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A case report with a literature review of trigeminal neuralgia secondary to a large posterior communicating artery aneurysm

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ABSTRACT

This is a case report of a 66-year-old female who developed atypical trigeminal neuralgia over ophthalmic-V1 and maxillary-V2 divisions secondary to a posterior communicating artery aneurysm (1.5 cm). Literature review of similar cases, description of the pain, its distribution, the aneurysm features, the pathophysiology, and the patient's management and outcome are presented.

1. Introduction

Trigeminal neuralgia (TGN), also known as tic douloureux, is a neuropathic facial pain. TGN is commonly a unilateral recurrent paroxysm shock-like pain, but some patients also have continuous pain. Trigeminal neuralgia is categorized into two types: primary (idiopathic) or secondary (Mafi et al., 2019). The most common secondary causes are enlarged or lengthened blood vessels – most commonly the superior cerebellar artery – compressing or throbbing against the microvasculature of the trigeminal nerve near its exit from the pons (Nurmikko et al., 2001). Other vascular causes include aneurysms and arteriovenous malformations. Less commonly TGN can be caused by tumors, such as vestibular schwannomas, cerebellopontine angle or Petro-clival meningiomas, or arachnoid cysts.

According to the literature, Posterior Communicating Artery (P-ComA) aneurysm is a rarely the cause of TGN (de San Pedro, 2017), as it usually presents with oculomotor nerve deficit. We will discuss our case of TGN secondary to P-ComA aneurysm with a review of similar reported cases in the literature, as well as the etiology of pain, treatment options, and results.

2. Case report

A 66-year-old female patient, known to have hypertension, and a previous history of hysterectomy, was referred to the department of neurosurgery at San Filippo Neri hospital complaining of right atypical trigeminal neuralgia for two months duration. The pain symptoms were distributed over the V1 and V2 territories, and she described continuous tingling and numbness. She had some pain-free periods, with no triggers for the pain. She denied any facial weakness, or any changes in her visual acuity.

The patient showed no signs of physical distress throughout the physical assessment. Her neurologic examination was unremarkable; tests of her strength, gait, and cranial nerves revealed that they were all intact.

Magnetic resonance imaging (MRI) showed multiple intracranial aneurysms, including a right P-ComA aneurysm with an irregular area of intensity inside the aneurysm, indicating mural thrombus and turbulent flow as seen in (Fig. 1).

Cranial computed tomography angiography (CTA), as seen in (Fig. 2), revealed a partially thrombosed right P-ComA aneurysm, that directed posteriorly lateral-inferior. The long axis measured 1.5 cm, the neck measured 0.5 cm. There was an aneurysm on the right M2-M3

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middle cerebral artery (MCA) junction. And another three aneurysms on the left side. Her findings of multiple aneurysms indicated an image of her chest, abdomen, and pelvis that showed multiple kidney cysts on both kidneys (Fig. 3), consistent with an autosomal dominant polycystic kidney disease (ADPKD).

The surgical procedure was carried out with intraoperative neuromonitoring (IONM) for the facial and trigeminal nerves (Nimbus i-Care-100, Newmedic-Hemodia, Labege, France) by performing right pterional craniotomy approach and clipping. Following the clipping of the MCA aneurysm first and while dissecting down to reach the P-ComA aneurysm, we had an irritation on the trigeminal nerve monitor, as seen in (Fig. 4), then it disappeared. When the aneurysm was reached, we could see it compressing the oculomotor nerve. After clipping, doppler ultrasonography was used to confirm normal distal ICA and A1-M1 branching flow. The aneurysm was then fully excluded from the parent circulation, then opened and the thrombus was removed. IGC confirmed the exclusion of both aneurysms from the circulation.

Post-operative course: The patient did not have any neurological abnormalities, her trigeminal pain disappeared completely and immediately, and she was discharged home after three days.

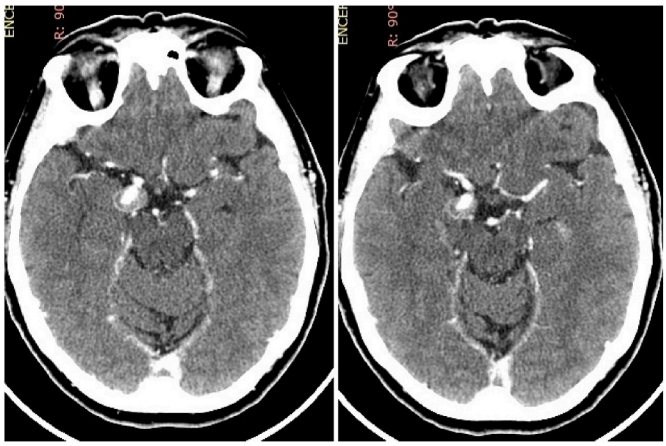

Fig. 2. Cranial computed tomography shows a large right posterior communicating artery aneurysm partially thrombosed.

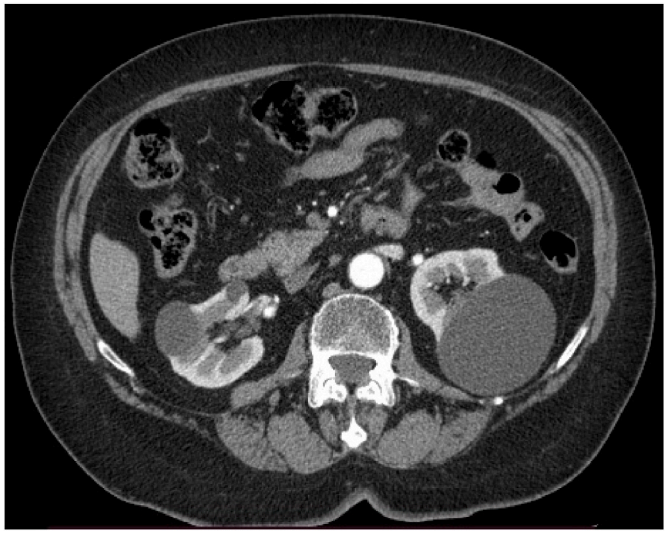

Fig. 3. Abdomen computed tomography shows multiple different size kidney cysts.

3. Methods

We described the case of 66-year-old women who suffered atypical TGN over V1 and V2 distributions secondary to a large right posterior communicating artery aneurysm.

A literature review was achieved in order to describe similar cases.

Search strategy: the following search strategies were used for database.

Pubmed: ("trigeminal neuralgia" and "aneurysm"), ("trigeminal neuralgia" and "posterior communicating artery aneurysm"), ("trigeminal neuralgia" and "posterior communicating artery").

Google scholar: ("trigeminal neuralgia" and "posterior communicating artery aneurysm").

Searching Pubmed yielded 162 studies, google scholar resulted 231 studies. Using our search strategy, we initially found 393 studies. After discarding the duplicates and non-related studies, we selected the studies of aneurysms and trigeminal neuralgia (result 30), eventually - for studies who have this entity of P-ComA aneurysm with TGN – a total number of 9 studies was collected (included 11 cases), 1 study had and incomplete data for 2 reported cases, therefore it was excluded.

The remaining studies were reviewed and data was collected on patient demographics, quality of neuralgia, aneurysm features, and type of cranial nerve involvement. These are reported in Table 1.

The type of trigeminal neuralgia was classified as typical or atypical. Typical TGN was defined as recurrent paroxysms of unilateral pain in the distribution(s) of one or more divisions of the trigeminal nerve which could be reproduced by a trigger. Atypical TGN was used when the pain did not meet the typical description. Also, we collected the distribution of pain from V1 to V3.

Aneurysm features collected were size and projection. Size was defined as the longest axis of the aneurysm in centimeter (cm). The aneurysms were classified as small <0.5 cm, medium 0.5–1 cm, large

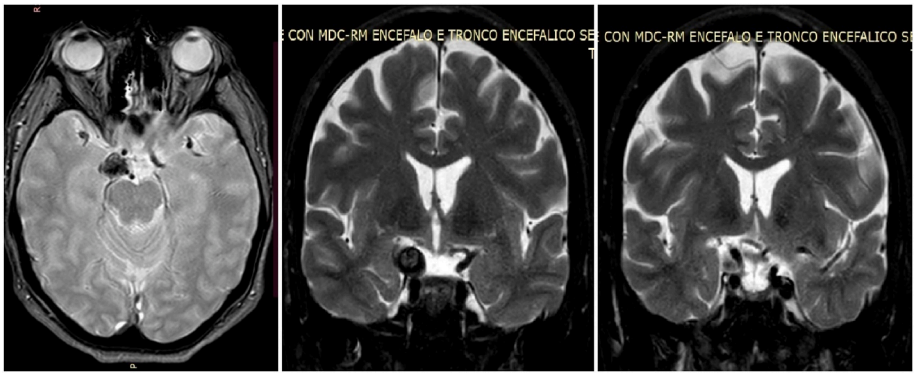

Fig. 1. MR images reveal a large right P-ComA aneurysm about 1.5 cm in diameter, with areas of iso- and low-intensity on a T2-weighted image (left), Coronal T2-weighted image (right) clearly demonstrates the relationship between the aneurysm and the cavernous sinus.

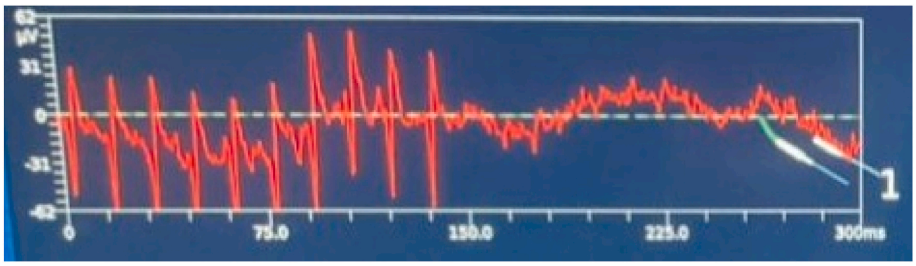

Fig. 4. Intraoperative Neuromonitoring for the trigeminal nerve shows bursts while manipulating the carotid artery reaching the aneurysm.

Table 1
Review of the literature, V1 (ophthalmic division), V2 (maxillary division), PMI (Posterior Medial Inferior), PLI (Posterior Lateral Inferior), CS (Cavernous Sinus), V (trigeminal nerve), III (oculomotor nerve).

Author	Age	Gender	TGN pain	Distribution	Size	Direction	Mechanism	III involvement	Intervention	Outcome
Leopold et al., 1980 (Leopold et al., 1980)	62	Male	Typical	V2	Large	PMI	CS	V then III 1.5 year	Clipping	Resolved
Broggi et al., 1982 (Broggi et al., 1982)	62	Female	Atypical	V1-2	Large	PMI	CS	V then III 1 year	Trigeminal Rhizotomy	Resolved
Trotter et al., 2000 (Trotter et al., 2000)	52	Female	Atypical	V2	2.5 cm	PMI	CS		Clipping	Resolved
Terao et al., 2001 (Terao et al., 2001) **ADPKD**	56	Male	Atypical	V1	3.2 cm	PMI	V and III	V and III	Clipping	Resolved in 3 months
Simonet et al., 2016 (Simonet et al., 2016)	73	Male	Typical	V1	3 cm	PLI	V		Clipping	Resolved
Zelman et al., 2016 (Zelman et al., 2016)	42	Female	Atypical	V1	0.7 cm	PLI	III	V then III 2 weeks	Clipping	Resolved
Nakagawa et al., 2017 (Nakagawa et al., 2017)	67	Female	Atypical	V1	2 cm	PMI	III	V then III 2year	Clipping	Resolved
Javier et al. 2017 (de San Pedro, 2017)	61	Female	Atypical	V1-2	4 cm	PMI	V		Clipping	Resolved
Javier et al. 2017 (de San Pedro, 2017)	51	Female	Atypical	V2	1.5 cm	PMI	CS		Medication	Fair control
Presenting case	78	Female	Atypical	V1-2	1.5 cm	PLI	III		Clipping	Resolved

1–2.5 cm, and giant >2.5 cm (Merritt et al., 2021). The direction of the aneurysm also was collected and as they all share one common thing that is posterior and inferior projection and the only difference was medial or lateral projection.

Care was taken to determine and record the mechanism of cranial nerve involvement. We identified: 1. direct cavernous sinus compression without compression on the trigeminal nerve directly, 2. direct trigeminal nerve compression, 3. direct oculomotor nerve compression without compression on trigeminal nerve, and 4. Compression on both trigeminal nerve and oculomotor nerve.

In the cases of involvement of third cranial nerve, the timeline of the trigeminal neuralgia with the oculomotor neuropathy was considered.

4. Results

Only 12 cases of TGN secondary to P-ComA aneurysm have been reported in the literature, including our case as shown in (Table 1) (de San Pedro, 2017; Leopold et al., 1980; Broggi et al., 1982; Trotter et al., 2000; Terao et al., 2001; Simonet et al., 2016; Zelman et al., 2016; Nakagawa et al., 2017) - which contains data for 10 cases - while the other 2 cases have been reported by Watanabe et al. (Watanabe et al., 1982). He only mentioned the pain distribution in those patients, one case had V1 distribution and the other had V1 + V2 distribution with no more data.

Data from the literature revealed that 70 % of patients were female, and the average age was 60.4 years (42–78 years).

Atypical TGN was presented in 80 % of cases. All cases have V1 (40 %), V2 (20 %), or both distributions (40 %), while no case had a V3 distribution.

In terms of aneurysm size, data were missing for 2 cases, which were described as having large aneurysms. Among the remaining 8 cases, the average size of the aneurysms was 2.4 cm, with a range of 0.7–4 cm.

Regarding treatment approaches, 8 cases were managed with clipping, 1 case underwent trigeminal rhizotomy, and 1 case was treated with medication.

Analyzing the etiology of pain, we found that 4 cases involved cavernous sinus compression, 3 cases involved oculomotor nerve compression, and 2 cases involved trigeminal nerve compression. Additionally, 1 case presented with both trigeminal nerve and oculomotor nerve compression.

Oculomotor nerve deficits were observed in 50 % of the cases. Among those, 80 % experienced trigeminal neuralgia prior to the onset of oculomotor deficits, with timelines ranging from 2 weeks to 2 years. For 20 % of the cases, the timeline regarding the onset of oculomotor involvement remained unclear.

The outcome was, All the treated patients had a satisfactory result, as 80 % had immediate pain relief, 10 % had pain relief within three months (Terao et al., 2001), and 10 % had fair control of pain on medications (de San Pedro, 2017).

The presenting case with another case (Terao et al., 2001) are the only cases that had ADPKD. None of the revised cases had bleeding.

5. Discussion

Typical TGN is characterized by “recurrent paroxysms of unilateral pain in the distribution(s) of one or more divisions of the trigeminal nerve”, the pain can be reproduced by touching a “trigger point” on the face or performing a certain activity, such as chewing or talking

(Jurjević et al., 2009; Montano et al., 2015). While Atypical TGN describes cases in which the clinical findings do not fully meet the standard diagnostic criteria for TGN, like a dull ache or burning sensation in one part of the face. While episodes of sharp pain can complicate atypical TGN, usually, there is no specific trigger point for the pain.

Trigeminal neuralgia is categorized into two types: primary (idiopathic) or secondary. The most common secondary causes are enlarged or lengthened blood vessels – the most common blood vessel is the superior cerebellar artery – compressing or throbbing against the microvasculature of the trigeminal nerve near its connection with the pons (Nurmikko et al., 2001; Jurjević et al., 2009; Montano et al., 2015). Other causes include vascular malformations like aneurysms and arteriovenous malformations, and less commonly tumors; such as an arachnoid cyst or meningioma in the cerebellopontine angle, or traumatic events (Jurjević et al., 2009; Kano et al., 2011).

The most common location of TGN secondary to the cerebral aneurysm is the superior cerebellar artery (Di Stefano et al., 2017). Other sites include the Anterior Inferior Cerebellar Artery (İldan et al., 1996), Vertebrobasilar artery (Mendelowitsch et al., 1990), Posterior Cerebral artery (Dzierżanowski et al., 2014), Persistent Trigeminal Artery (Chen et al., 2015), Internal Carotid Artery (ICA)-Cavernous Segment (Zachariades et al., 2004), ICA-Supraclinoidal Segment, and, very rarely, the Posterior Communicating Artery (P-ComA) (de San Pedro, 2017).

P-ComA aneurysm is known more to cause an oculomotor nerve deficit due to the anatomical relation, and it rarely has been reported to cause trigeminal neuralgia. We found that when these aneurysms cause TGN in 50 % of cases it is also associated with oculomotor nerve deficit. Furthermore, the TGN precedes the oculomotor deficit 2 weeks to 2 years. So, we have to keep in mind that trigeminal neuralgia can be a first presentation for P-ComA aneurysm.

Regarding the pain distribution, all cases had V1, V2, or both distributions, which is the expected distribution in the case of superior compression etiology (secondary TGN), unlike the primary (idiopathic) TGN where it has a V3 and V2 distribution mainly (Sindou et al., 2006).

We noticed that all the aneurysms that are compressing the cavernous sinus are large aneurysms (1–2.5 cm). In comparison, all giant aneurysms (>2.5 cm) compress on the trigeminal nerve. No relation was found between the size of the aneurysm and the oculomotor nerve compression.

Despite the limited number of cases, there are four etiologies for pain: The first is seen in 4 cases (de San Pedro, 2017; Leopold et al., 1980; Broggi et al., 1982; Trotter et al., 2000), which is compression of the cavernous sinus, and they all share similar projection of the aneurysm which is posterior medial and inferior. Also, they are all large aneurysms, and those patients exhibit pain mainly in the V2 distribution. A possible explanation for this is the position of V2 as it is intermediate.

The second which is seen in the presenting case and 2 other cases (Zelman et al., 2016; Nakagawa et al., 2017), is compression of the oculomotor cranial nerve. Those patients exhibit pain mainly on V1 distribution. Lanzino et al. (1993) studied orbital pain with unruptured P-ComA aneurysm on 14 human autopsies and found a sensory pathway within the third nerve, which means that the third CN is not only an efferent nerve but also contains sensory ganglion cells and conveys afferent fibers which come from V1 and synapse in the spinal trigeminal nucleus. Therefore, stress upon the oculomotor nerve may exacerbate V1-mediated orbital pain through the trigeminal ganglion and this may explain the pain in those cases.

The third etiology is seen in 2 cases (de San Pedro, 2017; Simonet et al., 2016), which is compression of the trigeminal nerve itself. Both of these were caused by giant aneurysms.

The fourth etiology is seen in one case (Terao et al., 2001) which is compression of both trigeminal nerve and oculomotor nerve, and in this case also the aneurysm size was giant.

Generally, the choice between open techniques and endovascular intervention for aneurysms causing cranial neuropathies remains controversial because there is no clinical evidence regarding the superiority of one over the other (Gaberel et al., 2016). More specifically, a review of the management of the P-ComA aneurysms (Golshani et al., 2010) showed that coiling has a higher risk of recurrence and needs close follow up. Furthermore, coiling in those cases would not solve the main problem of the mass effect of the aneurysm, and recent studies on aneurysm management are talking about the combination of treatment of endovascular with microsurgical bypass (Zomorodi et al., 2010). 80 % of the reported cases have been surgically (clipping) treated with satisfactory results; only one case treated by trigeminal rhizotomy due to the high risk of anaesthesia from chronic bronchitis and myocardial disease, but she also had a good outcome. One case was treated with medication and had a fair control of her pain.

It is possible that more cases may have been concealed by the rupture event due to the rapidity of events or the severity of presentation, as none of the patients exhibited subarachnoid haemorrhage in their histories or clinical presentations.

It was very helpful intra-operative to notice a trigeminal nerve irritation during dissection around the aneurysm which could not be explained clearly because there was no direct contact of the aneurysm itself with the trigeminal nerve.

6. Conclusions

TGN is rarely seen as an isolated symptom secondary to aneurysms, but finding TGN secondary to a P-ComA aneurysm is even more rare. TGN (especially Atypical) should cause a physician to pay attention to a serious underlying fatal intracranial aneurysm.

Every patient who complains of facial pain should undergo a thorough, detailed neurological examination. Any abnormalities on this exam should prompt further consideration of neuroimaging investigation since they may be the source of a potentially fatal intracranial disease like a P-ComA aneurysm. Early surgical clipping of large and/or symptomatic aneurysms can reduce mortality and improve patient quality of life. Using intraoperative neuromonitoring is essential and helpful in this condition.

Ethical approval

Patient Informed Consent, enrolled patients agreed to and signed informed consent for this study.

Authors' contributions

CG, FB and RR performed the literature search, included and excluded the papers and analysed data. GC prepared tables and figures. AAA oversaw data analysis, and wrote the main manuscript text. AZ revised the data and manuscript with editing. LM oversaw data analysis, and critically revised the article. The final manuscript was approved by all authors.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

References

Broggi, G., Franzini, A., 1982. Radiofrequency trigeminal rhizotomy in treatment of symptomatic non-neoplastic facial pain. J. Neurosurg. 57 (4), 483–486.

Chen, W.-H., et al., 2015. A case of giant thrombosed persistent primitive trigeminal artery aneurysm presenting with trigeminal neuralgia and successfully treated by a covered stent: case report and review of literature. Clin. Neuroradiol. 25 (2), 207–210.
de San Pedro, J.R., 2017. Posterior communicating artery aneurysms causing facial pain: a comprehensive review. Clin. Neurol. Neurosurg. 160, 59–68.
Di Stefano, G., et al., 2017. Trigeminal neuralgia completely relieved after stent-assisted coiling of a superior cerebellar artery aneurysm. World Neurosurgery 101, 812. e5–812.e9.
Dzierżanowski, J., Słoniewski, P., 2014. Trigeminal neuralgia caused by aneurysm of the posterior cerebral artery: a case description and the analysis of anatomical variety of vascular complex in the rood entry zone of trigeminal nerve. Folia Morphol. 73 (2), 224–228.
Gaberel, T., et al., 2016. Clipping versus coiling in the management of posterior communicating artery aneurysms with third nerve palsy: a systematic review and meta-analysis. World Neurosurgery 87, 498–506.e4.
Golshani, K., et al., 2010. A review of the management of posterior communicating artery aneurysms in the modern era. Surg. Neurol. Int. 1.
İldan, F., et al., 1996. Isolated trigeminal neuralgia secondary to distal anterior inferior cerebellar artery aneurysm. Neurosurg. Rev. 19 (1), 43–46.
Jurjević, A., et al., 2009. Trigeminal neuralgia secondary to meningioma of petroclival localization. Coll. Antropol. 33 (1), 323–325.
Kano, H., et al., 2011. Stereotactic radiosurgery for patients with trigeminal neuralgia associated with petroclival meningiomas. Stereotact. Funct. Neurosurg. 89 (1), 17–24.
Lanzino, G., et al., 1993. Orbital pain and unruptured carotid-posterior communicating artery aneurysms: the role of sensory fibers of the third cranial nerve. Acta Neurochir. 120 (1), 7–11.
Leopold, N., Hirsh, L., Ray, T., 1980. Paroxysmal facial neuralgia secondary to a posterior communicating artery aneurysm. Surg. Neurol. 14 (3), 221–223.
Mafi, M., Rezvani, F., 2019. A case series study of trigeminal neuralgia secondary to brain tumor meningioma. EC Neurol. 11, 417–418.
Mendelowitsch, A., Radue, E., Gratzl, O., 1990. Aneurysm, arteriovenous malformation and arteriovenous fistula in posterior fossa compression syndrome. Eur. Neurol. 30 (6), 338–342.
Merritt, W.C., et al., 2021. Definitions of intracranial aneurysm size and morphology: a call for standardization. Surg. Neurol. Int. 12.
Montano, N., et al., 2015. Advances in diagnosis and treatment of trigeminal neuralgia. Therapeut. Clin. Risk Manag. 11, 289.
Nakagawa, M., et al., 2017. Large thrombosed internal carotid-posterior communicating artery aneurysm presenting with atypical trigeminal neuralgia: a case study. Surg. Cerebr. Stroke 45 (1), 41–45.
Nurmikko, T., Eldridge, P., 2001. Trigeminal neuralgia—pathophysiology, diagnosis and current treatment. Br. J. Anaesth. 87 (1), 117–132.
Simonet, C., Arrese, I., Sarabia, R., 2016. Giant posterior communicating artery aneurysm presenting as trigeminal neuralgia. J. Stroke Cerebrovasc. Dis. 25 (9), 2243–2246.
Sindou, M., et al., 2006. 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. 148, 1235–1245.
Terao, S., et al., 2001. A giant internal carotid-posterior communicating artery aneurysm presenting with atypical trigeminal neuralgia and facial nerve palsy in a patient with autosomal dominant polycystic kidney disease: a case report. Surg. Neurol. 56 (2), 127–131.
Trotter, M., Choksey, M., 2000. Facial pain with intracranial aneurysm. J. R. Soc. Med. 93 (9), 479–480.
Watanabe, A., et al., 1982. Relation of cranial nerve involvement to the location of intracranial aneurysms. Neurol. Med.-Chir. 22 (11), 910–916.
Zachariades, N., et al., 2004. Nonidiopathic trigeminal neuralgia: report of a case. Quintessence Int. 35 (8).
Zelman, S., et al., 2016. Large posterior communicating artery aneurysm: initial presentation with reproducible facial pain without cranial nerve deficit. West. J. Emerg. Med. 17 (6), 808.
Zomorodi, A., et al., 2010. Combined microsurgical and endovascular treatment of a giant left middle cerebral artery aneurysm. J. Neurointerventional Surg. 2 (3), 213–216.