

FIG. 2. A: CT scan. B: Schema—the cross hatched parts are displaced fragments from the orbital roof. The normal configuration of the orbital cave is single hatched.

parenchyma of the brain surrounded by hypodensity. A marked hyperdensity of the falx cerebri was interpreted as a congestion or thrombosis of the sinus sagittalis superior. We noted the presence of pneumoencephaly. More peculiar was the scan of the orbits (see Figs. 1 and 2). We noted an elongation and protrusion of the eyeballs. Loose fragments of bone from the orbital roof protruded down into both orbits, and the perinasal sinuses were filled with blood and fluid.

EXPLICATION OF THE EXPLOSION PHENOMENON

The meninges and the brain are protected from harmful external stimuli, including electric current, by the ossae calvariae and the skin. Prolonged contact with high voltage electric current causes burning of the skin and calcinosis or even fracture of the skull. The passage of electric current results in an input of energy into the brain with heating, edema, extravasation of blood, heating of the cerebrospinal fluid, and the production of steam.

The heating and steam production have an explosive effect and the weakest part of the skull may be disrupted, i.e., the laminae cribrosae and the orbital roofs. The displaced and fragmented orbital roof may protrude into the orbits along with brain substance. A similar case was reported by Fritz in 1941 (1).

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Intrasellar Intercarotid Communicating Artery Associated with Agenesis of the Right Internal Carotid Artery: Case Report

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A 54-year-old man with a right hemiparesis was found to have an intrasellar intercarotid communicating artery associated with agenesis of the right internal carotid artery. Magnetic resonance imaging (MRI) studies demonstrated the spatial relationship of the anomalous artery to the surrounding structures, thus suggesting an embryonic enlargement of the capsular artery as a source of this anomalous artery. (*Neurosurgery* 23:770-773, 1988)

Key words: Agenesis, Capsular artery, Collateral artery, Internal carotid artery, Sella turcica

Unilateral agenesis of the internal carotid artery is a rare anomaly. In the majority of reported cases, collateral circulation to the intracranial arteries is from the contralateral internal carotid artery and the vertebrobasilar system via the circle of Willis. Very rarely, blood is carried to the affected side through an anomalous artery between the cavernous part of the two internal carotid arteries. To our knowledge, only 10 cases have been reported in the literature, although none had magnetic resonance imaging (MRI) studies that demonstrated the spatial relationship of this anomalous artery to the surrounding structures. We report a case of unilateral agenesis of internal carotid artery associated with an unusual transsellar intercarotid connection being demonstrated by MRI.

CASE REPORT

A 54-year-old, right-handed man was admitted to our hospital with sudden onset of right motor weakness 2 months

before admission. His medical history revealed a subtotal gastrectomy because of early stage gastric cancer 14 years previously and diabetes mellitus for which he had been treated with sulfonyl ureas for 2 years. No other vascular risk factors were found.

Routine skull roentgenograms showed a depression of the sellar floor (Fig. 1). A cranial computed tomography (CT) scan revealed small low density areas in the left basal ganglia and the posterior limb of the left internal capsule. No other abnormalities were shown even in the postcontrast study including around the sella turcica. Arch aortogram with transfemoral digital subtraction angiography showed normal origins and courses of the left common carotid artery and the left vertebral artery. No other remarkable findings were present except for a relatively small-sized right common carotid artery. The transfemoral right carotid angiogram revealed no filling of the internal carotid artery and only the external carotid artery filled with no transdural collateral circulation. The left carotid angiogram seemed normal in its extracranial portion, but an anomalous artery, originating from the cavernous portion, served as the main collateral circulation to the right middle cerebral artery. The A₁ portion of the right anterior cerebral artery was not filled, and the left carotid



FIG. 1. Plain x-ray film of the sella turcica, lateral projection, showing an area of focal scalloping in the floor.

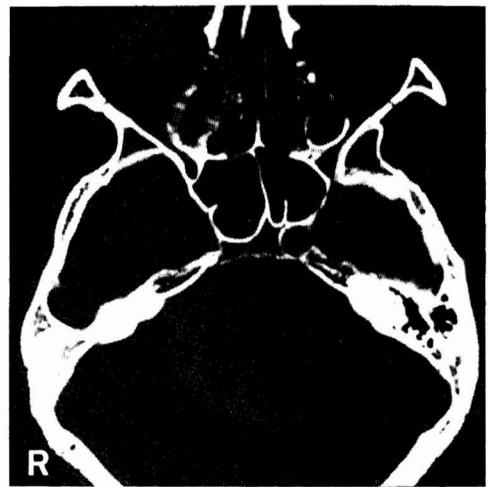


FIG. 3. CT scan of the skull base showing an absent right carotid canal and an enlarged left carotid canal.

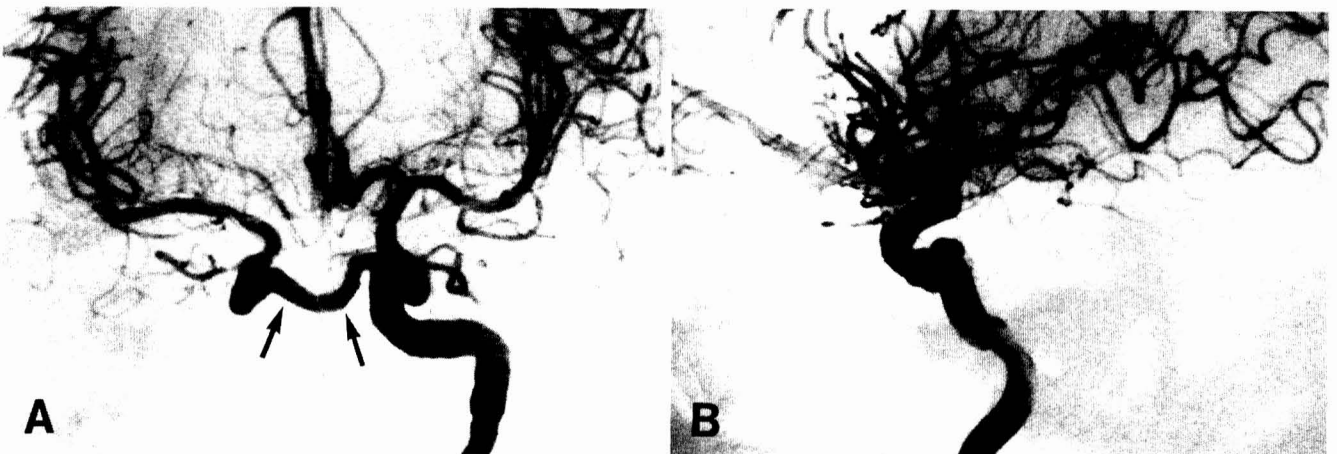


FIG. 2. Left carotid angiogram. The anteroposterior (A) and lateral (B) views demonstrate an anomalous artery (arrows) that originated from the cavernous portion and serves as the collateral circulation to the right middle cerebral artery.

artery provided bilateral anterior cerebral artery circulation (Fig. 2).

The vertebral angiograms showed normal configuration without filling of the posterior communicating and carotid arteries. Basal skull CT scan disclosed the absence of the right carotid canal and an enlarged left carotid canal, thus suggesting agenesis of the right internal carotid artery (Fig. 3). MRI was informative (Fig. 4). The T₁-weighted (TR/TE = 500/40, 0.15 Tesla) midsagittal view demonstrated a round low intensity area corresponding to the anomalous artery anteroinferior to the normal pituitary gland. The horizontal course of this artery underneath the pituitary gland was also clearly shown in the coronal view with the same imaging protocol.

DISCUSSION

This case demonstrated the absence of the right internal carotid artery with collateral circulation via an anomalous communicating artery between the intracavernous portion of the internal carotid arteries.

Lie was the first to describe this type of anomaly in an epileptic child (6), and there have been ten similar cases in the literature (1-6, 9-11). All of these anomalous arteries were found incidentally by conventional radiological studies. No case has been considered as a cause of neurological deficit. Our case had multiple small infarcts in the basal ganglia and internal capsule, showing right hemiparesis; however, no definite corresponding lesion was shown in the arch aortogram and left carotid angiogram. Furthermore, this anomalous artery did not seem to be a thromboembolic source because blood is carried to the side of cerebral infarct through the normal internal carotid artery. Although the pathogenesis of the cerebral infarcts remains unclear, diabetes mellitus may play a major role.

The embryogenesis of this anomaly is open to interpretation. Because agenesis of the internal carotid artery is a developmental anomaly that occurs before the embryo

reaches 24 mm (7), there is no question that this intercarotid anastomosis develops before the sixth week of embryological growth. Lie postulated that this anastomosis resulted from the fusion of two primitive trigeminal arteries because of its location near the trigeminal ganglion (6). In most of the reported cases, however, including ours (1, 3-5, 9-11), the intercarotid communicating artery extended across the sellar floor. Elefante et al. (1), described that the two primitive maxillary arteries were likely to remain and develop as an intercarotid anastomotic artery. According to the embryological studies of Padget (7), the primitive maxillary artery originates from the cavernous portion of the internal carotid artery lateral to the base of Rathke's pouch in the 3- to 4-mm embryo. Normally, this artery involutes, and only the stem remains by the 24-mm stage of embryonic growth. This stem is incorporated into the formation of the inferior hypophyseal artery.

In a study of over 200 cadavers, Parkinson reviewed the anatomy of the circulation in the area surrounding the sella turcica (8). There are some anastomoses between the two internal carotid arteries. The capsular arteries and inferior hypophyseal arteries are within the sella turcica. The remaining intercarotid anastomosis is found at a greater distance down the clivus. The anastomosis between the two inferior hypophyseal arteries occurs on the posterior surface of the posterior lobe of the pituitary gland, which formed the anterior part of the circulus arteriosus around the root of the dorsum sellae. The capsular artery leaves the inferomedial aspect of the internal carotid artery 2 to 3 mm anterior to the origin of the meningohypophyseal trunk and anastomoses with its counterpart extradurally within the sella turcica.

In this case, the spatial relationship of the anomalous artery to the surrounding structure was clearly shown by MRI. This artery is located anteroinferior to the normal pituitary gland in the sella turcica. Based on these MRI studies, embryonic enlargement of the capsular artery seems to be a more likely source of this anomalous artery than development of primi-

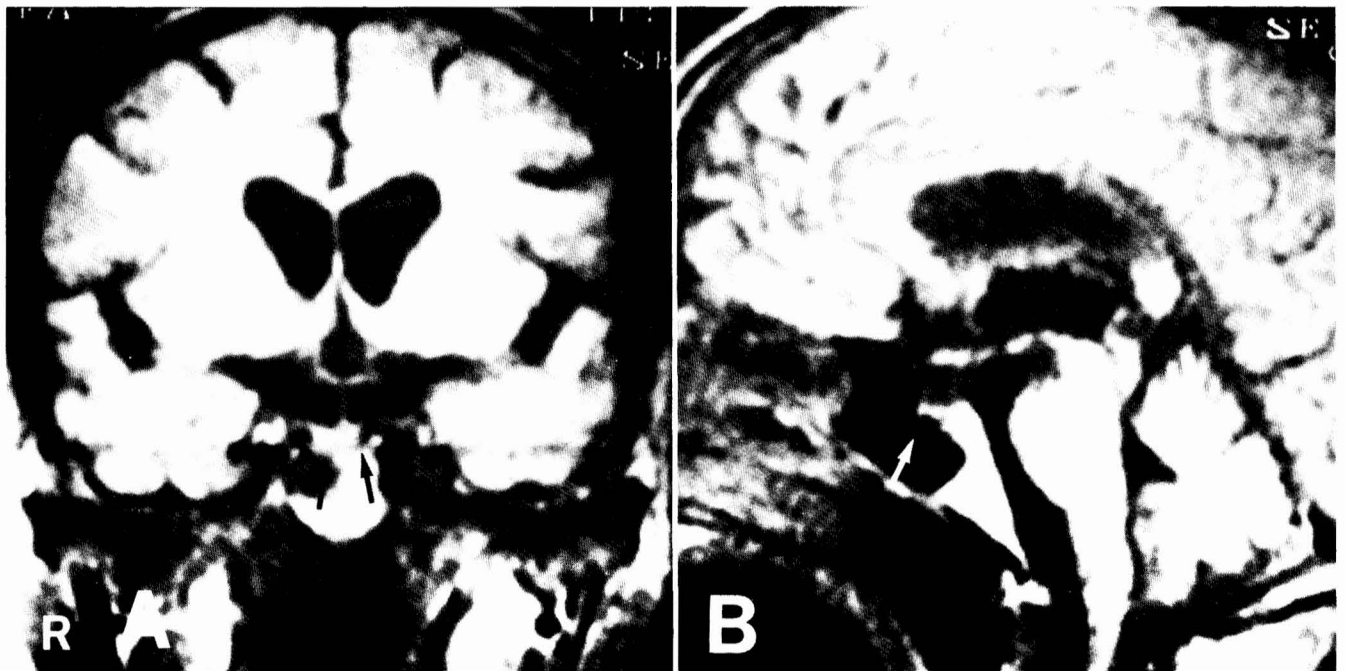


FIG. 4. Magnetic resonance images (TR/TE = 500/40, 0.15 Tesla). The coronal (A) and midsagittal (B) views demonstrate a low intensity area (arrows) corresponding to the anomalous artery anteroinferior to the normal pituitary gland.

tive maxillary artery or inferior hypophyseal artery as proposed by Janicki et al. (4).

Although the intrasellar intercarotid communicating artery is extremely rare, knowledge of the presence of this anomaly is important when considering cerebrovascular surgery or transsphenoidal pituitary surgery.

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COMMENTS

The authors present an unusual arterial anomaly that crosses through the sella. This case illustrates the adaptability of collateral circulation particularly during early development. The absence of a carotid canal is strong evidence to support an early developmental abnormality.

The authors give a strong argument in favor of the capsular artery as the source of this anomaly. The report also makes the neurosurgeon involved with transsphenoidal operations aware of the potential for arterial anomalies that could threaten the intracranial circulation if interrupted. It under-

scores the need to image the basic vascular anatomy surrounding the sella before operations with either the usage of MRI or angiography.

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The article by Udzura and colleagues provides an excellent description of a case of agenesis of the right internal carotid artery with a large intrasellar intercarotid communicating artery. The role of modern imaging techniques in the characterization of this rare anomaly is appropriately highlighted. Specifically, the CT scan of the base of the skull allows adequate evaluation of the bony carotid canal. The MRI provides complementary information by demonstrating the presence or absence of large vascular structures, which would appear as channels of signal void.

As in most previously reported cases of this rare entity, the anomaly was discovered incidentally. In this case, the patient presented with lacunar infarctions most likely unrelated to the vascular anomaly. Occasionally, agenesis of the internal carotid artery may become significant in the presence of contralateral hemodynamic lesions. Also, the abnormal collateral vessels may rarely serve as channels of embolism from contralateral ulcerated lesions.

We concur with the authors' anatomic description and embryological analysis of this anomaly. Because of its anterior inferior relationship to the hypophysis, it is unlikely that the major collateral artery represents connections between trigeminal, tentorial, maxillary, or inferior hypophyseal arteries. Instead, it is most likely a dilated connection between hypertrophied inferior capsular arteries. Such arteries are the most distal and least constant set of branches arising from the intracavernous internal carotid artery. They are found in slightly less than 30% of adult anatomic specimens and are known as McConnell's capsular arteries (Harris and Rhoton, *J Neurosurg* 45:169-180, 1976).

Another peculiar feature of this case is the incompetent anterior and posterior circle of Willis as demonstrated angiographically. The channels of the circle of Willis are typically hypertrophied in cases of agenesis of the internal carotid artery. In this case, the circle of Willis abnormalities must have antedated the intercavernous collateral channel embryologically and may have provided the hemodynamic incentive for the persistence of this primitive artery during development. One must not ignore another important potential source of collateral circulation to the right middle cerebral artery territory; i.e., the right ophthalmic artery. The authors mention the absence of transdural collaterals upon right external carotid artery injection, but do not mention the presence or absence of retrograde ophthalmic artery filling. This information would have been essential if there was any question about hemodynamic compromise.

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