Giant Posterior Communicating Artery Aneurysm Presenting as Trigeminal Neuralgia

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Background: We report an extremely rare case of trigeminal neuralgia (TN) caused by a giant posterior communicating artery aneurysm. *Methods:* We describe the case of a 73-year-old man who suffered disabling TN, which was deeply exacerbated when he laid down. Computed tomography angiography and digital angiography revealed an unruptured giant posterior left communicating artery aneurysm projecting to the posterior fossa and contacting the trigeminal root. *Results:* The size and projection of the aneurysm seemed responsible for both the neuralgia itself and its peculiar clinical characteristic of exacerbation when the patient laid down. The aneurysm was surgically clipped, then opened and emptied to decompress the trigeminal root. The patient reported an immediate complete remission of the neuralgia. *Conclusion:* The finding of TN secondary to aneurysms is rare but even more in the case of a posterior communicating aneurysm. The clinical clue that should alert physicians about the presence of an aneurysm as the cause of TN is exacerbation of pain by adopting a supine position. Due to the high risk of rupture associated with giant and symptomatic aneurysms, we believe that treatment should be aggressive in this case, not only to solve the symptomatic TN but also to avoid the risk of aneurysm rupture in the future. The treatment selection between surgical clipping or endovascular coiling with or without stenting, depends on patient's clinical condition and the size and shape of the aneurysm.
Key Words: Giant aneurysm—posterior communicating artery aneurysm—trigeminal neuralgia—surgical clipping.

Introduction

Vascular compression of the trigeminal nerve root is the most frequent cause of typical or essential trigeminal neuralgia (TN). It is usually related to arteries located near the trigeminal root exit from the brain stem, such as the superior cerebellar artery, which is the most commonly involved vessel.

TN secondary to vascular compression caused by an aneurysm has rarely been described and only isolated cases can be found in the literature. There are scarcely reported cases regarding trigeminal root compression by aneurysms of the anterior inferior cerebellar artery, persistent primitive trigeminal artery, and exceptionally, posterior communicating artery (PComA). The last association is extremely rare because of the anatomical position of PComA aneurysms, far away from the trigeminal nerve root. We describe a case of giant PComA aneurysm causing typical TN.

Clinical Case

A 73-year-old man with a previous clinical history of arterial hypertension and prostatic carcinoma was referred to our unit because of disabling TN unresponsive to medical treatment. The patient described an

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Figure 1. *Cranial computed tomography shows a giant posterior communicating artery aneurysm (notch).*

Figure 2. *Arteriography shows an unruptured giant elongated saccular posterior communicating artery aneurysm.*

agonizing pain affecting the left V1 distribution appearing in brief repetitive bursts. Characteristically, the TN was deeply exacerbated when the patient laid down.

Contrast-enhanced cranial computed tomography revealed an unruptured giant posterior left communicating artery aneurysm (Fig 1), which was confirmed by cerebral angiography (Fig 2). The aneurysm had an elongated and saccular form, measuring more than 30 mm in depth but with a 4-mm neck.

Surgical clipping of the aneurysm was offered to the patient after considering several different aspects, such as the age and good clinical condition of the patient, as well as the size and shape of the aneurysm.

The surgical intervention was performed through a left pterional approach using a microsurgical technique. The aneurysm was excluded by clipping the neck with a fenestrated clip with no complication during surgery, although clear signs of atherosclerosis were visible in the proximal internal carotid artery (ICA), mostly in the region of the aneurysm's neck. Intraoperative Doppler ultrasonography was used to confirm normal distal ICA and A1–M1 branching flow, with complete exclusion of the aneurysm sac from the parent circulation (Figs 3, 4). The aneurysm sac was thereafter surgically opened and its blood content was aspirated to eliminate any residual mass effect.

The patient's facial pain immediately disappeared following the surgery. The patient was awake and satisfied with no need of analgesic medication and was able to lie down without feeling any pain.

After 48 hours, the patient started with rapidly progressive right hemiparesis and speech disturbance, for which reason he was immediately transferred to the intensive care unit after suspicion of left ICA ischemia. This deferred ischemic complication was attributed to the instability of the atherosclerotic plaque seen in the surgical field, located in the intracranial left ICA. On the following days, the patient remained neurologically and clinically stable until the patient suffered severe tracheobronchitis, which required reintubation. Despite therapeutic efforts, the patient finally died after 10 days.

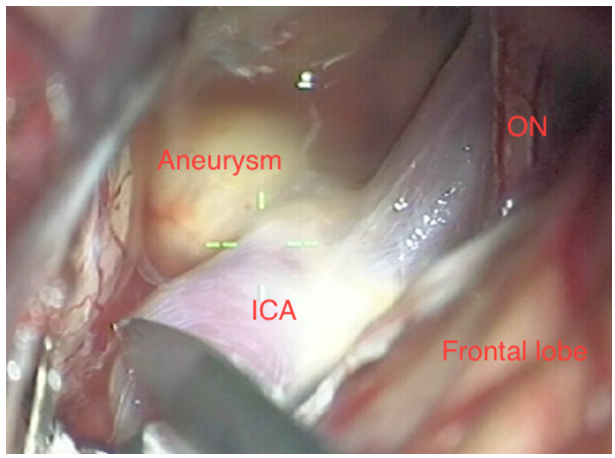

Figure 3. *Surgery procedure. Giant posterior communicating artery aneurysm. ICA. Abbreviation: ICA, internal carotid artery.*