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MRI of basilar artery hypoplasia associated with persistent primitive trigeminal artery

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Abstract We report three patients with persistent trigeminal arteries, in all of whom the proximal basilar artery was hypoplastic. We draw attention to this common observation, which should not be mistaken for acquired narrowing.

Key words Basilar artery · Trigeminal artery · Congenital variants

Introduction

Several reports have documented demonstration of a persistent primitive trigeminal artery (PPTA) using magnetic resonance (MR) [1–4]. The associated basilar artery hypoplasia was discussed in one case [2] but not emphasized. Recently, six instances of imaging the PPTA by MR angiography (MRA) have been reported [5–7]. In one report, an associated hypoplastic proximal basilar artery was described [5] and in another case the finding was illustrated but not discussed [6]. We add three further cases of basilar artery hypoplasia associated with PPTA to the literature to emphasize that basilar artery hypoplasia is frequently associated with PPTA [8], can be recognized on MRI, and should be interpreted as an associated congenital variant, not atherosclerotic stenosis or occlusion of the basilar artery, and to suggest that MRI and MRA may be sufficient for diagnosis and confirmation of basilar artery hypoplasia associated with PPTA.

Materials and methods

MRI studies were acquired using T1- and T2-weighted spin-echo sequences at 1.5 T. MRA was performed in case 2 using a sequential 2D time-of-flight (TOF) method with parameters of 45/8.7/1 (TR/TE/exications), 60° flip angle, and 256 × 128 matrix with a 20 cm field of view (FOV) and 1.5 mm contiguous slices. In case 3 multiple overlapping thin slab acquisition (MOTSA) TOF MRA [7] using parameters of 30/3.8/1 (TR/TE/excitations), 30° flip angle, 256 × 128 matrix, with a 20 cm FOV were performed. Post-processing with a maximum-intensity projection (MIP) alogrithm was done to provide three-dimensional stacked or two-dimensional collapsed reprojection images. Conventional angiography was performed in all three cases.

Case reports

Case 1

A 60-year-old diabetic woman presented with recurrent episodes of left arm numbness. MRI revealed an acute watershed infarct between the right middle and posterior cerebral arteries. A normal-diameter basilar artery with flow void could not be seen at the midpons level (Fig. 1 a), suggesting occlusion. An aberrant vessel with flow void was seen connecting the right internal carotid and distal basilar arteries, representing a PPTA (Fig. 1 a, b). Angiography confirmed the PPTA (Fig. 1 c), and revealed atherosclerosis of

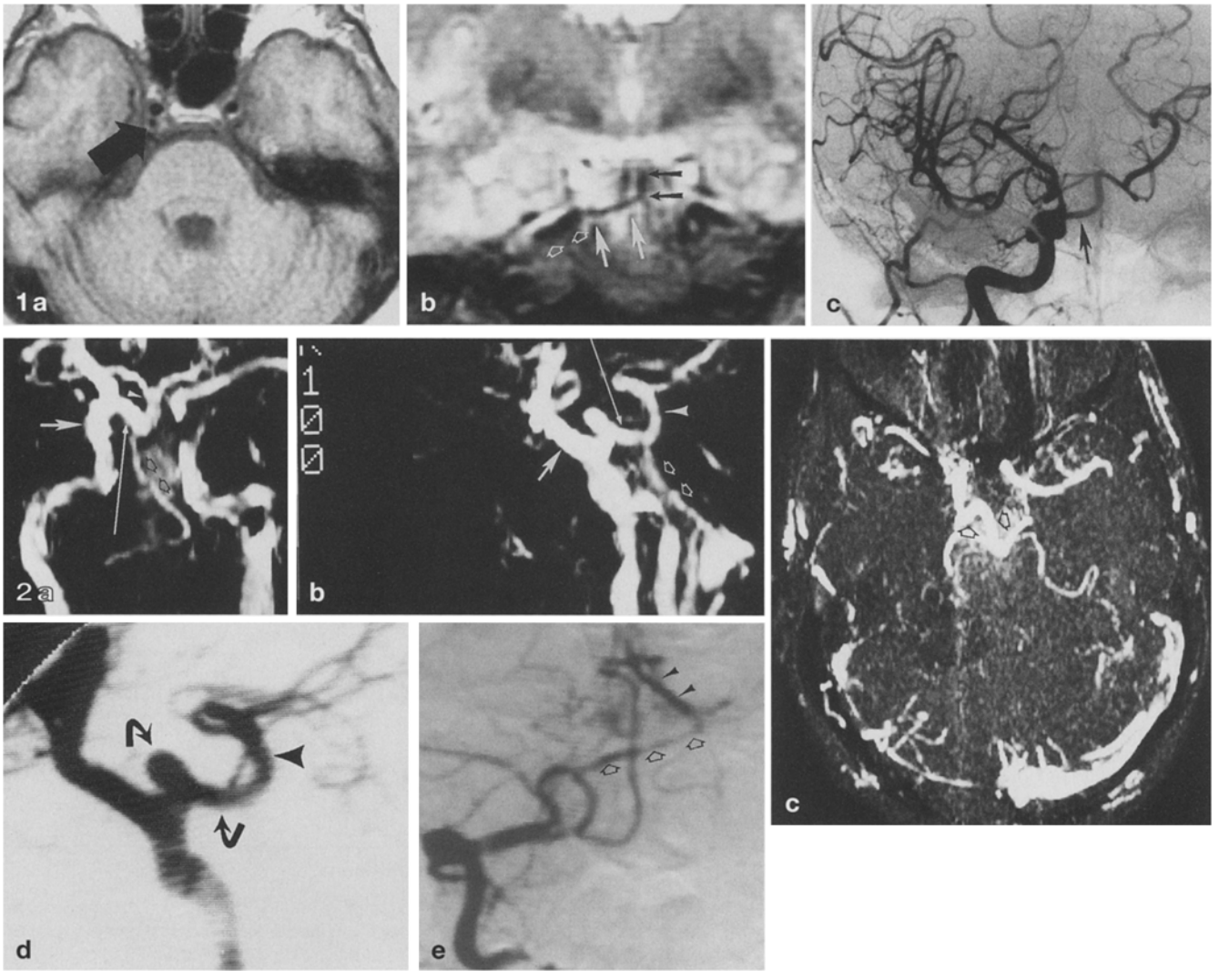

Fig. 1 **a** Axial 500/200 MR. The persistent primitive trigeminal artery (PPTA) can be seen dorsally in the cavernous sinus *(arrow)*. A normal basilar artery cannot be identified in the pontine cistern; it is atretic. **b** Coronal 2500/80 MR shows the anomalous PPTA *(large white arrows)* connecting the internal carotid *(open arrows)* and the mid-basilar arteries *(black arrows);* the basilar artery is not seen proximally. **c** Right common carotid angiogram demonstrates the PPTA *(arrow)*. A tight stenosis is present in the right supraclinoid segment of the internal carotid artery

Fig. 2 AP (**a**) and lateral (**b**) 3D MIP of 2D TOF MRA shows carotid *(short arrow)*, distal basilar *(arrowhead)*, persistent primitive trigeminal *(long arrow)* and hypoplastic proximal basilar *(open arrows)* arteries. **c** 2D TOF MRA MIP demonstrates the course of the PPTA *(open arrows)*. Occlusion of the right transverse sinus was related to the patient's tenth nerve schwannoma. **d** Selective internal carotid angiogram, lateral view, confirms the PPTA *(arrows)* connecting it to the basilar artery *(arrowhead)*. **e** Selective right vertebral angiogram shows distal *(arrowheads)*, and hypoplastic proximal *(open arrows)* basilar artery

the supraclinoid internal carotid artery (ICA). Both vertebral arteries were small and ended as posterior inferior cerebellar arteries.

Case 2

A 21-year-old man presented with headaches, dizziness, and difficulty in swallowing. MRI revealed a large right tenth nerve schwannoma with intracranial extension. MRA was performed to assess patency of the venous sinuses. A large PPTA, seen initially on axial spin-echo images, was well demonstrated by MRA (Fig. 2a–c). Proximal basilar artery hypoplasia was noted (Fig. 2a, b). Right common carotid angiography confirmed a large right PPTA (Fig. 2d), proximal basilar artery hypoplasia was confirmed on vertebral angiography (Fig. 2e). MRI 2 months after surgery again demonstrated proximal basilar artery hypoplasia. There were no brain stem ischemic changes.

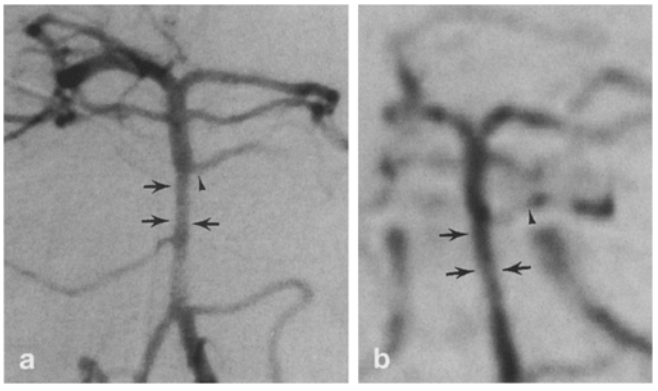

Fig. 3 **a** Vertebral angiogram demonstrates reflux into the PPTA *(arrowhead)* and subtle proximal basilar artery hypoplasia *(arrows)*. **b** AP MOTSA MRA 3D MIP reprojection shows saturation of flow in the PPTA *(arrowhead)* and subtle proximal basilar artery hypoplasia *(arrows)*

Case 3

A 24-year-old woman presented with sudden headache and acute subarachnoid hemorrhage. Conventional angiography demonstrated reflux into a left PPTA with mild proximal basilar artery hypoplasia (Fig. 3a). No aneurysm or vascular malformation was identified. MRI 2 days later showed hemorrhage in the pontine cistern, the left PPTA as a flow void and subtle hypoplasia of the proximal basilar artery as compared to the distal segment but with a complete flow void on T2-weighted images. There was no ischemic change in the pons. MOTSA MRA again demonstrated the subtle proximal basilar artery hypoplasia (Fig. 3b). No aneurysm was seen.

Discussion

Demonstration of a persistent primitive trigeminal artery (PPTA) on MRI was first reported by Richardson et al. [1]. Fortner and Smoker demonstrated basilar artery hypoplasia in a case complicated by a thrombosed aneurysm [2]. In a recent review of MRI in 11 cases of PPTA associated proximal basilar artery was not discussed [4]. Three cases of PPTA imaged by 3D TOF MRA have been reported [5, 6] but specific reference to basilar artery hypoplasia was made in only one [5].

Our three cases confirm the association of proximal basilar artery hpyoplasia with PPTA and illustrate that this can be identified on MRI. It is important to recognize the basilar artery narrowing and possible lack of flow void on T2-weighted images as being hypoplasia, a congenital anomaly. In case 1, atherosclerotic disease of the basilar artery secondary to diabetes was part of the clinical differential diagnosis prior to MR and conventional angiography, but the watershed infarction was more due likely hypoperfusion of the middle cerebral artery due to narrowing of the supraclinoid ICA.

Wollschlaeger and Wollschlaeger [9] documented 134 cases of PPTA on conventional angiography and added two of their own. They reported proximal basilar artery hypoplasia in both cases (one with autopsy proof) and stated that this was common in the cases they reviewed, although an exact frequency was not given [9]. In his definitive monograph, Lie [8] indicated that the vertebral and basilar arteries are "generally hypoplastic", and our experience, as well as our review of the literature suggests that the incidence is close to 100 %, with a wide spectrum of severity.

The trigeminal artery is the most common of the primitive anastomotic carotid basilar connections, autopsy and angiographic studies documenting an incidence of 0.1–1.0 % [9]. 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 [10]. Once fusion occurs, the posterior communicating arteries develop into the primary connection between the termination of the basilar artery and the internal carotid arteries. Subsequent development normally leads to regression of the PPTA. The occasional presence of the PPTA in adult life mirrors the 11–14 mm embryonic stage [10], when essentially all midbrain arteries are supplied by the trigeminal branch of the internal carotid system and the basilar artery caudal to the trigeminal artery anastomosis. At this stage, one or both vertebral arteries remain relatively small. The primitive trigeminal artery supplies blood to both posterior cerebral arteries and superior cerebellar arteries via the distal basilar artery and when it persists there is no flow-related stimulus for the basilar artery proximal to the anastomosis to develop along with the embryo. This observation explains the frequent association of basilar artery hypoplasia with PPTA.

In most cases, MRA demonstrates the basilar hypoplasia and its relation to the PPTA better than carotid on vertebral angiography alone, as in case 1, which had the most significant hypoplasia. In case 3, MRA and conventional angiography were nearly equivalent, with conventional angiography displaying the hypoplasia slightly better.

Blatter et al. [7] reported consistent visualization of intracranial vessels at least 0.9 mm in diameter; they described, but did not illustrate two PPTA [7]. Vertebral conventional angiography was performed in one case confirming the significant basilar artery hypoplasia shown by MOTSA MRA (D. Blatter, personal communication).

References

1. Richardson DM, Elster AD, Ball MR (1989) Intrasellar trigeminal artery. AJNR 10: 205
2. Fortner AA, Smoker WRK (1988) Persistent primitive trigeminal artery aneurysm evaluated by MR imaging and angiography. J Comput Assist Tomogr 12: 847–850
3. Shintani S, Kuzuhara S, Toyokura Y (1990) MR imaging of persistent primitive trigeminal artery. Neuroradiol 32: 79
4. Silbergleit R, Mehta BA, Barnes RD, Patel SC, Hatfield MK, Spickler EM (1993) Persistent trigeminal artery detected with standard MRI. J Comput Assist Tomogr 17: 22–25
5. Masaryk TJ, Modic MT, Ross JS, et al (1989) Intracranial circulation: preliminary clinical results with three-dimensional (volume) MR angiography. Radiology 171: 793–799
6. Schuierer G, Laub G, Huk WJ (1990) MR angiography of the primitive trigeminal artery: report on two cases. AJNR 11: 1131–1132
7. Blatter DD, Parker DL, Ahn SS, Bahr AL, Robinson RO, Schwartz RB, Jolesz FA, Boyer RS (1992) Cerebral MR angiography with multiple overlapping thin slab acquisition, part II. Early clinical experience. Radiol 183: 379–389
8. Lie TA (1968) Congenital anomalies of the carotid arteries. Excerpta Medica, Amsterdam, pp 53–70
9. Wollschlaeger G, Wollschlaeger P (1964) The primitive trigeminal artery as seen angiographically and at postmortem examination. Am J Roentgenol Radium Ther Nucl Med 92: 761–762
10. Padget DH (1948) The development of the cranial arteries in the human embryo. Contrib Embryol 32: 205–261

BOOK REVIEW

Malignant Brain Tumours.
Eds.: D. G. T. Thomas, D. I. Graham. Berlin, Heidelberg, New York: Springer 1995, (ISBN 3-540-19689-7), hardcover, DM 238.00.

The title of this book is *Malignant brain tumours,* yet the text for a major part is concerned with tumours of the central nervous system in general. There are 14 chapters written by 28 international authors, which may account for some of the diversification from the central theme.

The first two chapters concern cellular and genetic aspects of tumours and the third is an excellent review of the histopathological classification. There then follow chapters dealing with the clinical presentation, complications and management of tumours in adults and children and the specific problems found with AIDS and metastatic lesions. Curiously positioned next is the chapter on radiological diagnosis, interposed between the chapter dealing with surgery of primary malignant brain and the other management chapters.

The neuroradiology chapter deals with the general CT and MR appearances of the characteristics which make up the elements of tumour structure, localisation and mass effect and the specifics of many tumour types. The differential diagnosis of mass lesions is covered but many lesions, identified by the authors themselves as being "unlikely" or "rarely" confused with tumours e.g., colloid cysts, cysticercosis, hydatid disease and progressive multifocal leucoencephalopathy are given a paragraph each and the common differentials of ischaemia, haemorrhage, abscess and encephalitis are given no greater discussion. Angiography is given consideration in many subgroups of tumours but positron emission tomography (PET) is dismissed with a one line comment on a par with the value of skull radiographs.

By now the reader has become aware that the overall text of this book, published in 1994, must have been written much earlier. There is only a smattering of references from 1990 and a handful from 1991 in the whole of this extensively referenced text. This is unfortunate because a comprehensive review of malignant brain tumours would be welcome to bring together the advances and basic sciences, cell biology, immunology and the increasing data available from PET and other isotope work. A closer adherence to the subject of malignant brain tumours would also serve to reduce reader confusion and irritation.

The chapters are laid out with well-identified headed subdivisions and a useful short conclusion is provided at the end of many chapters. The majority of the illustrations are clear although two of the scans are displayed upside down. The detail of the contents of the chapters makes it very easy to select an areas of interest. Overall it is easy to access and review specific points of interest. The large number and the physical separation of the authors perhaps contributed to a delay in producing the text and to the lack of focus on the theme of malignant brain tumours. While there are many good aspects to this book it cannot be recommended as an up-to-date state-of-the-art text.

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