Persistent Trigeminal Artery Detected by Conventional Angiography and Magnetic Resonance Angiography

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Objective: A persistent trigeminal artery (PTA) may be found incidentally on conventional cerebral angiography and magnetic resonance(MR) angiography. Our goal is to examine the course and relationships of the vessel to the surrounding structures.

Methods: Cerebral angiography was performed in 494 patients and MRA in 880; the patients had or were suspected to have cerebrovascular disease. In the images, the incidence, origin, course, and relationships of the PTA were evaluated.

Results: A PTA was found in two (0.4%) of the patients undergoing cerebral angiography and three (0.34%) receiving an MR angiography. In four patients, the PTA arose from the lateral part of the cavernous segment of the internal carotid artery, then passed caudally and around the base of the dorsum sellae. In the other patient, the PTA arose from the medial aspect of the sphen, and ascended sharply to pierce the dorsum sellae and join the basilar artery. In four cases, there was hypoplasia of a proximal basilar artery below the abnormal communication; the vessel was of increased diameter above the communication.

Conclusion: Identification of a PTA with a trans-sellar course is crucial if trans-sphenoidal surgery is planned. Hypoplasia of a proximal basilar artery should not be mistaken for an acquired narrowing.

KEY WORDS: Trigeminal artery · Cerebral angiography · Magnetic resonance angiography.

Introduction

A persistent trigeminal artery(PTA) is the most cephalic and frequent type of persistent carotid-basilar arterial anastomosis. In cerebral angiographic studies, a PTA was present in 0.1% to 0.6% of large series of patients. The angiographic patterns of PTA are well known, but the exact relationships between the PTA and surrounding structures cannot be appreciated in cerebral angiographic images. In this in vivo study, we used high-resolution magnetic resonance imaging, magnetic resonance(MR) angiography, and conventional angiography, to demonstrate the incidence, origin, course and relationships of the PTA.

Materials and Methods

Conventional cerebral angiography (Philips V-5000, Philips Medical Systems, Eindhoven, Netherlands) was performed in 494 of the patients (between July 1997 and June 2004) and cranial MR angiography (1.5T, Signa MR/i, General Electric, Milwaukee, WI, USA) in 880 (between May 2001 and June 2004). Both cerebral angiography and MR angiography were performed in 66 of the patients. The MR angiography and angiography were undertaken for a variety of clinical reasons, including symptoms of cerebral ischemia, cerebral infarction, hemorrhagic contusion, intracranial hemorrhage, and headache. For the MR angiography studies, a three-dimensional time-of-flight(ToF) technique with a neurovascular phased array coil (MRI devices, Milwaukee, Wis., USA.) and a multiple overlapping thin slab acquisition technique were used. The following imaging parameters were selected: repetition time = 30ms; echo time = 6.9ms; field of view = 26 × 26cm; number of slices = 108-112; slice thickness = 1.6mm; slab thickness = 24-28mm; imaging matrix = 256 × 192; number of excitation = 1. No intravenous paramagnetic contrast agent was administered to any of the patients. In each patient, a total of 20 maximum-intensity projection(MIP) images in the frontal view (both from left lateral to right lateral, 180° and craniocaudally 180°) were routinely displayed stereoscopically. The images were obtained either from a routine diagnostic study or from the initial diagnostic part of an interventional procedure. All the angiograms and MR angiography were evaluated retrospectively.
for cerebral arterial anatomic variations by one of the authors (MS Kim). In patients with PTA, special attention was given to defining the origin and size of the anomalous vessel as well as its course. We also recorded the presence of associated vascular lesions, including a cerebral aneurysm, and a vascular stenosis or occlusion.

Results

A PTA was detected incidentally in images of five patients (three men, two women; 21 to 73 years old; median age = 66 years). Cerebral angiography showed the PTA in two (0.4%) of the 494 patients. In the three (0.34%) of the 880 patients, the PTA was visualized by MR angiography. The vessel was absent in 66 patients receiving both a conventional cerebral angiogram and a MR angiography. The diameter of the PTA was 3 to 4 mm. An aneurysm, an internal carotid artery stenosis, a fenestration of a middle cerebral artery, and a middle cerebral artery occlusion were associated with the PTA (Table 1).

The PTA originated from the left internal carotid artery in four patients and from the right artery in one. In four patients, the PTA arose from the lateral part of the cavernous segment of the internal carotid artery, and then passed caudally around the base of the dorsum sellae (Fig. 1). After a short cisternal course, medial to the trigeminal nerve, it joined the basilar artery. In the other patient, the PTA arose from the medial aspect of the siphon and passed caudally within the pituitary fossa, in close contact with the gland. It then ascended sharply to pierce the dorsum sellae, passed across the ambient cistern, and joined the basilar artery (Fig. 2). In four patients, hypoplasia of the proximal basilar artery was demonstrated below the abnormal communication; the vessel was of increased diameter above the communication (Fig. 3).

Discussion

PTA is the most frequently encountered persistent carotid-vertebrobasilar anastomosis. It is usually discovered
incidently at autopsy, or during a radiological examination (cerebral angiography, routine MR imaging or MR angiography). However, isolated cases of oculomotor paresis and trigeminal neuralgia have been reported as suggestive signs of PTA. PTA has been associated with other anomalies of intracranial vessels, including asymmetry (with aplasia or hypoplasia of the posterior communicating artery) and aneurysms of the circle of Willis, and aneurysms of the trigeminal artery.

In cerebral angiographic and MRA observations of four patients, we found an association between the PTA and hypoplasia of the proximal basilar artery. The trigeminal artery is the first connection between the primitive internal carotid arteries and the rudimentary paired longitudinal neural arteries, which ultimately fuse (by the 8-mm embryo stage) to form the basilar artery. Following fusion, the posterior communicating arteries develop as the primary connection between the termination of the basilar artery and the internal carotid arteries. Subsequent development normally leads to regression of the trigeminal artery. The occasional presence of the PTA in adult life mirrors the 11–14 mm embryonic stage, when essentially all midbrain arteries are supplied by the trigeminal branch of the internal carotid system and the basilar artery (causal to the trigeminal artery anastomosis). At this stage, one or both vertebral arteries remain relatively small. The PTA supplies blood to the posterior and superior cerebral arteries via the distal basilar arteries, and when it persists there is no flow-related stimulus for the basilar artery (proximal to the anastomosis) to develop in association with the growth of the embryo. Thus, there is frequently an association between a PTA and hypoplasia of the basilar artery.

Hypoplasia of the basilar artery is important because it suggests the presence of a PTA (it can be considered an ancillary sign of PTA) and it may indicate an important functional contribution of the anterior circulation to the posterior circulation, via the PTA. In these patients, iatrogenic or spontaneous occlusion of the carotid artery could cause infarction of the upper brainstem. Therefore, interventional neuroradiological or neurosurgical procedures may need to be appropriately modified. In addition, hypoplasia of a proximal basilar artery should not be mistaken for an acquired narrowing.

A PTA originates from the internal carotid artery, proximal to the meningo-hypophyseal trunk, at the point where it leaves the carotid canal and penetrates the cavernous sinus. The usual course is from the postero-lateral wall of the internal carotid artery to the posterior dural surface of the cavernous sinus, where it lies medial to the ophthalmic branch of the trigeminal nerve and around the dorum sellae. In other cases, the PTA originates from the postero-medial aspect of the internal carotid artery and has an intrasellar course in close contact with the pituitary gland. The artery ascends from the sellar floor and pierces the dorum sellae. In our study, one PTA with an intrasellar course was shown by conventional angiography and computed tomographic image (Fig. 2). We found that the course of PTA could be clearly determined by MR angiography, as the vessel could be visualized from many different angles; such images cannot be obtained by cerebral angiography. It is important for neurosurgeons, operating on or near the sella turcica, to know the course of a PTA.

The clinical significance of this anomalous vessel has not been fully discussed, but a PTA may be important in the following situations. Firstly, there is the risk of hemorrhage, followed by ischemia, if the PTA is manipulated during a surgical approach to the parasellar region. Secondly, there is the potential risk of cerebellar ischemia following therapeutic or spontaneous occlusion of the carotid artery. Finally, there is risk of hemorrhage related to complications during a percutaneous gasserian ganglion procedure for the treatment of trigeminal neuralgia, because the PTA passes very close to the Meckel's cave.

Conclusion

PTAs are rarely observed on conventional cerebral angiograms and cranial MR angiography. Although they hold little clinical significance, knowledge and recognition of a PTA is important during trans-sphenoidal surgery and a surgical approach to the parasellar region.

References

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