may be similar.

and Bhattacharya, 2014). When a patient does not have common atherosclerotic risk factors such as older age, cigarette smoking, high blood pressure and high total cholesterol levels, extracranial stenosis of the internal carotid artery by surrounding structures should be considered.

Conclusions

Compressive syndromes of the cervical arteries are becoming more widely recognised. The keys to successful diagnosis are patient history and use of appropriate diagnostic modalities. These conditions should be kept in mind in the differential diagnosis for cerebrovascular events. As far as the authors are aware, there has been no research comparing treatment options. However, as the syndromes are rare, statistical analysis of the outcome of different treatment options would not be feasible. Further research is needed to understand the underlying causes of compressive syndromes.

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Conflicts of interest

The authors declare that there are no conflicts of interest.

References

Anaizi AN, Sayah A, Berkowitz F, McGrail K. Bow hunter's syndrome: the use of dynamic magnetic resonance angiography and intraoperative fluorescent angiography. J Neurosurg Spine. 2014;20(1):71–74. https://doi.org/10.3171/2013.9.SPINE121019

Curriculum checklist

This article addresses the following requirements from the general internal medicine curriculum:

- Managing an acute specialty-related take
- Managing patients in an outpatient clinic, ambulatory or community setting, including management of long-term conditions
- Managing medical problems in patients in other specialties and special cases.

Bruneau M, De Witte O, Yasuda M, George B. Compression by the vertebral artery. Pathology and surgery around the vertebral artery. Paris: Springer; 2011:285–295

Buch VP, Madsen PJ, Vaughan KA et al. Rotational vertebrobasilar insufficiency due to compression of a persistent first intersegmental vertebral artery variant: case report. J Neurosurg Spine. 2017;26(2):199–202. https://doi.org/10.3171/2016.7.SPINE163

Caplan LR, Wityk RJ, Glass TA et al. New England medical center posterior circulation registry. Ann Neurol. 2004;56(3):389–398. https://doi.org/10.1002/ana.20204

Chaturvedi S, Bhattacharya P. Large artery atherosclerosis: carotid stenosis, vertebral artery disease, and intracranial atherosclerosis. Continuum Minneap Minn. 2014;20(2):323–334. https://doi.org/10.1212/01.CON.0000446104.90043.a5

Darkhabani MZ, Thompson MC, Lazzaro MA, Taqi MA, Zaidat OO. Vertebral artery stenting for the treatment of bow hunter's syndrome: report of 4 cases. J Stroke Cerebrovasc Dis. 2012;21(8):908.e1–e5. https://doi.org/10.1016/j.jstrokecerebrovasdis.2011.09.006

Eagle WW. Elongated styloid process: symptoms and treatment. Arch Otolaryngol Head Neck Surg. 1958;67(2):172–176. https://doi.org/10.1001/archotol.1958.00730010178007

Endo K, Ichimaru K, Shimura H, Imakiire A. Cervical vertigo after hair shampoo treatment at a hairdressing salon: a case report. Spine. 2000;25(5):632. https://doi.org/10.1097/00007632-200003010-00017

Etheredge SN, Effeney DJ, Ehrenfeld WK. Symptomatic extrinsic compression of the cervical carotid artery. Arch Neurol. 1984;41(6):672–673. https://doi.org/10.1001/archneur.1984.04210080084020

Flaherty ML, Kissela B, Khoury JC et al. Carotid artery stenosis as a cause of stroke. Neuroepidemiology. 2013;40(1):36–41. https://doi.org/10.1159/000341410

Fusco DJ, Asteraki S, Spetzler RF. Eagle's syndrome: embryology, anatomy, and clinical management. Acta Neurochir. 2012;154(7):1119–1126. https://doi.org/10.1007/s00701-012-1385-2

Hernandez RN, Wipplinger C, Navarro-Ramirez R et al. Bow hunter syndrome with associated pseudoaneurysm. World Neurosurg. 2019;122:53–57. https://doi.org/10.1016/j.wneu.2018.10.102

Jost GF, Dailey AT. Bow hunter's syndrome revisited: 2 new cases and literature review of 124 cases. Neurosurg Foc. 2015;38(4):E7. https://doi.org/10.3171/2015.1.FOCUS14791

Kameda T, Otani K, Tamura T, Konno S. Beauty parlor stroke syndrome due to a bone fragment from an osteophyte of the atlas: case report. J Neurosurg Spine. 2018;28(4):389–394. https://doi.org/10.3171/2017.7.SPINE17226

Kuether TA, Nesbit GM, Clark WM, Barnwell SL. Rotational vertebral artery occlusion: a mechanism of vertebrobasilar insufficiency. Neurosurgery. 1997;41(2):427–433. https://doi.org/10.1097/00006123-199708000-00019

Martinelli O, Fresilli M, Jabbour J, Di Girolamo A, Irace L. Internal carotid stenosis associated with compression by hyoid bone. Ann Vasc Surg. 2019;58:379.e1–379.e3. https://doi.org/10.1016/j.avsg.2018.09.043

McMurtry JG, Yahr MD. Extracranial carotid artery occlusion by an anomalous digastric muscle. J Neurosurg. 1966;24(1):108–110. https://doi.org/10.3171/jns.1966.24.1.0108

Morimoto T, Kaido T, Uchiyama Y et al. Rotational obstruction of nondominant vertebral artery and ischemia. case report. J Neurosurg. 1996;85(3):507–509. https://doi.org/10.3171/jns.1996.85.3.0507

Moulin T, Tatu L, Vuillier F et al. Role of a stroke data bank in evaluating cerebral infarction subtypes: patterns and outcome of 1,776 consecutive patients from the Besançon stroke registry. Cerebrovasc Dis. 2000;10(4):261–271. https://doi.org/10.1159/000016068

Netuka D, Beneš V, Mikulík R, Kuba R. Symptomatic rotational occlusion of the vertebral artery - case report and review of the literature. Zentralbl Neurochir. 2005;66(4):217–222. https://doi.org/10.1055/s-2005-836600

Safain MG, Talan J, Malek AM, Hwang SW. Spontaneous atraumatic vertebral artery occlusion due to physiological cervical extension: case report. J Neurosurg Spine. 2014;20(3):278–282. https://doi.org/10.3171/2013.12.SPINE13653

Sorensen BF. Bow hunter's stroke. Neurosurgery. 1978;2(3):259–261. https://doi.org/10.1227/00006123-197805000-00013

Standring S (ed). Gray's anatomy: the anatomical basis of clinical practice. 41st edn. New York: Elsevier Limited; 2016

Weintraub MI. Beauty parlor stroke syndrome: report of five cases. JAMA. 1993;269(16):2085. https://doi.org/10.1001/jama.1993.03500160051022

Zangbar B, Pandit V, Rhee P et al. Beauty parlor stroke syndrome: a rare entity in a trauma patient. Am Surg. 2015;81(3):120–122. https://doi.org/10.1177/000313481508100313