Endovascular Treatment of Intracranial Posttraumatic Lesions

Assessing arteriovenous fistulas, dissections, and aneurysms.

BY ROSANA CERATTO, MD; JORGE CHUDYK, MD; CARLOS BLEISE; AND PEDRO LYLYK, MD

The incidence of cerebrovascular trauma has increased in recent decades, due to a greater amount of car accidents, military confrontations, and urban violence. The improvement of diagnostic and therapeutic procedures allows us to have a better understanding of the pathology and improve treatment.

Many vascular head and neck injuries are immediately lethal. In the surviving patients, the most frequently diagnosed entities are:

- Arteriovenous fistulas: when the injury compromises both the artery and the vein parallel to the lesion, and no immediate vascular repair is performed; deflected blood flow to the vein may occur through this communication, which is called an *arteriovenous fistula*.
- Dissections: the entry of blood from the arterial wall to the intramural hematoma formation; this can be located in relation to the intima (subintimal) or to the adventitia (subadventitial).
- Pseudoaneurysms: if the vascular injury has no communication with the outside, it produces a hematoma, usually formed by a fibrous capsule that, in time, turns into a pulsatile mass known as a *false aneurysm* due to the lack of arterial wall as in true aneurysms.

ARTERIOVENOUS FISTULAS

The most frequently observed cases are carotid cavernous fistulas (CCF), although they are a rare complication (0.2%–0.3%) of traumatic brain injury.[1]

A CCF is any abnormal communication established between the carotid artery and the cavernous sinus causing a pathological arteriovenous shunt, which can manifest in an anterograde way to the orbit, producing a severe ocular condition known as *pulsating exophthalmos*.

Within this group of fistulas, we can distinguish two different entities:

- Direct CCF: presents direct communication between the intracavernous internal carotid artery (ICA) and cavernous sinus (Figure 1). These are usually related to severe trauma with basal skull fractures, but have also been associated with direct surgical trauma and rupture of intracavernous carotid aneurysms.
- Indirect or dural fistulas: these have no posttraumatic etiology.

Direct CCFs

Direct CCFs are posttraumatic abnormal communications between the carotid artery and the cavernous sinus. Trauma is the etiology in 75% of the different carotid cavernous shunts, which have an incidence of

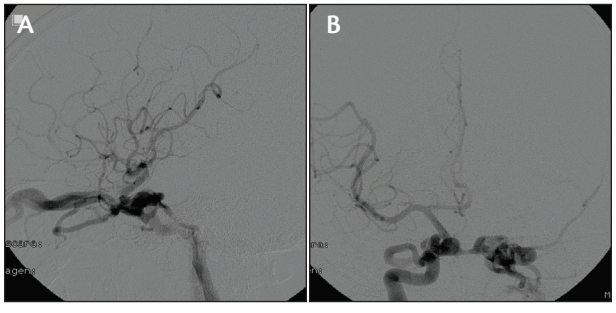

Figure 1. Right lateral carotid artery (A) and anteriorposterior (AP) angiograms (B) showed a direct CCF, single orifice with anterior venous drainage to the ophthalmic vein and contralateral to the inferior petrosal sinus through the coronary sinus.

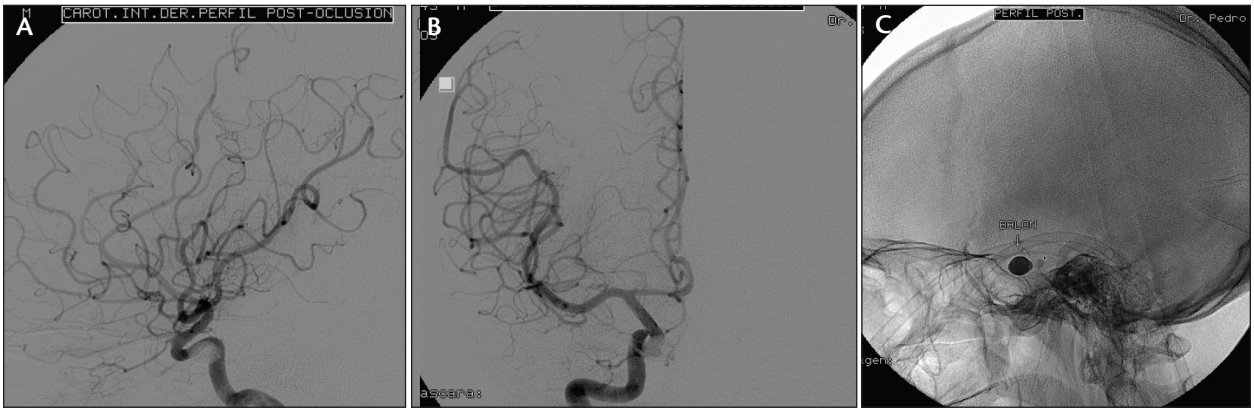

Figure 2. Right lateral carotid artery angiogram (A) and AP angiogram (B) after embolization. Lateral balloon mask (C).

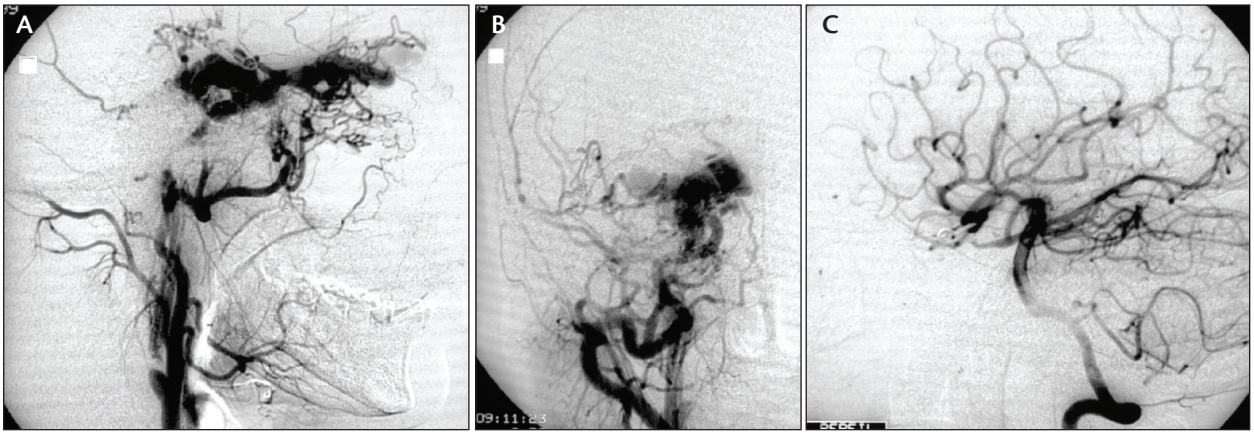

Figure 3. Right lateral (A) and AP carotid (B) angiograms. Surgical ligation of the supraclinoid ICA and supraclinoid clipping. Type A CCF before treatment; after surgery, type D. Left lateral vertebral artery angiogram (C). Posterior communicating artery irrigates sylvian segment.

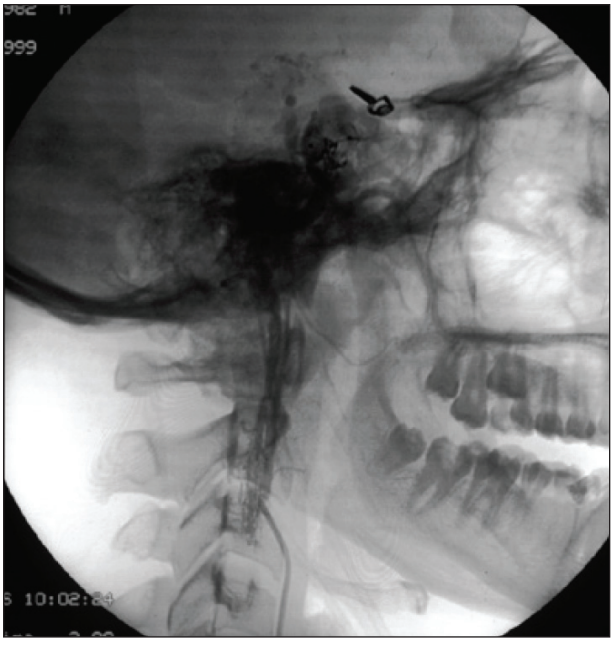

Figure 4. Lateral mask showing the position of the surgical clip of the ICA.

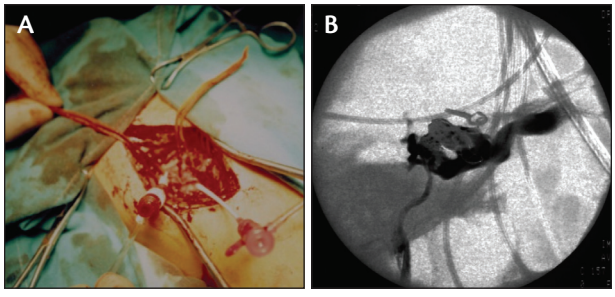

Figure 5. Direct surgical approach through the superior ophthalmic vein (A). Occlusion of a CCF with platinum coils (B).

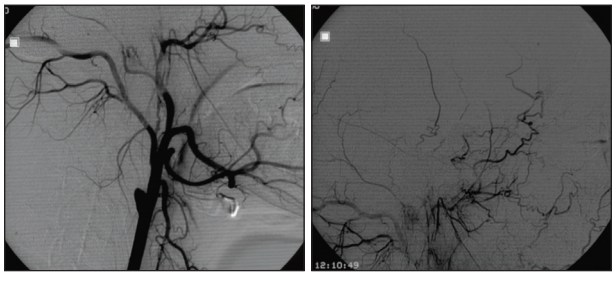

Figure 6. Carotid angiograms, lateral view after embolization.

1/10,000 to 1/20,000 in hospital admission. As many as 90% of the patients with direct CCFs may lose vision in the absence of treatment.[2]

The progressive loss of vision or the identification of cortical drainage veins in the angiogram determines urgent therapy. A detailed four-vessel angiogram including visualization of the circle of Willis pattern with and without carotid compression is mandatory to plan an accurate endovascular treatment. The location of the fistula in the cavernous carotid, presence of arterial dissection with the fistula, and maintenance of the flow in the ophthalmic vein and petrous sinuses must also be determined. Sometimes, a bilateral carotid cavernous shunt is found.

Embolization (Figure 2) allows access to the CCF through direct arterial access via the ICA.[3] In patients already treated with surgical clipping of the ICA (Figures 3, through 5), the superior ophthalmic vein can be approached directly or by retrograde venous catheterization through the ipsilateral or contralateral petrosal sinus (Figure 6).

POSTTRAUMATIC DISSECTION

Carotid dissections (Figure 7) have an annual incidence of 2.6/100,000 habitants.[4] Information involving vertebral territory is unknown. Although they usually affect young population groups between 30 and 50 years, carotid dissections have also been described in children and older adults.

It is the cause of 2.5% of all ischemic cerebrovascular events, whereas in patients younger than 60 years, it represents 22% of the cases.

The most common site for dissections is the extracranial portion of the cervical vessels.

Etiology

The origin of carotid dissections is primarily idiopathic, with traumatic injury as the second most common origin.

Many publications have shown the association between minor cervical trauma and significant physical effort with cerebrovascular dissections (25%–41% of cases). It has been described in relation to cough, vomiting, childbirth, weightlifting, pushing vehicles, chiropractic maneuvers of the neck, flexion and extension cervical rockers, prolonged use of the phone with neck flexion, and cervical rotation.

Treatment

The classic clinical triad of carotid dissection is ipsilateral headache, symptoms or signs of cerebral ischemia, and ipsilateral Horner syndrome. However, the presence of the three symptoms occurs in only 20% of the cases.[5] When the dissection affects intracranial vessels, it can also involve subarachnoid hemorrhage, because the vessels across the dura lose the external elastic membrane and have thinner adventitia.

Once the diagnosis is confirmed, carotid dissection is usually treated with intravenous heparin anticoagulation, and 100 mg of aspirin per day is sometimes prescribed.

Oral anticoagulation is prescribed, and frequent cervical Doppler scans are scheduled to evaluate the evolution of the dissection. In patients with multiple traumas, anticoagulation is usually contraindicated due to the increased risk of cerebral bleeding.

In cases where a pseudoaneurysm (Figure 8) is pres-

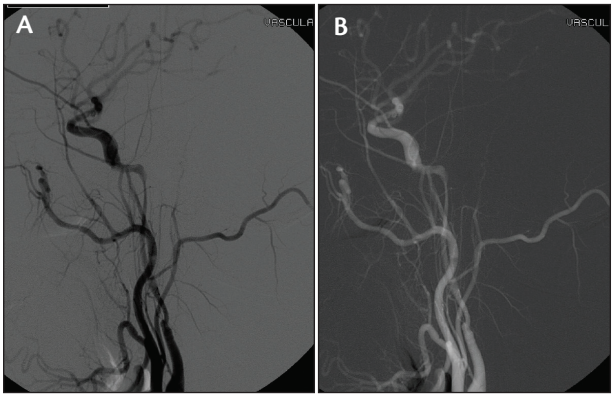

Figure 7. Extensive dissection of the left ICA (A) and lateral view digital subtraction angiogram (DSA) (B).

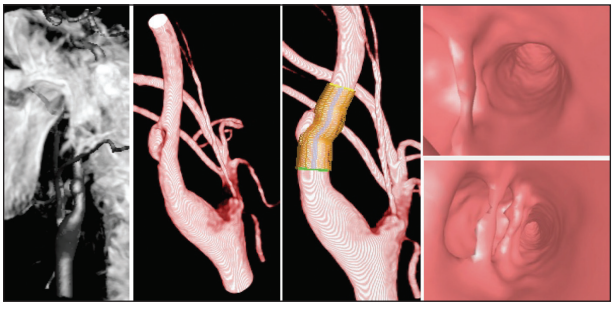

Figure 8. A traumatic pseudoaneurysm. Intimal dissection of the right ICA.

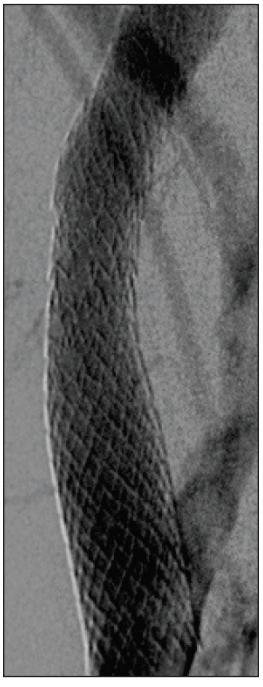

Figure 9. After angioplasty with stent placement. Immediately after treatment, vessel reconstruction was observed.

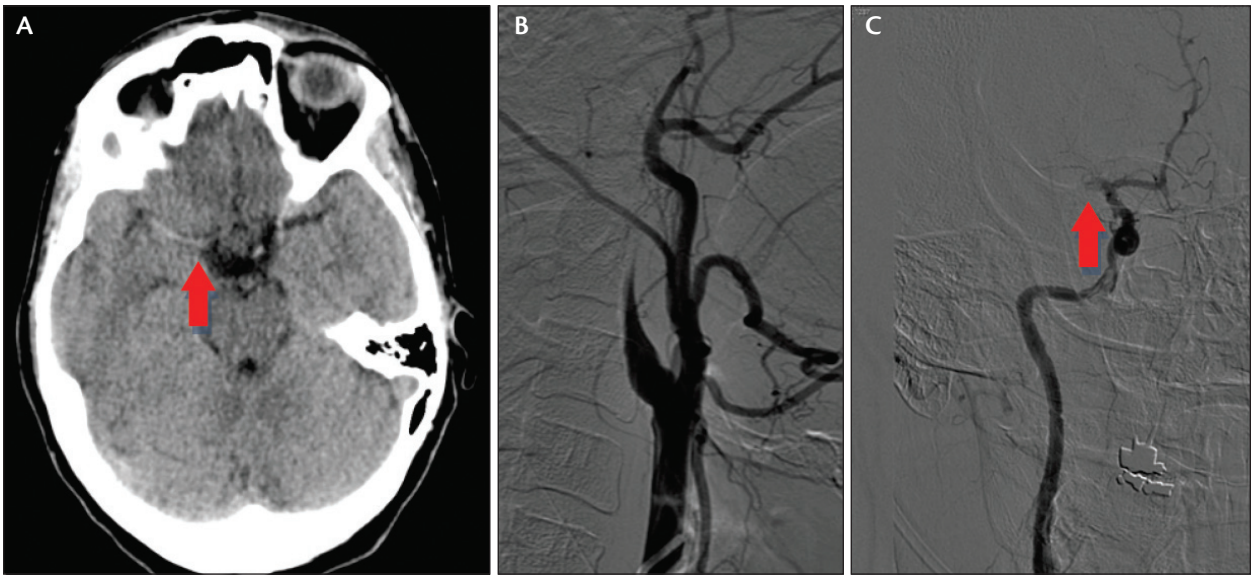

Figure 10. Patient admitted with left hemiplegia after a car accident. Corresponding CT scan shows string sign (A). DSA shows typical image of carotid dissection (B). There was leptomeningeal flow from the ipsilateral posterior cerebral artery. The right ICA was permeabilized, and the image of the thrombus in the middle cerebral artery is viewed (C).

ent in the diseased segment or cerebral ischemia or severe stenosis occurs, treatment of the lesion is indicated.

Surgical techniques with vein patching have fallen into disuse. The current treatment of choice is endovascular therapy with angioplasty and stent placement (Figures 9 - 11).[6] When a pseudoaneurysm is diagnosed, angioplasty is indicated in order to repair the vessel by flow diverter stent placement.[7] In some of these cases, additional treatment with platinum coils may be necessary.

TRAUMATIC ANEURYSMS

Traumatic intracranial aneurysms are considered a rare entity, representing < 1% of all aneurysms. They can be produced by a blunt or penetrating trauma and are most frequently described in the pediatric population; symptoms depend on the affected territory.

Early diagnosis of a traumatic aneurysm is a challenge, because it is sometimes not evident in the acute phase, and it may take 2 or 3 weeks until it becomes visible on the CT angiogram,[8] which is the method of choice for diagnosis.

Delayed hemorrhage (3 to 4 weeks after trauma) is typical and carries a high mortality rate (about 50%).

Until recently, these were treated by craniotomy and clipping, showing better results than conservative management, but with a mortality rate of approximately 30%. Recent publications showed successful endovas-

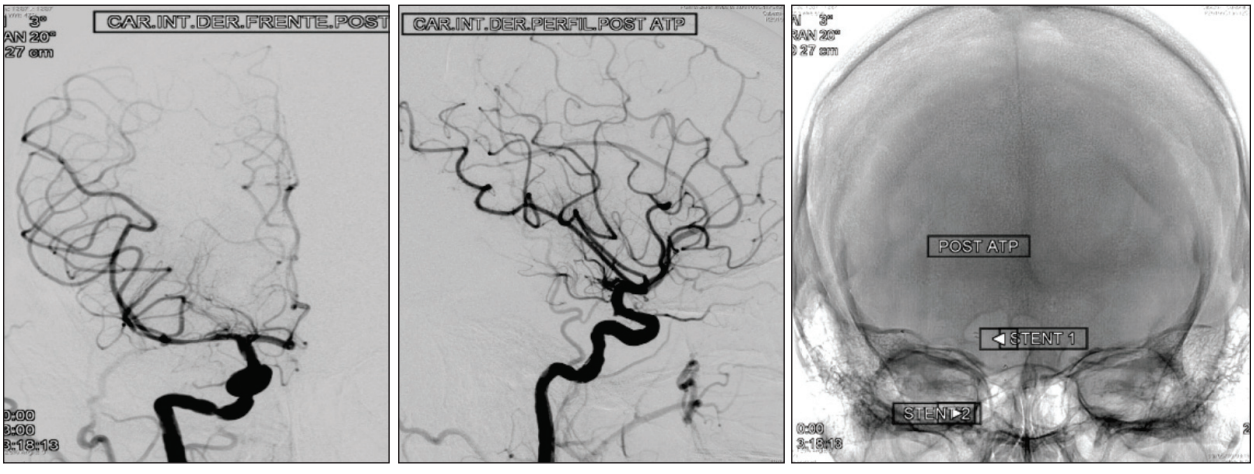

Figure 11. The right ICA after thrombectomy and proximal and sylvian stenting.

CASE REPORT: TRAUMATIC INTRACRANIAL ANEURYSM

A 30-year-old, right-handed man had a history of hypertension and surgical aortic valve replacement with a mechanical prosthesis (July 2010) due to a bicuspid aortic valve. He took 5 mg/day of perindopril and acenocoumarol for a target international normalized ratio of 2 to 3.

Forty-eight hours before hospitalization, the patient suffered head trauma without loss of consciousness. Two days later, there was acute onset headache (cranial throbbing, of moderate intensity) accompanied by numbness in his right hand and a fever of 100.4°. Physical exam was normal. CT scan of the brain showed subarachnoid hemorrhage located in the convexity of the left parietal lobe (Figure 12A). Angiography showed a small aneurysm (2 mm) in the distal portion of a parietal branch of the left middle cerebral artery (Figure 12B and C). Selective catheterization of related artery (Figure 13). Lateral mask view of the brain (Figure 14A) and CT scan after embolization with NBCA (Figure 14B).

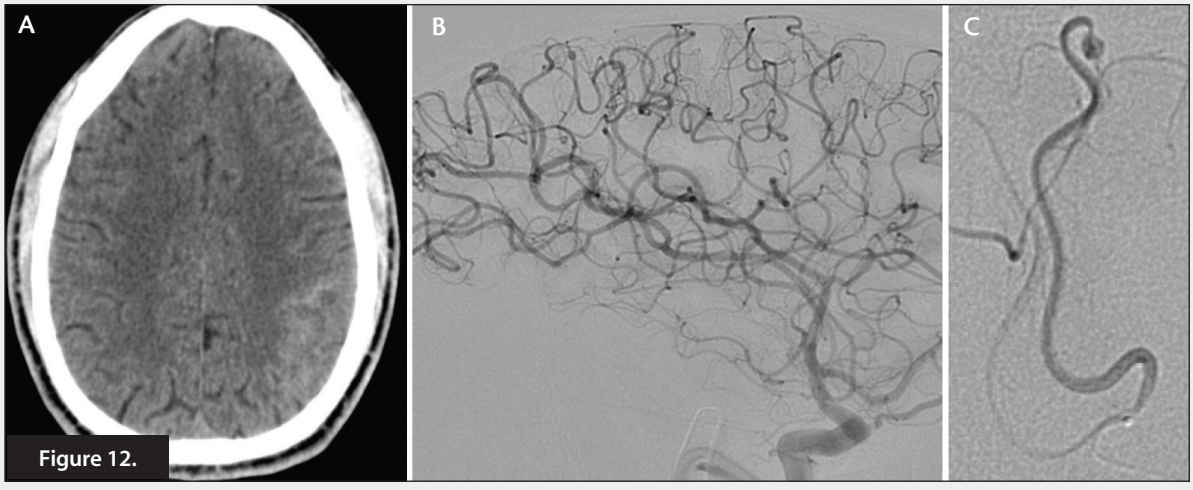

Figure 12.

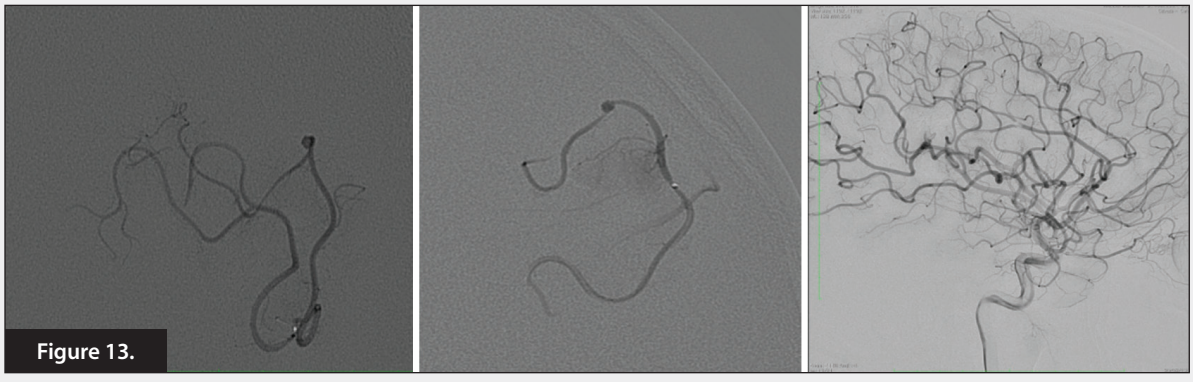

Figure 13.

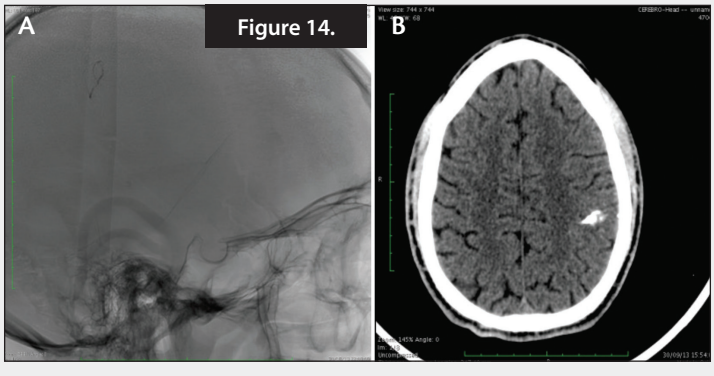

Figure 14.

cular treatment with low morbidity and no mortality.[9-11] For these patients, endovascular approaches have emerged as a valid therapeutic alternative.

This technique avoids craniotomy, brain retraction and dissection, and external manipulation of the vessels during surgery. Thus, other related complications to craniotomy, such as epilepsy or wound infection, are avoided.

The goal of endovascular treatment is to achieve complete occlusion of the aneurysm with preservation of the parent vessel. Platinum microcoils, acrylics (NBCA), polymers (Onyx, Covidien), or flow diverter stents may be used, depending on the location and size of the aneurysm vessel.

CONCLUSIONS

Multiple mechanisms of neural injury act in concert in the traumatized brain, including axonal shearing, hemorrhages, and increased intracranial pressure. Consequently, the traumatized brain is especially vulnerable to ischemia after emboli or hemodynamic compromise. The loss of vascular autoregulation in the injured brain reduces the ability to compensate for hypoperfusion.

In conclusion, endovascular treatment for CCF, pseudoaneurysms, and vascular traumatic dissections seems to be a rational and effective way to restore the artery lumen and prevent late hemorrhages or neurological deficits.[12] ■

Rosana Ceratto, MD, is an interventional neuroradiologist at ENERI in Buenos Aires, Argentina. She has disclosed that she has no financial interests related to this article.

Jorge Chudyk, MD, is a radiologist at ENERI in Buenos Aires, Argentina. He has disclosed that he has no financial interests related to this article.

Carlos Bleise is a radiologist at ENERI in Buenos Aires, Argentina. He has disclosed that he has no financial interests related to this article.

Pedro Lylyk, MD, is Chairman of ENERI in Buenos Aires, Argentina. He has disclosed that he has no financial interests related to this article.

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