Headache and Aneurysm

O-Ki Kwon, MD, PhD

KEYWORDS

• Aneurysm • Headache • Pathophysiology • Trigeminal nerves • Upper spinal nerves

KEY POINTS

- No pain from the aneurysm develops without pain nerves.
- The pain nerves involved in headache are the trigeminal nerve and high cervical spinal nerves (C1, 2, and 3).
- The pain nerves for intradural structures are present on blood vessel (mainly artery) walls.
- Various types of stimulation to the arterial walls producing pain include compression, distension, traction, chemicals, and simple touching.
- With the basic knowledge, the mechanism of the headache from cerebral aneurysms can be better understood.

INTRODUCTION

Headache requires a wider definition than just a pain in the head.[1,2] In this article, a narrow concept of headache (a pain) is used. It is a basic concept that headache develops from peripheral pain nerve fibers. No headache develops without pain nerves. Brain does not have pain nerve fibers.[3] Therefore, as seen in awake brain surgery, direct surgical manipulation of the brain does not produce pain. Structures that have pain fibers are dura (pachymeninges), major arteries, and veins. The entire dura does not sense pain. Pain is perceived only in dural parts with pain nerve fibers. Likewise, the other source of intracranial pain-cerebral vessels can also produce pain only when they have pain fibers.

CONTENT

Nerves that innervate intracranial vessels are autonomic (sympathetic and parasympathetic) and sensory nerves. Direct innervation from fibers of the brain may be present[4] but its function is not yet fully determined. The pain nerves involved in headache are trigeminal nerve (and its 3 divisions) and high cervical spinal nerves (dorsal [sensory] roots of C1, 2, and 3). The sensory trigeminal nerve fibers terminate in caudal and interpolar divisions of the spinal trigeminal nucleus. The sensory fibers of cervical spinal nerves terminate in the dorsal horns of the spinal cord. Small fibers of the general somatic afferent system of cranial nerve VII (through parts of the nervus intermedius), IX (tympanic nerve, nerve of Jacobson), and X (auricular nerve, Arnold nerve) also have nociception but the sensing area is limited to ear structures. The cell bodies that receive these pain senses are also in the trigeminal nucleus. The central nervous system itself contributes to relation, perception, and modulation of the headache. Centrally produced pain such as thalamic pain, in which no activation of peripheral nerve fibers is necessary for generating pain, exists but it is not a typical form of headache. The brain stem itself may also have headache-producing structures,[5,6] but it is not thought to be a mainstay mechanism of headache development. Stimulation or modulation of the trigeminal nucleus and its connections may be a pain-producing mechanism in this situation.

The exact mechanisms that cause major primary headaches like migraines, tension headaches, and cluster headaches are still not clearly understood. Proposed theories are divided into peripheral origin (arteries and accompanying trigeminal or cervical spinal nerves) and central

Department of Neurosurgery, Seoul National University Bundang Hospital, Gumiro 173 Beongil, Bundanggu, Seongnamsi, Gyeonggido 463-707, South Korea
E-mail address: meurokwonoki@gmail.com

Neuroimag Clin N Am ■ (2019) ■–■
https://doi.org/10.1016/j.nic.2019.01.004

(brain) origin. Unlike primary headaches, the headache associated with aneurysms has a definite peripheral origin, the aneurysm. In general, cerebral aneurysms arise from major cerebral arteries on which pain nerve fibers are present. These nerves are branches from the trigeminal nerve (almost all from V1, the ophthalmic division) and the upper 3 cervical spinal nerves.

All of the 3 trigeminal divisions have sensory nerves. Their main sensing structures are dura. The ophthalmic division of the trigeminal nerve (V1) innervates the anterior cranial fossa and falx through the anterior and posterior ethmoidal nerves. The tentorial nerve is the first branch of the ophthalmic nerve. It takes a recurrent course to innervate the superior surface of the tentorium and posterior falx. The maxillary nerve (V2) innervates part of the dura of the anterior and middle cranial fossa. The mandibular nerve (V3) also innervates part of the anterior and middle cranial fossa dura.

In general, these nerves use vessels (mainly arteries) to reach dura (via meningeal arteries) and intracranial structures (via internal carotid artery). Sympathetic nerves also use a similar pathway.

Supratentorial intradural structures are innervated mainly by branches of V1. It enters the intradural space along with the ICA. It begins from cavernous sinus and dura around the ICA level. On the surface of the ICA, the fibers go along into the middle cerebral artery (MCA) and anterior cerebral artery (ACA) as with the ICA course. The exact level at which they end seems to vary.

Posterior cranial fossa dura and intradural structures, mainly arteries, are innervated by C1, C2, and C3. The cervical spinal nerve 1 (C1) is unique. Unlike other spinal nerves that have both motor and sensory fibers, only about half of C1 has sensory fibers.[7] It enters the skull with the hypoglossal nerve.

How far and how many these pain nerves reach on the dura seem to vary. Distribution of pain sense is not uniform over the dura. A study of awake brain surgery showed that the intensity of pain differed by location. The intensity of the pain caused by touching the temporal part of the skull base dura was 7.9 out of 10 (range, 0–10), falx and tentorium was 4.2 (range, 0–9), and perisylvian leptomeninges was 6.6 (range, 0–10).[8] Convexity dura has less pain. Stimulation of some parts of the dura does not induce pain. Likewise, large areas of posterior fossa dura over rostral and lateral convexities of the cerebellum were insensitive to pain by stimulation.[9]

The proximal portions of arteries have more abundant sensory innervation than distal. Proximal arterial segments near the circle of Willis were exquisitely sensitive to stimuli but were progressively less sensitive over the convexity.[10,11] In awake brain surgery, neurosurgeons confirmed that pain-sensitive structures are the dura of the skull base, falx cerebri, and the leptomeninges of the sylvian fissure and neighboring sulci.[9] Some sensory fibers reach opercular or proximal cortical segments of MCA.

Several types of stimulation on cerebral arteries produce pain, including dilatation (distension), traction, chemical, thermal, and electrical stimulation.[10–12] Among these, dilation of intracranial and extracranial arteries has been studied extensively.[12,13] Balloon inflation of cerebral arteries caused headaches, which disappeared immediately with balloon deflation.[12] Stent-assisted coiling caused headache more than simple coiling.[14,15]

Simpler manipulation like just touching of the arteries can also produce pain. Experience from awake brain surgery has provided information about pain-sensitive structures.[8] In most cases, just touching causes pain. The pain from these structures was described as sharp, acute, intense, and brief. When the touching stopped, the pain disappeared. The pain from large aneurysms or tumors could be explained by just touching (compression with low pressure) of neighboring pain-sensitive structures.

Displacement of the ICA also produces pain.[16] Frequent development of frontal and periorbital pain after stent-assisted coiling of ICA aneurysms can be explained by ICA stretching and displacement.[17]

As with other sensory neurons, activation of pain fibers is influenced by temporal and spatial summation. The number of nociceptive receptors and neurons can be related with pain strength.[18,19] Low-intensity stimulation for a short duration may not produce pain. Likewise, slowly forming aneurysms may not induce headache. Acute tearing dissection[20] or acute formation of a daughter sac or a pseudoaneurysm from a preexisting aneurysm could produce sudden severe headache. This headache may disappear if there is no further acute change or it may persist if other subsequent arterial changes, such as rupture, that newly stimulate pain nerve fibers on the arterial wall occur.

In addition, neural adaptation could affect pain production and its intensity, so the intensity of pain caused by long-lasting low-grade stimulation may decrease over time despite pain sensation, especially via C-type nociceptive fibers, adapts slowly. New headaches can develop after coiling or stent-assisted coiling, probably caused by stretching of arterial walls, and generally are of

low intensity and spontaneously disappeared days or weeks later.[14,15]

Patients have pulsatile or throbbing headache with increased intracranial pressure. Increased pulsatility of the whole dura can be a mechanism for headache. Increased arterial pulsatility can also contribute to headache.[21–23] Likewise, the pulsatile motion of aneurysms may be a source of headache. Conditions with high pulsatile movement, such as large size and thin wall, may be factors of headache development. The pulsatile motion can be reduced or removed by treatment. Disappearance of headache after coil packing of aneurysmal sacs can be explained by this mechanism.[21] Many reports have showed that headache had substantial reductions after treatment, including surgery and coiling.[24–29]

Acute severe stimulation, such as abrupt arterial tearing or distension of major cerebral arteries, causes a sudden intense headache such as thunderclap headache. Vasospasm, also produces severe pain from the uncontrollable smooth muscle contraction and compensatory dilatation. The same one can be the mechanisms of pain associated with reversible cerebral vasoconstriction syndrome (RCVS).

Long-lasting stimulation with constant intensity could have diverse results. Pain fiber activation may decrease with adaptation, or, in some situations, temporal summation can provoke more severe headache. Prolonged intensive headache from subarachnoid hemorrhage or bacterial meningitis or bearable headache from chronic increase of intracranial pressure can be examples.

When the meningeal artery is stimulated, the pain location varies depending on the innervating nerves. All 3 divisions of the trigeminal nerve take part in dural innervation.[30] But for intracranial arteries, V1 is the main innervating nerve. In general, pain from stimulating intracranial arteries is referred to the ipsilateral temporal, retrorbital and frontal regions. The pain caused by stimulation of the superior surface of the tentorium, torcular, or straight sinus is also referred to the ipsilateral forehead and periorbital region because this part is innervated by the tentorial nerve from V1.[30]

In a study on balloon inflation of cerebral arteries, balloon inflation produced pain and its location was reproducible. Inflation in the proximal MCA stem produced pain primarily in the ipsilateral temple. Ballooning in the middle of the MCA stem produced pain referred primarily retro-orbitally, and inflation in the distal MCA stem produced pain referred primarily to the forehead.[12] These findings show that there are patterns of pain fiber distribution. Anatomically it is also related to localization of cells of the trigeminal nucleus in the brain stem. Some patients did not perceive any headache with the balloon inflation, which indicates that not all major arteries, or not all parts of major arteries, have pain fibers, and the number of pain receptors may vary by location and among individuals. Stimulation of the superficial temporal and middle meningeal arteries and the large intracranial venous channels can also produce similar referred pain.[10,11,31,32] Therefore, it is natural to think that the precise localization of arterial pain cannot be determined from the location of the pain. One obvious point is a tendency for carotid headaches to be located anteriorly and vertebrobasilar-associated headaches to be located posteriorly.[10,33]

Likewise, pain develops at the posterior neck when the posterior fossa dura is stimulated. It is innervated by the upper 3 cervical spinal nerves. But trigeminal fibers can reach the rostral part of the basilar artery via the posterior communicating artery. The pain from the rostral part of the basilar artery therefore can be referred to the orbital, retro-orbital, and frontal areas. Of interest, sensory signal from the C1 spinal nerve may enter the trigeminal caudal neucleus.[34,35] The nucleus is located from the medulla to the C2 level of the spinal cord, where it continues with the dorsal horn of the spinal cord. The spinal trigeminal tract is also analogous to the Lissauer tract of the spinal cord. One study showed that C1 stimulation produced periorbital and frontal pain.[36] Simultaneous sensitization or interaction between neurons may be the mechanism of common occurrence of both headache and neck pain.[37] Accordingly, pain from the vertebrobasilar system can be perceived as orbital–retro-orbital and frontal pain.[37] Cases of frontal or retro-orbital headaches associated with vertebrobasilar dissection[38] and posterior fossa tumor[39] have been reported. Similarly, sensory fibers from the upper cervical spinal nerve can also reach the distal ICA and neighboring arteries, so pain from the anterior circulation system may also be felt in the posterior neck.[37] In addition, midline crossing fibers that innervate the contralateral side have been noted in both trigeminal and spinal nerves.[40]

Patients with giant aneurysms have plausible reasons for a high incidence of headaches. They have more chance to compress adjacent pain-sensitive structures, including the dura and the artery.[41,42] Traction, compression, and displacement of adjacent arteries produce pain. Pulsatile motion of a large aneurysm sac, thrombus formation within the sac, and extravasation of the thrombin followed by inflammation can also be factors.

Periorbital pain with ptosis from an aneurysm compressing the third nerve is a well-known symptom. Posterior communicating artery aneurysm is the most common cause.[21,43,44] The occulomotor nerve has parasympathetic fibers and motor fibers for eye movement. When a posterior communicating artery aneurysm compresses the third nerve from above, ptosis develops because motor fibers for the levator palpebra muscle are located superficially. The third nerve does not have sensory function, so theoretically pain should not be a symptom unless the pain fibers on the ICA wall is stimulated. However, studies have shown that fibers from V1 of the trigeminal nerve may join and travel with the third nerve at the level of the lateral wall of the cavernous sinus, which accounts for the periorbital pain with third nerve compression.[42]

Some patients with unruptured intracranial aneurysms present with trigeminal neuralgia. Compression or distortion of trigeminal nerves at various levels could be a mechanism. Many aneurysm locations have been reported, including the ICA, posterior cerebral artery, superior cerebellar artery, anterior and posterior inferior cerebellar artery, and basilar artery.[45–47] Typical trigeminal neuralgia involves the V2 and V3 divisions.[48] However, in cases of posterior communicating artery aneurysm associated with trigeminal neuralgia, it mainly occurs in the V1 and V2 area. It is explained by the anatomic location of V1 fibers, which are distributed superiorly and medially in the trigeminal nerve and posterior communicating artery compression of the cavernous sinus from above.[48,49]

The hallmark of subarachnoid hemorrhage (SAH) is a sudden, severe headache. It is often described as head explosion or thunderclap. It is very intense and severe, so many patients describe it as unbearable, or the worst headache of their lives. Typically, it peaks within minutes and last hours or days.[50] In a report, about 50% of the headaches reached a maximum instantaneously. In the others, it took 1 to 5 minutes or longer.[51] In terms of pain location, 70% of the headaches are bilateral. In cases of bilateral headache, generalized headache is most common in 66%. In the case of lateralized headache, frontal and frontoparietal locations are common.[50] Given that a frequent location of ruptured aneurysms is the circle of Willis (anterior circulation) and the subarachnoid space is an open space with bilateral communication, these findings are expected. Meningeal irritation signs, such as nuchal rigidity and the Kernig sign, do not clearly develop at the onset. They take 2 or 3 days to become apparent[52] because development of meningeal inflammation from blood degradation products takes time.

Sentinel headache, which is similar to that of subarachnoid hemorrhage, characterized by sudden severe headache, has been thought to be caused by a small amount of hemorrhage from the aneurysm. When a patient has SAH and the patient has a history of sudden severe headache hours, days, or months before, physicians can consider the previous one to be a sentinel headache. If cerebral aneurysms that will rupture soon could be detected and treated before catastrophic SAH using this warning sign, many patients could be saved.[53] However, real causes of the headaches could be various, including migraine, vasospasm, inflammation, or just tension headache. Other mechanisms, such as acute expansion of the aneurysm, acute formation of a daughter sac, and hemorrhage into the aneurysm wall, can also be considered to be aneurysm-associated sudden severe headache.

Perimesencephalic SAH, a distinct form of SAH with a benign clinical course, is characterized by a small amount of SAH around the midbrain. Angiograms show no source of the bleeding. The cause is thought to be venous or small arterial bleeding.[54] Headache characteristics are not distinct from conventional SAH, which suggests that the real mechanism that produces a sudden severe headache is not necessarily high-pressure bleeding, a large artery rupture hole, or a large subarachnoid hemorrhage. Regardless of pressure, sudden severe headache can develop at the time of rupture of vessels that have pain nerve fibers.

SUMMARY

The pain from an unruptured cerebral aneurysm can occur if it develops in proximal cerebral arteries in which pain fibers are present. These arteries can be the ICA, proximal ACA and MCA, vertebrobasilar arteries, and proximal posterior cerebral artery. Aneurysms at distal arteries do not produce headache unless they stimulate other pain-sensitive structure. The exact level which pain nerve fibers reach on the vessel walls varies between individuals, so some distal aneurysms may produce pain. Slowly formed aneurysms may not induce pain. Rapidly growing or forming aneurysms can cause pain. Acute changes to preexisting aneurysms or arteries can produce pain. Stimulation of larger arterial areas (large aneurysms) possibly causes more pain than small aneurysms.[55] Pain location and characteristics from the aneurysm can be various and nonspecific. The pain-sensing nerves for intracranial structures are the ophthalmic nerve (V1 of the trigeminal nerve)

and upper cervical spinal nerves. Maxillary nerves, mandibular nerves, and other minor nerves from VII, IX, and X have sensory function but mainly innervate dura and ear structures. Therefore, pain from supratentorial structures is felt in the orbitofrontal area and that from infratentorial structures is felt at the posterior neck. Pain occurs mostly on the ipsilateral side but some fibers cross-innervate, so bilateral perception may be possible.[21] A sudden severe headache possibly indicates that an acute pain-producing stimulation has occurred. They could include a variety of situations such as rupture of vessels or cerebral aneurysms, tearing (dissection), acute expansion (daughter sac formation), spasm and dilatation (RCVS), embolism (sudden imbedding or stretching of major cerebral arteries by emboli), and migraine. Pain location does not provide decisive information. Diagnosis still depends on the physician's clinical suspicion and work-up.

REFERENCES

1. International Study of Unruptured Intracranial Aneurysms Investigators. Unruptured intracranial aneurysms–risk of rupture and risks of surgical intervention. N Engl J Med 1998;339(24):1725–33.
2. Raps EC, Rogers JD, Galetta SL, et al. The clinical spectrum of unruptured intracranial aneurysms. Arch Neurol 1993;50(3):265–8.
3. Lebedeva ER, Gurary NM, Sakovich VP, et al. Migraine before rupture of intracranial aneurysms. J Headache Pain 2013;14:15.
4. Edvinsson L. Calcitonin gene-related peptide (CGRP) in cerebrovascular disease. ScientificWorldJournal 2002;2:1484–90.
5. Veloso F, Kumar K, Toth C. Headache secondary to deep brain implantation. Headache 1998;38(7):507–15.
6. Goadsby PJ, Lipton RB, Ferrari MD. Migraine–current understanding and treatment. N Engl J Med 2002;346(4):257–70.
7. Tubbs RS, Loukas M, Slappey JB, et al. Clinical anatomy of the C1 dorsal root, ganglion, and ramus: a review and anatomical study. Clin Anat 2007;20(6):624–7.
8. Fontaine D, Almairac F. Pain during awake craniotomy for brain tumor resection. Incidence, causes, consequences and management. Neurochirurgie 2017;63(3):204–7.
9. Wolff DL, Naruse R, Gold M. Nonopioid anesthesia for awake craniotomy: a case report. AANA J 2010;78(1):29–32.
10. Ray B, Wolff H. Experimental studies on headache. Pain sensitive structures of the head and their significance in headache. Arch Surg 1940;41:813–56.
11. Penfield W, McNaughton F. Dural headache and innervation of the dura mater. Arch Neurol Psychiatr 1940;44:43–75.
12. Nichols FT 3rd, Mawad M, Mohr JP, et al. Focal headache during balloon inflation in the internal carotid and middle cerebral arteries. Stroke 1990;21(4):555–9.
13. Nichols FT 3rd, Mawad M, Mohr JP, et al. Focal headache during balloon inflation in the vertebral and basilar arteries. Headache 1993;33(2):87–9.
14. Choi KS, Lee JH, Yi HJ, et al. Incidence and risk factors of postoperative headache after endovascular coil embolization of unruptured intracranial aneurysms. Acta Neurochir (Wien) 2014;156(7):1281–7.
15. Hwang G, Jeong EA, Sohn JH, et al. The characteristics and risk factors of headache development after the coil embolization of an unruptured aneurysm. AJNR Am J Neuroradiol 2012;33(9):1676–8.
16. Fay T. Atypical facial neuralgia, a syndrome of vascular pain. Ann Otol Rhinol Laryngol 1932;41:1030–62.
17. Baron EP, Moskowitz SI, Tepper SJ, et al. Headache following intracranial neuroendovascular procedures. Headache 2012;52(5):739–48.
18. Price DD, McHaffie JG, Larson MA. Spatial summation of heat-induced pain: influence of stimulus area and spatial separation of stimuli on perceived pain sensation intensity and unpleasantness. J Neurophysiol 1989;62(6):1270–9.
19. Staud R, Koo E, Robinson ME, et al. Spatial summation of mechanically evoked muscle pain and painful aftersensations in normal subjects and fibromyalgia patients. Pain 2007;130(1–2):177–87.
20. May A, Buchel C, Turner R, et al. Magnetic resonance angiography in facial and other pain: neurovascular mechanisms of trigeminal sensation. J Cereb Blood Flow Metab 2001;21(10):1171–6.
21. Rodriguez-Catarino M, Frisen L, Wikholm G, et al. Internal carotid artery aneurysms, cranial nerve dysfunction and headache: the role of deformation and pulsation. Neuroradiology 2003;45(4):236–40.
22. Drummond PD, Lance JW. Extracranial vascular changes and the source of pain in migraine headache. Ann Neurol 1983;13(1):32–7.
23. Arndt JO, Klement W. Pain evoked by polymodal stimulation of hand veins in humans. J Physiol 1991;440:467–78.
24. Schwedt TJ, Gereau RW, Frey K, et al. Headache outcomes following treatment of unruptured intracranial aneurysms: a prospective analysis. Cephalalgia 2011;31(10):1082–9.
25. Qureshi AI, Suri MF, Kim SH, et al. Effect of endovascular treatment on headaches in patients with unruptured intracranial aneurysms. Headache 2003;43(10):1090–6.
26. Kong DS, Hong SC, Jung YJ, et al. Improvement of chronic headache after treatment of unruptured

intracranial aneurysms. Headache 2007;47(5): 693–7.
27. Li H, Zhang X, Zhang QR, et al. Resolution of migraine-like headache by coil embolization of a primitive trigeminal artery aneurysm. Pain Med 2014;15(6):1052–5.
28. Choxi AA, Durrani AK, Mericle RA. Both surgical clipping and endovascular embolization of unruptured intracranial aneurysms are associated with long-term improvement in self-reported quantitative headache scores. Neurosurgery 2011;69(1): 128–33 [discussion: 133–4].
29. Gu DQ, Duan CZ, Li XF, et al. Effect of endovascular treatment on headache in elderly patients with unruptured intracranial aneurysms. AJNR Am J Neuroradiol 2013;34(6):1227–31.
30. Wirth FP Jr, Van Buren JM. Referral of pain from dural stimulation in man. J Neurosurg 1971;34(5):630–42.
31. Penfield W. A contribution to the mechanism of intracranial pain. Assoc Res Nerv Ment Dis 1932;15: 399–416.
32. Feindel W, Penfield W, Mc NF. The tentorial nerves and localization of intracranial pain in man. Neurology 1960;10:555–63.
33. Edmeads J. Vascular headaches and the cranial circulation–another look. Headache 1979;19(3): 127–32.
34. Le Doare K, Akerman S, Holland PR, et al. Occipital afferent activation of second order neurons in the trigeminocervical complex in rat. Neurosci Lett 2006; 403(1–2):73–7.
35. Bartsch T, Goadsby PJ. Stimulation of the greater occipital nerve induces increased central excitability of dural afferent input. Brain 2002;125(Pt 7):1496–509.
36. Johnston MM, Jordan SE, Charles AC. Pain referral patterns of the C1 to C3 nerves: implications for headache disorders. Ann Neurol 2013;74(1):145–8.
37. Piovesan EJ, Kowacs PA, Tatsui CE, et al. Referred pain after painful stimulation of the greater occipital nerve in humans: evidence of convergence of cervical afferences on trigeminal nuclei. Cephalalgia 2001;21(2):107–9.
38. Berger MS, Wilson CB. Intracranial dissecting aneurysms of the posterior circulation. Report of six cases and review of the literature. J Neurosurg 1984;61(5):882–94.
39. Kerr RW. A mechanism to account for frontal headache in cases of posterior-fossa tumors. J Neurosurg 1961;18:605–9.
40. Kimmel DL. Innervation of spinal dura mater and dura mater of the posterior cranial fossa. Neurology 1961;11:800–9.
41. Goedee HS, Depauw PR, vd Zwam B, et al. Superficial temporal artery-middle cerebral artery bypass surgery in a pediatric giant intracranial aneurysm presenting as migraine-like episodes. Childs Nerv Syst 2009;25(2):257–61.
42. Hahn CD, Nicolle DA, Lownie SP, et al. Giant cavernous carotid aneurysms: clinical presentation in fifty-seven cases. J Neuroophthalmol 2000;20(4):253–8.
43. Lanzino G, Andreoli A, Tognetti F, et al. Orbital pain and unruptured carotid-posterior communicating artery aneurysms: the role of sensory fibers of the third cranial nerve. Acta Neurochir (Wien) 1993;120(1–2):7–11.
44. Kojo N, Lee S, Otsuru K, et al. A case of ophthalmoplegic migraine with cerebral aneurysm. No Shinkei Geka 1988;16(5 Suppl):503–7 [Japanese].
45. Di Stefano G, Limbucci N, Cruccu G, et al. Trigeminal neuralgia completely relieved after stent-assisted coiling of a superior cerebellar artery aneurysm. World Neurosurg 2017;101:812.e5-9.
46. Ildan F, Gocer AI, Bagdatoglu H, et al. Isolated trigeminal neuralgia secondary to distal anterior inferior cerebellar artery aneurysm. Neurosurg Rev 1996;19(1):43–6.
47. Mendelowitsch A, Radue EW, Gratzl O. Aneurysm, arteriovenous malformation and arteriovenous fistula in posterior fossa compression syndrome. Eur Neurol 1990;30(6):338–42.
48. Sindou M, Leston J, Howeidy T, et al. Micro-vascular decompression for primary trigeminal neuralgia (typical or atypical). Long-term effectiveness on pain; prospective study with survival analysis in a consecutive series of 362 patients. Acta Neurochir (Wien) 2006;148(12):1235–45 [discussion: 1245].
49. Sindou M, Howeidy T, Acevedo G. Anatomical observations during microvascular decompression for idiopathic trigeminal neuralgia (with correlations between topography of pain and site of the neurovascular conflict). Prospective study in a series of 579 patients. Acta Neurochir (Wien) 2002;144(1):1–12 [discussion: 12–3].
50. Al-Shahi R, White PM, Davenport RJ, et al. Subarachnoid haemorrhage: lumbar puncture for every negative scan? Authors' reply. BMJ 2006;333(7567):550.
51. Linn FH, Rinkel GJ, Algra A, et al. Headache characteristics in subarachnoid haemorrhage and benign thunderclap headache. J Neurol Neurosurg Psychiatry 1998;65(5):791–3.
52. Sarner M, Rose F. Clinical presentation of ruptured intracranial aneurysm. J Neurol Neurosurg Psychiatry 1967;30:67–70.
53. Polmear A. Sentinel headaches in aneurysmal subarachnoid haemorrhage: what is the true incidence? A systematic review. Cephalalgia 2003;23(10): 935–41.
54. van Gijn J, van Dongen KJ, Vermeulen M, et al. Perimesencephalic hemorrhage: a nonaneurysmal and benign form of subarachnoid hemorrhage. Neurology 1985;35(4):493–7.
55. Ji W, Liu A, Yang X, et al. Incidence and predictors of headache relief after endovascular treatment in patients with unruptured intracranial aneurysms. Interv Neuroradiol 2017;23(1):18–27.