

# Complete oculomotor palsy caused by persistent trigeminal artery

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**Abstract** Primitive trigeminal artery (PTA) is the most frequent embryonic communication between the carotid and vertebro-basilar system. PTA is a pathophysiology phenomenon which has been implicated as a rare cause of cranial nerve dysfunction. We report the case of a 40-year-old woman who developed a complete oculomotor nerve palsy caused by a persistent ecstatic trigeminal artery. Brain MRI and MRA studies documented a neurovascular conflict between the oculomotor nerve and a PTA. To the best of our knowledge there is no report about complete third cranial nerve palsy NC due to a PTA. A role of this rare vascular condition is discussed.

**Keywords** Persistent trigeminal artery · Oculomotor palsy · Neurovascular conflict

## Introduction

Persistent primitive carotid–basilar anastomoses are embryological vascular remnants and are uncommon [1]. Primitive trigeminal artery (PTA) is the most frequent type of persistent carotid–basilar estimated in various angiographic studies to be between 0.1 and 0.7% [2]. The first angiographic description of PTA was provided by Sutton in 1950 [3]; since then many radiological investigations have demonstrated that PTA and their variants have been identified as a rare cause of cranial nerve dysfunctions [4, 5] and associated with various vascular pathologies: carotid cavernous fistula, basilar hypoplasia, cerebral aneurysms, infra-tentorial arteriovenous malformation, and other rare conditions, such as Moya-Moya disease [6, 7]. The PTA originates from the cavernous segment of the internal carotid artery (ICA) and communicates with the basilar artery. The trigeminal arteries are first seen in the 3-mm embryo, and communicate with the fragments of the neural arteries during the 4-mm stage. The basilar artery becomes evident in the 7–12 mm stage through the union of the longitudinal neural arteries, and the trigeminal artery starts involution at the same time and finally disappears in the 14 mm stage [8]. We reported a rare case of a patient who developed complete oculomotor palsy as a sole manifestation of the third nerve contact with a PTA.

## Case report

A 42-year-old who had positive history for unstable hypertension was treated with calcium-antagonist for about 5 years. At the age of 40, in course of pregnancy, she developed signs of right third nerve dysfunction. The neurological examination showed a complete right

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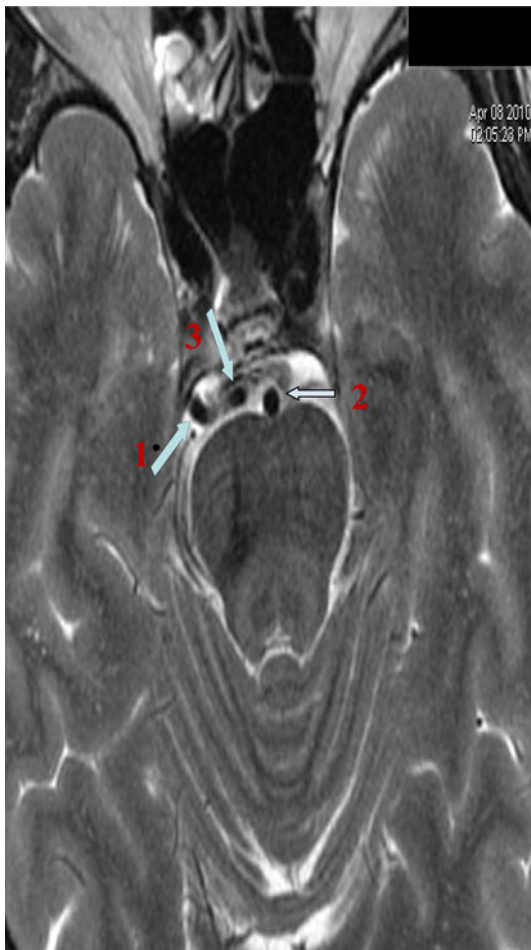
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oculomotor palsy; mydriatic pupil was present in the right eye. Orthoptic evaluation showed eso-hypertropia on the right (Cover Test), right third palsy (Hess Lancaster Test) including ipsilateral ptosis. No cranial nerve, motor or sensitive limb deficits were found. Mydriatic pupil was present in the right eye. The electrophysiological study, single fiber electromyography (SFEMG), acetylcholine receptor antibody titers, thyroid function, glucose and insulin values at fasting, and 2-h oral glucose tolerance test (2h-OGTT), respectively, HgA1c and Insulin resistance (IR), were negative [9]. A cranial CT scan was normal and excluded a subarachnoidal bleeding. The magnetic resonance imaging (MRI) and Angio-MRI (MRA) showed a right persistent trigeminal artery characterized by a moderate ectasia in its proximal tract (Fig. 1a, b). The utilized magnet was a 3T GE MR 750 discovery (Fig. 2).



**Fig. 1** The course of the third cranial nerve (1) was obtained with MPR reconstruction from volumetric sequence T2; the superior cerebellar artery (2) is arranged on the left side of the third nerve, while the ascending trait of the trigeminal artery (3) is adjacent on the right side of the oculomotor nerve



**Fig. 2** The angio-MRA showing a right ecstatic persistent trigeminal artery (1) and left hypoplastic posterior communicating artery (2)

## Discussion

Primitive trigeminal artery (PTA), like the optic, hypoglossal, and pre-atlantal variants, is a fetal anastomosis between the carotid and vertebro-basilar systems. PTA originates just before or at the point where internal carotid artery enters the cavernous sinus. As PTA leaves the carotid, it runs extradurally below the oculomotor and trochlear nerves and medial to ophthalmic branch of trigeminal nerve. Finally, PTA passes under petroclinoidal ligament or perforates the dura near the clivus to join the basilar artery between superior or anterior inferior cerebellar artery. In contrast, the PTA originates from the ICA and enters the posterior fossa either from Meckel's cave or from an isolated dural foramen, directly supplying the cerebellum without anastomosing with a basilar artery [2, 10]. These relationships may explain the reported cranial syndromes related to PTA persistence. Anatomical and angiographic studies suggested that trigeminal neuralgia and abducens nerve palsy might result from anomalous PTA [4, 5]. Conversely, literature has reported only one case of PTA associated with recurrent craniofacial pain and non-concomitant strabismus by partial ipsilateral oculomotor palsy [11]. We reported the case of a patient who developed complete oculomotor palsy as the sole manifestation of the third nerve neurovascular conflict (NC) with a PTA. Our case is characterized by a supposed contact between the third cranial nerve and the ascending

trait of the PTA, as it is visible in the MPR reconstruction from volumetric sequence T2. From its origin the artery is first positioned lower and then reached the basilar artery. Near to the anastomosis the basilar artery was hypoplastic (variant I of Saltzman). The posterior communicating artery was not visible. In Fig. 1 the course of the third cranial nerve was pointed out (it was obtained with MPR reconstruction from volumetric sequence T2): the superior cerebellar artery was arranged on the left side of the third nerve, while the ascending trait of the trigeminal artery was adjacent to the right side of the oculomotor nerve. From a pathogenetic point of view, we hypothesized that the oculomotor involvement responsible for the complete ophthalmoparesis may be assumed to be secondary to the pulsating action exerted by the artery, generating an intermittent mechanical stress. Persistent primitive arteries must be kept in mind as a causative agent of certain cranial nerve dysfunctions. Spontaneous recovery of cranial disorder due to the vascular compression has occasionally been encountered. In such cases, cranial nerve dysfunction could be caused by the transient increase of the flow of the vessel. Besides, in our patient, history of unstable hypertension during pregnancy may favor this hypothesis. From a diagnostic point of view, the detection of NC is based on conventional brain MRI and, in particular, MRA and thin-section T2-weighted MRI studies, which are the first choice techniques that allow a careful definition of PTA and its relationships. From a therapeutic point of view the microvascular decompression is the most utilized technique. Fukuda et al. [12] described a case of a 69-year-old woman who presented with trigeminal neuralgia caused by tortuous vertebrobasilar artery associated with PTA. Yamada et al. [13] described a case of a 31-year-old man presented with typical trigeminal neuralgia caused by an anomalous variant type of anterior inferior cerebellar artery (AICA) directly branching from the persistent PTA. Microvascular decompression surgery disclosed the trigeminal nerve compressed by this AICA variant together with the superior cerebellar artery. In our case we did not consider microvascular decompression because the third cranial nerve deficit lasted 7 years, so we did not prospect a real benefit from the intervention.

**Conflict of interest statement** The authors report no conflict of interest.

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