Internal Carotid or Ascending Pharyngeal Artery

To The Editor: We read with great interest the recent article by Heth, et al. (Heth JA, Loftus CM, Piper JG, et al: Hypoplastic internal carotid artery mimicking a classic angiographic “string sign.” J Neurosurg 86:567–570, March, 1997). They described a patient affected by recurrent transient right-sided weakness, with duplex scanning evidence of occlusion of the right internal carotid artery (ICA). Angiography performed in the right common carotid artery (CCA) showed a thin vessel with a very linear course, located medially and posteriorly to an apparently normal external carotid artery (ECA). Right carotid siphon was not clearly apparent in these images; the right middle cerebral artery was filled by an enlarged posterior communicating artery, and the right anterior cerebral artery was filled by the anterior communicating artery. These findings were interpreted as being the result of a severe stenosis of the right ICA. Magnetic resonance studies showed the lack of a flow void in the cavernous left ICA, and hypointensity on the right side was interpreted as normal flow. Because the left ICA presented with stenosis greater than 70%, they planned a bilateral endarterectomy. During the operation, the authors discovered a thin vessel arising at the end of the right CCA corresponding to the thin linear vessel recognizable on the angiogram. In the Discussion section, the authors hypothesized that they were dealing with an hypoplastic ICA and pointed out that the angiographic findings were consistent with the alternative hypothesis of stenosis greater than 95% of a normally developed ICA. Moreover, they correctly underlined that an examination by computerized tomography (CT) of the carotid bone canal, whose dimensions are strictly dependent on the diameter of the ICA, would have suggested the correct diagnosis.

An angiogram obtained in this case (Fig. 2 of the Heth paper) indicates an alternative hypothesis. The course and the dimension of the “anomalous” vessel are very similar to that of a normal vessel arising at the end of the CCA, usually the first posteromedial branch of the ECA, the ascending pharyngeal artery (APhA), the ICA in this case being completely absent. The lack of adequate visualization of the carotid siphon in Fig. 2 of the paper by Heth and coworkers supports this hypothesis. In normal angiograms, the APhA is partially masked by the ICA, particularly in the lateral projection, whereas it appears more evident if the ICA is absent. This possibility would have been tested by a CT scan of the carotid bone canal, which is mandatory in those rare cases; moreover, the APhA is easily identifiable by carefully looking at the course, extremely linear and medially directed, and at the terminal branch, which can fill the siphon in some cases.

Aplasia of the ICA is a well-known condition, and the collateral circulation may involve a number of normal—or abnormal—vessels, as correctly noted in the authors’ discussion of the case. Most frequently, compensatory flow involves the posterior communicating artery, as occurred in this case. A rare anomaly, not mentioned by the authors, is a transsellar anastomosis. The practical question is whether a more accurate neuroradiological study would have avoided an unnecessary operation. In this context, a CT scan of the skull base, as correctly suggested by the authors, would have been useful; moreover, an examination of the intracranial vessels after contrast injection into the right CCA would probably have detected the relationship between extracranial vessels and carotid siphon.

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Response: We were interested to read the letter by Drs. Bergui and Bradac regarding our case report of a hypoplastic internal carotid artery (ICA). They suggest that the vessel we identified in Fig. 5 of our article is not an ICA at all, but rather the ascending pharyngeal artery (APhA) branch of the external carotid artery. Whereas this is a plausible hypothesis and we have no reason to state dogmatically that it is not so, the operative appearance both to the senior author and as demonstrated in the operative photograph are not consistent with our concept of the anatomy of APhA. It was our clear impression that we were dealing with a rather routine carotid bulb with a very minor and rudimentary ICA based on the usual anatomical pattern. In our experience, the APhA arises either from the crotch of the carotid bifurcation or, most commonly, from the posterior medial wall of the external carotid artery, as Drs. Bergui and Bradac suggest.

We are unable to resolve this controversy and suspect that it will remain a permanent curiosity and unanswered question. It is, however, our belief that we were correct in our assessment that this was an ICA and that this anatomical variant was consistent with the literature that we researched in the preparation of our case report.

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Hypermetabolism or Hyperglycolysis

To The Editor: This letter is in regard to a recently published paper (Bergsneider M, Hovda DA, Shalmon E, et al: Cerebral hyperglycolysis following severe traumatic brain injury in humans: a positron emission tomography study. J Neurosurg 86:241–251, February, 1997). The authors claim to have found cerebral hypermetabolism in acutely comatose patients, contrary to all previously reported studies in this area in which cerebral hypometabolism has been the general rule.

The methodology and technique used in the study by Bergsneider, et al., however, involved two totally distinct and not cross-validated measurements. Instead of simultaneously assessing global cerebral glucose and oxygen consumption obtained from arteriojugular measurements or cortical glucose and oxygen consumption obtained

References

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