

Malignant Cerebral Infarction Secondary to Internal Carotid Injury in Closed Head Trauma: Good Outcome with Aggressive Treatment

A Case Report

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A 25-year-old male patient in whom occlusion of the internal carotid artery developed secondary to a skull base fracture is presented. The diagnosis of internal carotid artery occlusion was reached 12 hours after the admission and 17 hours after the injury. The patient was initially treated for ischemic edema and when the patient showed signs of cerebral herniation, decompressive craniectomy was necessary. The outcome was good. The clinical and radiologic characteristics of internal carotid artery occlusion in closed head injury are highlighted and treatment options are reviewed in light of pertinent literature.

Introduction

Internal carotid artery (ICA) injury is a rare complication of closed head injuries first reported by Erikson in 1943.¹ Since then, less than 30 cases have been reported as of 1987² and only a handful of cases since then.³⁻⁶ The condition carries a significant mortality and, if the patient survives, significant neurologic sequela. Most often, the injury to the vessel occurs within the bony canals of the skull base but the upper cervical segment may also be injured by mechanisms other than

compression. The vessel wall injury will dictate the scope of the injury, which ranges from minute intimal injury to intimal-medial layer injuries and finally to complete transection. The vessel wall injury may then cause ICA dissection leading to partial or total ICA occlusion or aneurysm and fistula formation. When the blood supply through an artery as great as ICA is partially or totally impaired, the resultant risk of ischemia of the involved hemisphere (but particularly the middle cerebral artery territory) becomes a threat to an already injured patient's life. Reviews have shown that of all the previously reported cases, 60% to 65% died and 20% were left with a major neurologic deficit while only 15% recovered.^{1,7} The literature shows that even if a diagnosis was reached while the patient was still alive, treatment attempts were often kept at a conservative level, an approach similar to blunt carotid injuries in the neck.⁸

We hereby present a case of a young male patient in whom a malignant cerebral infarction developed secondary to post-traumatic right ICA oc-

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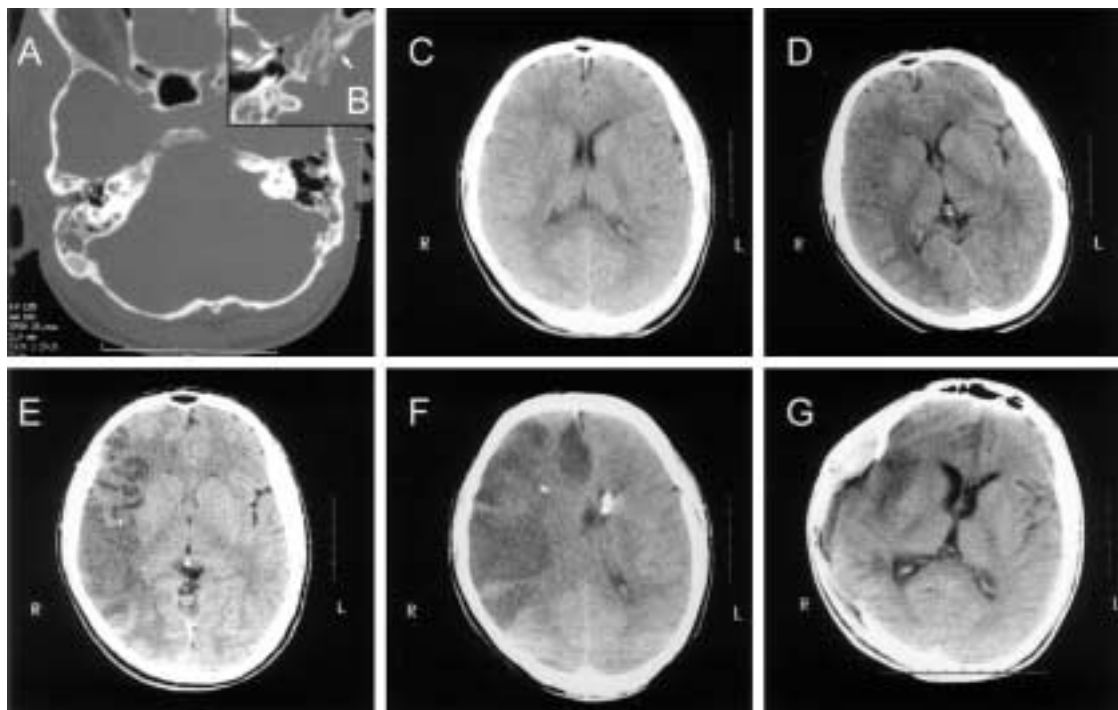
clusion and who was successfully treated with barbiturate coma, ventriculostomy, surgical decompression of the involved hemisphere when transtentorial herniation occurred, and mild hypothermia in the postoperative period.

Case History

A 25-year-old man was brought to our institution 5 hours after his head became accidentally squeezed between the metal fork of a forklift and the wooden supports it was about to carry. On the way to our A/E the patient vomited five times. On admission, he was fully conscious and orient-

ed with a full GCS score of 15. The neurologic examination was normal except limited abduction of the right eye. Cranial CT scan showed an oblique skull base fracture extending from the mastoid air cells to the petrous apex encompassing the carotid canal (Figure 1A). The anterior end of the fracture clearly crossed the foramen lacerum (Figure 1B). There was also a contralateral fracture in the left occipital region further proving the significance of the initial impact. The brain parenchyma was normal with normal ventricular and cisternal sizes (Figure 1C). Cervical computed tomography (CT) was normal. Four hours after hospital admission (9 hours after the event), GCS score dropped to 8 (E3M4V1) with sudden left-sided hemiplegia. An impending epidural hematoma was suspected, and an emer-

Figure 1. A. Cranial CT scan with bone window shows a right temporal fracture which extends from the mastoid tip to petrous pyramid. Note contralateral occipital fracture. B. The anterior end of the right temporal fracture crosses the carotid canal. C. Cranial CT scan on admission shows normal ventricular and cisternal sizes and normal parenchyma. D. CT scan 4 hours after admission shows slightly hypodense right hemisphere. The hypodensity does not correspond to a known arterial territory. Please note that the ventricular system is still symmetrical. E. CT scan 12 hours after admission shows clear-cut ischemia of the right hemisphere with some compression of the ventricular system and minimal midline shift. F. CT scan after the insertion of a left frontal EVD catheter shows malignant infarction of the right hemisphere. Please note that tissue density of the right occipital lobe is normal. G. CT scan 2 weeks after admission shows atrophy of the right hemisphere most marked around right insula with compensatory enlargement of the right lateral ventricle.

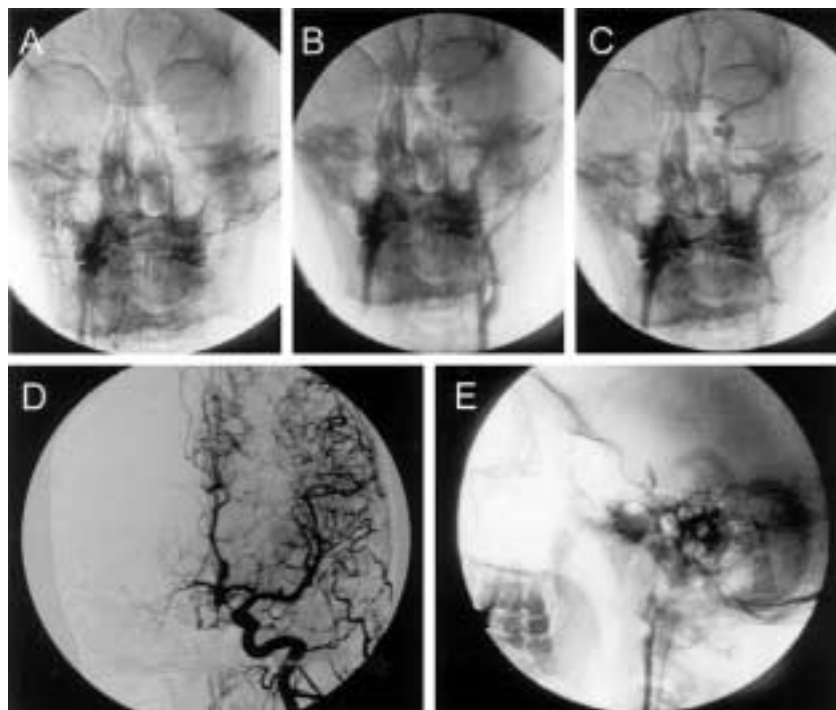


gent repeat CT scan was done. To our surprise, CT did not show any blood within the cranium (Figure 1D). The right hemisphere was slightly hypodense but there was no distortion of the ventricular system to suggest a localized pressure. Although the impact was neither a decelerating nor an accelerating injury, CT scans were initially interpreted to represent a mild contusion and antiedema treatment consisting of dexamethasone and low doses of intermittent mannitol were started. A CT scan obtained 8 hours later showed clear-cut hypodensity over the right middle cerebral artery (MCA) territory (Figure 1E). Cerebral angiography was performed and showed that no dye passed beyond the skull base, suggestive of complete obstruction of the right ICA at its entry point to the petrous bone (Figure 2A,E). Subsequent images showed that the contrast material initially injected into the right ICA was trapped within the wall of an ICA segment between the ICA origin and its point of entry into the skull

base (Figure 2B,C). This was interpreted as a sign of dissection. Subtraction images showed minimal collateral flow through the anterior communicating artery (Figure 2D).

Anticoagulation with heparin was immediately started. The patient was then intubated and mechanically ventilated under pentothal infusion. A parenchymal intracranial pressure (ICP) sensor was inserted through a right frontal drill hole. ICP and cerebral perfusion pressures were closely monitored (Figure 3) and when the ICP reached 40 mm Hg levels, an external ventricular drainage (EVD) catheter was inserted into the left frontal horn. CT scan showed malignant infarction of MCA with a midline shift (Figure 1F). On the 44th hour after admission, the ICP exceeded 40 mm Hg despite a functioning EVD and the right pupil became dilated and unresponsive to light. The patient was taken to surgery for decompressive craniectomy. A large fronto-parieto-temporal bony decompression was carried out and the dura

Figure 2. Cerebral angiogram, right ICA injection, (A) anteroposterior and (E) lateral views show that the dye rises up to but not beyond the point where the artery enters the skull base. Cerebral angiogram, left common carotid injection, (B,C) anteroposterior views show suspended dye within the right ICA wall from the point where the flow was obstructed initially all the way down to carotid bifurcation. There is good flow into the contralateral anterior cerebral artery via anterior communicating artery (D).



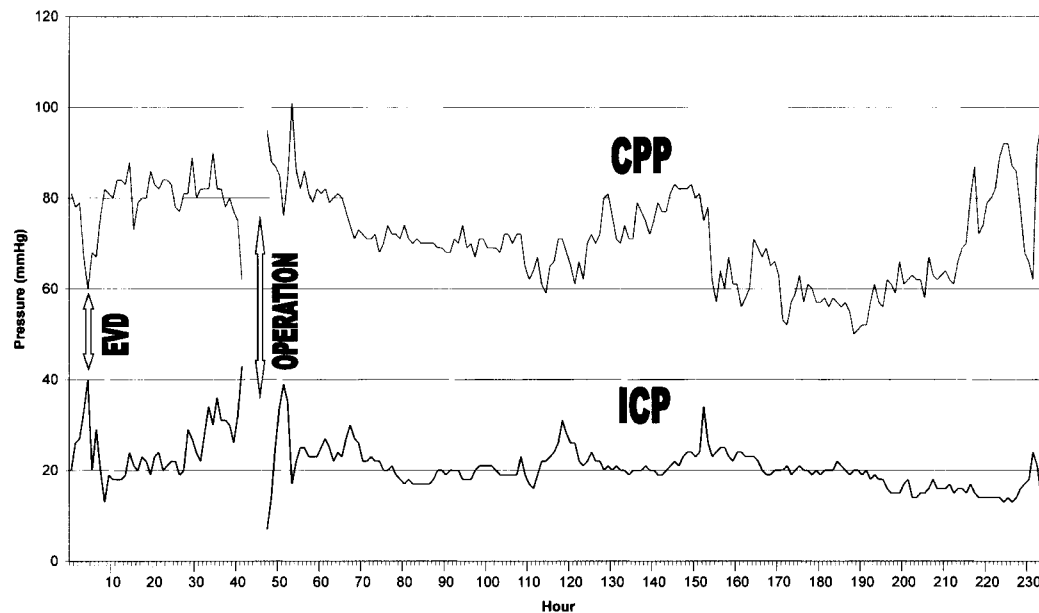


Figure 3. Graph depicts the intracranial pressure and cerebral perfusion pressure values of the patient with reference to therapeutic interventions.

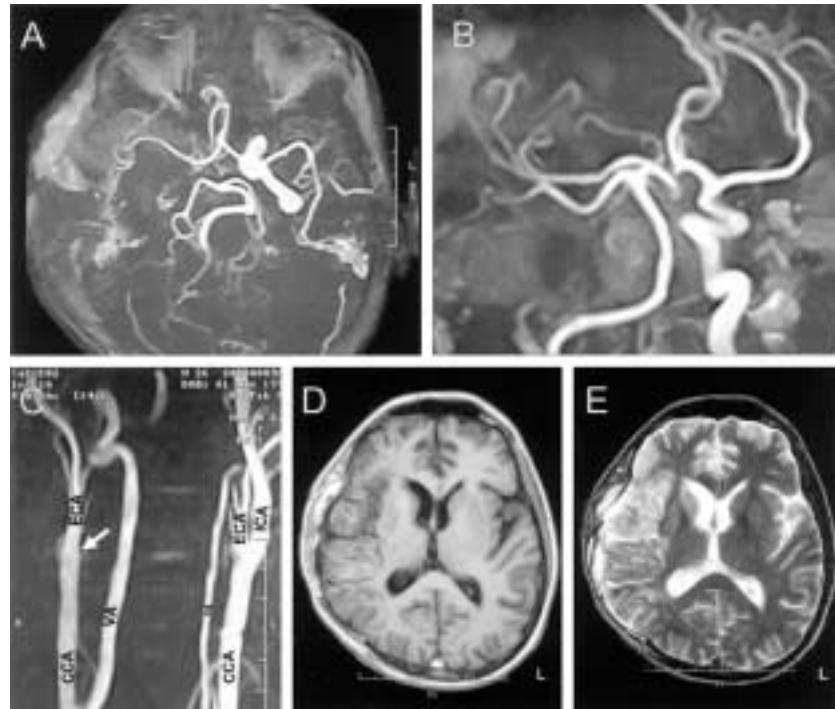
was repaired using a large fascia lata graft. Following surgery, the patient was cooled to 35°C. The barbiturate coma and hypothermia was maintained until the 7th day after the injury. Mannitol was continued until the end of 2nd week. A CT scan obtained on the 14th day showed that hypodensity related to ischemia had almost disappeared and that the midline shift had disappeared (Figure 1G). At the 3rd week after the injury, the GCS was up to 15. Magnetic resonance angiography of the cranial and neck vessels obtained a month after surgery showed total obliteration of the right ICA immediately distal to common carotid bifurcation (Figure 4A,B,C). Magnetic resonance imaging of the brain showed encephalomalacia of the right MCA territory (Figure 4D,E). At 2 months after trauma, the patient is cooperative, oriented to time, person, and place with normal speech and cognition. His left hemiparesis improved to a power of 3/5. A late carotid angiogram was done and did not show any aneurysmal or fistulous pathology. Angiograms did in fact show exactly what was demonstrated with MR angiography. The bone flap was replaced 4 months later.

Discussion

There are 4 possible mechanisms for blunt ICA injury.⁹ There are 2 mechanisms that apply to cervical ICA and 2 mechanisms for ICA segments other than the cervical segment. The cervical ICA may be injured after a sudden blow to the neck or by stretching due to hyperextension and rotation of the head to the opposite side (usually as part of a chiropractic manipulation). In either case, the distal cervical ICA is injured by tethering and stretching over the lateral masses of the upper cervical vertebrae. These 2 mechanisms are however beyond the scope of this article, which will focus on injury to the petrous or cavernous ICA secondary to a basal skull fracture with or without displacement of bone and briefly on injury of the intradural ICA.

The course of ICA within the skull base is more complex than its cervical or even supraclinoid course and deserves a brief review. In 1996, Bouthillier and associates proposed a new classification for ICA segments based on the direction of flow that we prefer. This classification subdivides ICA into 7 segments, from C1 to C7.¹⁰ The

Figure 4. MR angiography (A,B,C) shows no flow through the right ICA (arrow points the bifurcation). T1-weighted (D) and T2-weighted (E) axial MR scans demonstrate encephalomalacia along the right MCA territory. Please note that except for widened CSF spaces and shallower sulci, white matter anatomy is close to normal.



cervical segment (C1) of the ICA lies within the carotid sheath that divides into 2 layers: the inner layer continuous with the periosteum of the carotid canal and the outer layer continuous with the periosteum of the exocranium. The artery enters the carotid canal of the petrous bone anterior to the jugular foramen. The entrance of this canal is bordered anterolaterally by a lamina of bone called the vaginal process that is part of the tympanic bone. The petrous ICA (C2) runs within the periosteum of the carotid canal and is surrounded by areolar tissue, a venous plexus, and postganglionic sympathetic nerves. The petrous ICA first vertically ascends then makes a posterior bend and then courses anterolaterally. The greater and lesser petrosal nerves travel in distinct bony grooves immediately lateral to the horizontal portion of the petrous segment. The petrous segment ends where the carotid canal ends and next lacerum segment (C3) ascends in

the vertical canal of the foramen lacerum toward the posterior cavernous sinus. The C3 segment is also surrounded by periosteum. This periosteal layer and additional fibrocartilaginous tissue covers the exocranial foramen lacerum and makes a canal with a closed floor and an open roof through which the ICA exits. The lacerum segment ends at the superior margin of the petrolingual ligament and immediately distal to this structure the ICA enters the cavernous sinus.

A blunt injury to the ICA wall most commonly takes place immediately before the artery enters the skull base; within the petrous or lacerum portions and within the cavernous segment immediately and immediately distal to the posterior communicating artery (PCoA).^{2,3} Intradural injury of the C5, C6, and C7 segments is rare and may take place secondary to an uncommon fracture of the cribriform plate and/or anterior clinoid process. In the absence of a canalicular com-

promise, vessel wall injury is still possible by torsion and shearing forces.^{5,11} One example to this is injury distal to PComA where the PComA acts as a point of fixation of the circle of Willis. Yet, the likelihood of injury to ICA segments occurs in a pattern inversely proportional with the mobility of that specific ICA segment. Points at which the ICA is more vulnerable are (in the order of decreasing frequency) entry point to the carotid canal, the canal itself, foramen lacerum, and the dural penetration point between the distal and proximal dural rings. Wherever and however, the ICA wall damage will lead to thrombosis that may extend anterograde and/or retrograde.

The true incidence of ICA injury associated with closed head trauma is difficult to estimate firstly because angiography is not routinely carried out in head injury patients since 1975 and also because a significant number of patients may tolerate occlusion of one ICA without any symptoms due to collateral flow through contralateral anterior communicating and ipsilateral posterior communicating arteries. The relevant literature quotes an incidence ranging between 0.11% and 3%.^{4,12,13} The higher incidence is likely to result from small denominators representing small number of cohort patients since Arseni and associates from the pre-CT era evaluated an enormous number (19,664) of head injury patients and found 22 cases with thrombosis of the ICA (0.11%).¹² In the 1990s, Rommel and associates found an incidence of 3.3% (2/60)¹³ and Berlot and associates reported an incidence of 2.7% (4/145).⁴ Specific reviews of patients only with skull base fractures suggest that the likelihood of such an injury triples if the fracture crosses the trajectory of the petrous and/or cavernous ICA. Unger and associates found 5 ICA injuries in 83 patients (6%) with skull base fractures¹⁴ whereas Resnick and associates' study showed 6 ICA injuries in 55 cases (10.9%).¹¹ West and associates, who proposed a classification system for basal skull fractures, underlined that patients with simple transverse fractures (anterior or posterior) had better outcomes than those with diagonal fractures that pass along the long axis of the petrous bone or the occipitomastoid suture.¹⁵ Therefore a diagonal skull base fracture in proximity to the intracranial trajectory of the ICA is clearly a serious risk factor for vascular injury.

ICA injury should be suspected in any trauma patient in whom a focal neurologic deficit develops in the absence of a focal lesion on CT or when a neurologic deficit cannot be explained by routine imaging alone. The clinical picture of ICA

occlusion usually takes time to develop and this "lucid" interval may vary according to completeness of the initial obstruction and/or to collateral flow and even to perfusion pressures that may change over time. This lucid interval may be as short as 20 to 30 minutes as reported by Morgan and associates,² but usually symptoms will appear during the first 12 hours after the impact as reported by Arseni and associates.¹² Lucid interval was 12 to 24 hours for patients as reported by Shirai and associates⁹ and rarely it may take as long as 48 hours for the ischemia to appear.⁴ The longest reported interval between the impact and the ischemic symptoms was 38 days.¹⁶ Lucid interval is not only important for the arrangement of urgent angiography but also for the initiation of anticoagulant therapy because without heparin, an incomplete obstruction or even a minute dissection of the intima will cause retrograde thrombus formation all the way down to the ICA origin.

After a thorough review of patients with traumatic ICA injury, Morgan and associates underlined that an urgent angiogram is indicated for all patients with focal deficit in the absence of a CT appearance unrelated to the clinical picture; those with neurologic deficit after minor-appearing head injury and when there are signs of Horner's syndrome immediately after trauma.² Carter and associates, cost-conscious and cautious about inherent risks of conventional angiography, stressed that cost-efficient and low-risk screening regimens are necessary to evaluate patients at risk and further advocated the use of transcranial Doppler imaging (TCD), CT spiral angiography, MR, and MR angiography in place of conventional angiography.⁵ Considering the fact that ICA injury does not always have to follow a lucid interval and that it may also be associated with a severe head injury, screening with TCD represents a solid standard in the diagnosis or treatment of any patient with a head injury (mild, moderate, or severe) with a basal skull fracture. Tartara and associates also suggested use of MR angiography in place of conventional angiography, although their decision to proceed with angiography came after surgical decompression to alleviate increased ICP that may not be the ideal algorithm we want to suggest.⁶ We stress that the vascular study should be conventional angiography because with current technology, angiograms are not just static images and the surgeon can follow the blood flow from the very moment after dye injection. This was particularly useful in our patient because the initially

injected dye into the right ICA did not fill the intracranial ICA trajectory and when contralateral images were being obtained, the dye previously injected into the injured right ICA was still visible due to entrapment between intima and media clearly showing more than an MR angiography could show. As for CT findings, Pedraza and associates stressed that the most common CT finding in a patient with traumatic ICA occlusion was infarction in MCA territory but on the other hand same authors also stated that 33% of patients with posttraumatic ICA injury had normal CT scans.¹⁷ We believe that assessment of changes on serial CT scans is more helpful in elucidating the diagnosis rather than commenting on initial scans alone. Considering the fact that generally 24 to 48 hours must pass for evidence of ischemia to be seen on CT, no matter what, the crucial diagnosis is bound to be delayed. A high index of suspicion and use of angiography are the only reliable keys for a timely and proper diagnosis. Although we routinely obtain a second CT scan 4 hours after admission for all patients with minor-appearing head injury, it was not until the third CT scan (12 hours from admission, 17 hours from injury) that the radiographic signs suggesting ischemia appeared for the case reported here. Although our diagnosis cannot be considered immediate, the outcome was fortunately good with aggressive treatment. With experience and a high index of suspicion, one is bound to diagnose similar cases much earlier.

As for the role of MR in the diagnosis, Tartara and associates stressed the usefulness of MR and MR angiography to show occlusion of petrous, lacerum, and cavernous segments of the ICA although these authors used MR in the diagnosis postoperatively and almost 3 days after the injury.⁶ We were satisfied with the contribution of MR and MR angiography images although they were obtained long after the ischemic episode. During this interval a retrograde and possibly antero-gradate thrombus seemingly had formed and even became organized. Mainly because of redirected flow, MR and MR angiography was far from being precise as to at which point the vessel injury started. MR angiography of the cervical and intracranial ICA in the case reported here showed no flow at all, further proving that the organism stabilizes itself by completely excluding the injured vessel from the circulation and directing the flow from right common carotid artery directly into the right external carotid artery.

Our review of the pertinent literature revealed that there was no agreement or a stan-

dardized treatment for post-traumatic ICA injury. Nevertheless, considering the fact that reported patients were within a clinical spectra rather than representing a uniform clinical picture and that some arteries were totally occluded while in some patients there was still blood flow through the injured artery; it is only logical that some authors tried to modify certain clinical parameters while some tried to prevent more catastrophic clinical pictures. In general, management strategy was either observation alone or was similar to that of carotid artery stenosis with impending stroke. Arseni and associates conservatively observed 18 of their 22 patients.² In 2 patients, thrombectomy was added to conservative therapy but one of those died. Goodwin and Johnson and Carter and associates summarizing their experiences with single cases reported good results with conservative treatment and/or observation alone.⁸ However, these patients had either good collateral vessels or decreased flow through the injured ICA. Morgan and associates used acetylsalicylic acid for patients who were observed conservatively.² One patient deserves attention. This was a young male worker whose head was crushed between iron girders. This unfortunate patient had dissection of the right ICA with carotidocavernous fistula and complete transection of the left ICA. This patient was intubated and ventilated but even though CT showed malignant infarction of the left hemisphere, the researchers thought that "supportive therapy was all that could be offered."² Tartara and associates' approach to their patient with left ICA occlusion was contemporarily aggressive—similar to ours—but then their patient underwent decompressive surgery for intractable ICP only and without a preoperative diagnosis of major artery occlusion with malignant infarction.⁶ Unfortunately their patient died 24 hours later. As for the medical treatment of our patient, we started with an anti-edema protocol consisting of mannitol, dexamethasone (and furosemide when necessary), and continued with heparin.

Conclusion

ICA injury concomitant with head injury is difficult to diagnose but a focal deficit that cannot be explained by CT findings and/or not associated with impairment in the level of consciousness should suggest an ischemic origin. Timely diagnosis is crucial so despite its inherent risks, con-

ventional cerebral angiography should be considered in any patient with a head injury in whom neurologic deficit develops with a CT appearance that is unlikely to explain this presentation. With an angiographic solid diagnosis of ICA occlusion in hand and with the aid of available anesthesia and monitoring techniques, the brain can be kept relaxed and protected despite decreased perfusion pressures. In the end, the most important factor that determines the effectiveness of therapy for traumatic ICA occlusion will be patient's saved life.

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