

Coil Embolization For Sixth Cranial Nerve Palsy Caused By Persistent Primitive Trigeminal Artery

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Citation

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Abstract

The author reports on the case of a 60-year-old woman who presented with recurrent left sixth cranial nerve palsy. The patient was initially hospitalized to rule out an aneurysm of the internal carotid artery. Angiography revealed segmental dilation of the internal carotid artery and a persistent primitive trigeminal artery (PPTA) that coursed through the dorsolateral cavernous sinus, opening into the left anterior inferior cerebellar artery. Superselective visualization of the angioarchitecture allowed us to successfully occlude the cavernous segment of the PPTA by coil embolization. The palsy resolved and the patient remained asymptomatic for 24 months.

The data on the role that PPTAs play in compression syndromes in the cavernous sinus is inconclusive. The author concludes that the pathogenesis of the sixth nerve palsy can be caused in the existence of PPTA.

INTRODUCTION

The persistence of a primitive trigeminal artery (PPTA) in cerebral angiograms has been described often, however its clinical relevance is still the subject of discussion. Several authors have suggested associations with other vascular anomalies, particularly with the development of aneurysms and chronic pain syndromes in the craniofacial region.^{1,2,4,5} The suspicion that lesions of the sixth cranial nerve may also be caused by PPTA was expressed as early as 1989.⁵

We report on a patient with an anatomically unusual PPTA in the cavernous segment associated with sixth cranial nerve palsy who was successfully treated by coil embolization. The knowledge gained on this case may, for the first time, prove a causality between sixth cranial nerve palsy and primitive trigeminal arteries.

CASE REPORT

A 60-year-old, otherwise healthy woman presented with symptoms of recurrent left sixth cranial nerve palsy. She had a previous 3-year history of repeated episodes of palsy; both their duration and frequency had been on the increase. The patient was initially hospitalized to rule out an aneurysm of the internal carotid artery. A differential MRI scan was performed, but we were unable to clarify the pathology in the cavernous sinus due to the prominence of pulsation

artifacts.

Angiography revealed dilatation of the internal carotid artery (ICA) in segment C5 along with a persistent primitive trigeminal artery (PPTA) (Fig.1). The ipsilateral posterior cerebral artery (PCA) originated from the ICA, the ipsilateral anterior inferior cerebellar artery (AICA) was not contrasted over the basilar artery. Superselective angiography showed that the PPTA joined with the missing AICA (Fig. 2). We noted a permanent impression in the cavernous segment of the PPTA presumably caused by the sixth cranial nerve (Fig.3).

Figure 1

Figure 1: Lateral view of the ICA with a persistent primitive trigeminal artery giving off several strong branches into the posterior fossa.

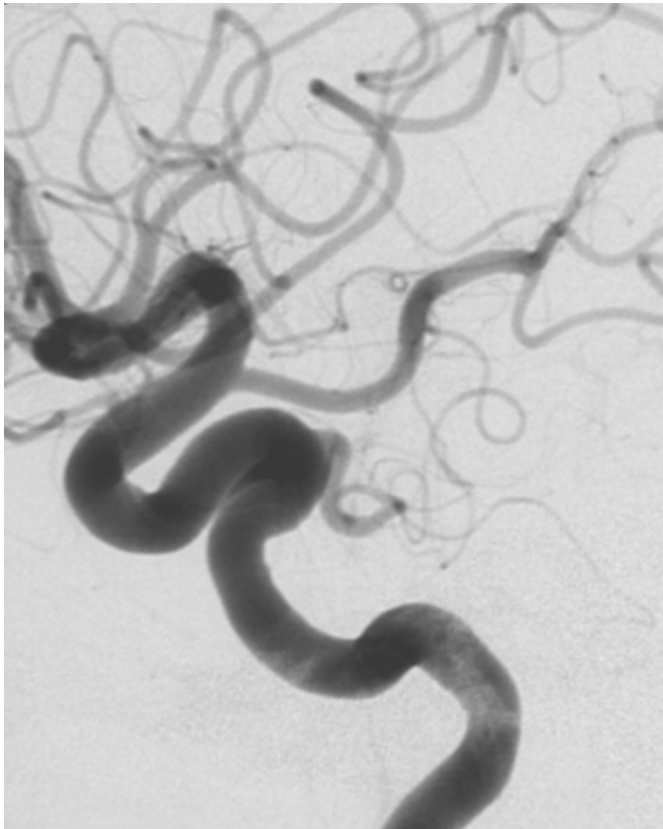


Figure 2

Figure 2: Lateral angiogram of the posterior fossa after superselective probing of the PPTA over the ICA (arrows) with antegrade filling of the AICA supply area. Retrograde staining of the top of the basilar artery (asterisk) over where it exits the original AICA.



Figure 3

Figure 3: After superselective probing of the PPTA, we achieved visualization of the PICA supply area. Note the impression of 1.2 mm in diameter, presumably caused by the sixth nerve (right oblique projection). The bursting caliber of the artery results from its dural penetration (arrow).



Figure 4

Figure 4: ICA after embolization of the PPTA using GDC Ultrasoft coils, lateral view.



Superselective visualization of the AICA exiting the basilar artery was also performed. It became obvious that the dilated, intracavernous course of the PPTA could be the only causal factor responsible for the patient's sixth nerve palsy. Therefore, we embolized the PPTA up to the point of dural penetration into the posterior fossa utilizing three short GDC Ultrasoft coils (Boston SCI) (Fig. 4). The non-subtracted venous angiograms of the cavernous sinus taken postembolization demonstrated the position of the PPTA in relation to the cavernous sinus (Fig. 6A/B). In the postprocedural angiogram, the supply to the AICA took the normal route, namely through the basilar artery (Fig. 5). At 3-month follow-up, the sixth cranial nerve had completely recovered.

Figure 5

Figure 5: Basilar artery after embolization of the PPTA with antegrade filling of the AICA, left (arrow).

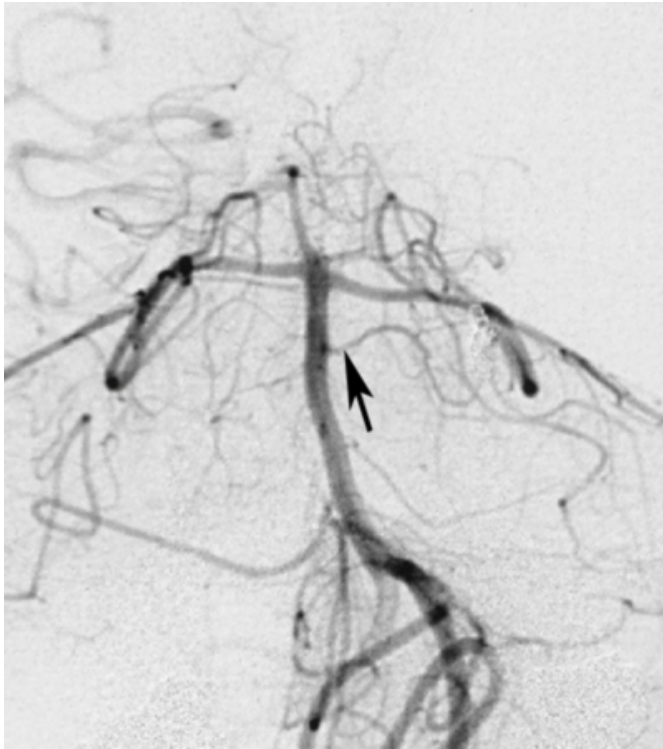


Figure 6

Figure 6A / B: A/P and lateral angiograms of the ICA, not subtracted, venous phase, with visualization of the coils in their positional relation to the cavernous sinus.



Figure 7

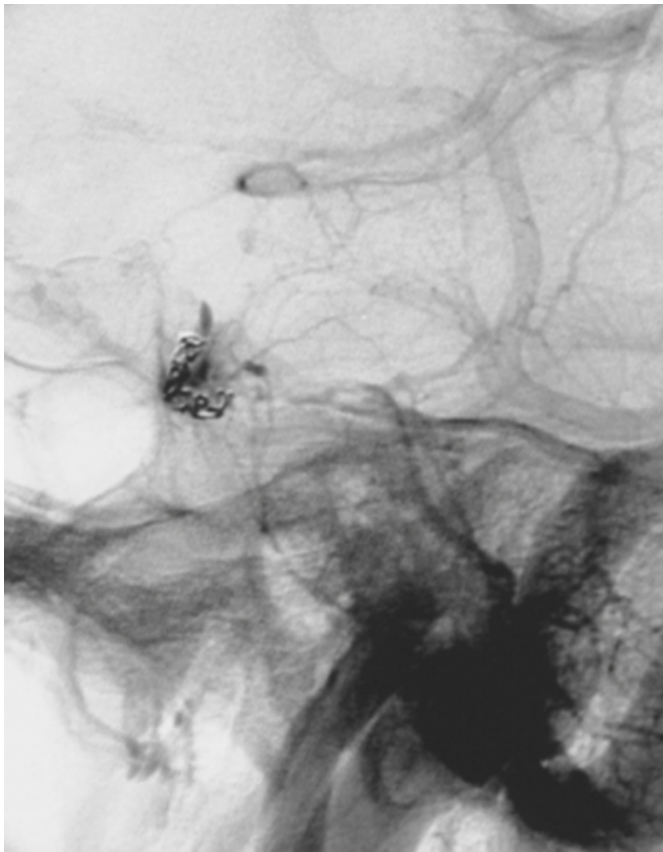
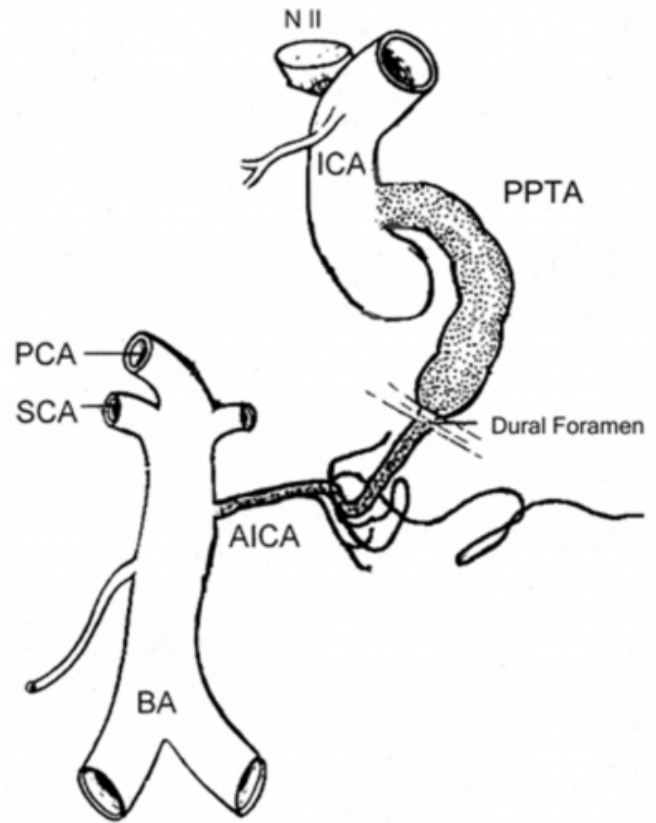


Figure 8

Figure 7: Schematic diagram of the flow patterns between AICA and PPTA. AICA = anterior inferior cerebellar artery, BA = basilar artery, ICA = internal carotid artery, N II = optic nerve, PCA = posterior cerebral artery, SCA = superior cerebellar artery, PPTA = persisting primitive trigeminal artery. The diagram has been modified according to Oshiro .



RESULTS

In the case described here, coil embolization of a PPTA localized in the cavernous segment was successfully used to treat sixth cranial nerve palsy.

DISCUSSION

Great variations can exist in the course and opening of a PPTA, which normally reaches the basilar artery between the superior cerebellar artery and the anterior inferior cerebellar artery. Vessels exiting the PPTA can branch off to supply the cavernous sinus, the meningo-hypophysial trunk and, further distally, can penetrate the dura to course into the posterior cranial fossa and supply the trigeminal nerve and the pons. ³ Anomalous branches of the cavernous carotid artery originating from the ICA have been described both anatomically and angiographically. ^{6,7}

Previous reports have involved PPTA that anastomosed with the AICA (Fig. 7) as in our case. We were only able to

superselectively visualize the vessels exiting the AICA, which, at the same time, was the precondition for embolization of the PPTA. The fact that we were able to indirectly visualize the sixth nerve in the cavernous sinus and that coil embolization of the PPTA led to a successful outcome confirmed the pathogenesis of this sixth nerve palsy. We presume that the nerve was damaged by pulsatile compression.

CONCLUSIONS

There is still debate as to whether persistent primitive trigeminal arteries are responsible for compression syndromes in the cavernous sinus. In the present case, there was no doubt that embolization of the PPTA in the cavernous segment led to conclusive resolution of the patient's sixth cranial nerve palsy. We were able to superselectively visualize the presence of an anastomosis between PPTA and AICA and its angiographically "blind" connection to the basilar artery. Our present findings provide further evidence supporting the presumption that there is a relationship between PPTA and cavernous syndromes.

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