Traumatic lesions of circle of Willis: Imaging finding and therapeutic aproach

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Learning objectives

1. To review the traumatic cerebrovascular injuries with special attention to the circle of Willis.
2. To understand the mechanisms of damage, special anatomical characteristics, typical types of injuries, as well as the clinical presentation, associated injuries and possible complications.
3. To describe the angiography features and the endovascular management of these lesions.

Background

Neurovascular injuries are uncommon complications after blunt or penetrating head trauma. They are of paramount importance because of their potentially devastating consequences that can lead to stroke or death. These lesions are often under diagnosed and detection is frequently delayed because of the late appearance of symptom manifestation and by failure to suspect a cerebrovascular injury in patients with multiples system injuries. Fortunately, the advances in non-invasive imaging, particularly CT angiography (CTA), and in interventional neuroradiology may be possible an early diagnostic and treatment of these injuries.

Traumatic lesions of vessels form the circle of Willis or proximal arteries can result from penetrating trauma, such as gunshot wounds, stab wounds, nail-gun injuries and skull fractures, and from closed head injuries, by shear injury or impaction of arteries against fixed dural structures. They also can be caused by iatrogenic arterial injury during surgical procedures, such as tumor removal, aneurismal clipping, transsphenoidal surgery and other neurosurgical procedures.

These lesions tend to be dynamic with a variable spectrum of diseases that involve partial or complete disruption of the vessel wall: from minimal separation of the intima or medial development dissection or pseudoaneurysm, to complete transection and subsequent exsanguinations or arteriovenous fistula formation when arteries and veins result in communication.

Imaging findings OR Procedure details
1. ANATOMY

The internal carotid artery (ICA) is intracranial after it crosses the base of skull through the carotid canal and leaves the petrolingual ligament, and includes the intracavernous, clinoid, paraoftalmic and communicant segments (Fig. 1).

The cavernous carotid roams inside the cavernous sinus and is surrounded by its venous spaces, fat and sympathetic plexus. The cavernous sinus (SC) is an extradural structure located in the lateral side of the sphenoid bone. It extends from the petrous apex to the superior orbital fissure. Like the other great dural sinus, the SC is formed by endothelial tubes surrounded by connective collagenous tissue without valves. The superior, lateral and medial surfaces of the CS are covered by dura matter, while the floor of the SC is formed by the periost of the middle cranial fossa. The upper surface is formed by the anterior extension of the tentorium and the lateral extension of the diaphragm sellae. The lateral and medial surface is formed by the dura matter of the middle cranial fossa. The cavernous ICA is near important structures, such as Gasserian glanglion, the CNs III, IV, 1st and 2nd division of V, and VI, and anterior clinoid process laterally. The CN VI is considered the only true intracavernous nerve because of its course along the lateral surface of the ICA inside the cavernous sinus. The sphenoid sinus is inferomedial to cavernous ICA and the sella turcica, hypophysis and optic chiasm are uppermedial (Fig.2). The CS is a central collector for the blood coming from sylvian veins, the inferior surface of the brain and the orbits. The communicating venous structures of the SC can be divided into afferent veins and efferent veins (1) (Fig. 3).

1. Afferent veins:

- Superior ophthalmic vein (SOV).
- Inferior ophthalmic vein (IOV).
- Angular vein (to SOV).
- Superficial middle cerebral vein (SMCV).
- Uncal vein (UV).
- Sphenoparietal sinus (SPPS).
- Veins of the foramen rotundum.
- Intercavernous sinus (ICS).

1. Efferent veins:

- Superior petrosal sinus (SPS).
- Inferior petrosal sinus (IPS).
- Basilar plexus (BP).
- Pterygoid plexus (PP).
- Inferior petroclival vein (IPCV).
- Petro-occipital sinus
- Internal carotid venous plexus.
- Foramen ovale plexus.
- Foramen lacerum plexus.

The most important afferent veins are the SOV, the SMCV and the SPPS. The principal venous drainage of the CS is in the IPS and the PP. Twenty-two per cent (22%) of the SMCV drains into the laterocavernous sinus, a venous channel in the lateral wall of the SC without communication with it in mostly of cases.

The cavernous segment ends at the root of the anterior clinoid process, when the ICA is surrounded with two dural rings (proximal and distal ring dural) that are the anterior continuation of the tentorium and of the dura in the wall of the cavernous sinus which insert in anterior clinoid process. After piercing the dura and the arachnoid membrane, the ICA courses within the subarachnoid space, upward and posteriorly underneath the optical nerve, and ascends to reach its bifurcation into the middle and anterior cerebral artery to form the circle of Willis.

The circle of Willis connects both side of the anterior cerebrovascular system with the vertebrobasilar system through the anterior communicating artery and posterior communicating arteries. A complete circle of Willis (without agenesis or hypoplasia in any of its elements) is composed of ten elements: the both internal carotid arteries, anterior communicating artery, A1 segments of both anterior cerebral arteries, both posterior communicating arteries, both P1 segments of the posterior cerebral arteries and basilar artery.

The circle of Willis is located below the hypothalamus and third ventricle and surrounds the stem hypophyisis and mamilares bodies. The A1 segment of the anterior cerebral arteries courses above the optic nerves. The posterior communicating arteries course above the optic tracts. The oculomotor nerves have an oblique course below the posterior communicating arteries (2) (Fig. 4).

1. TYPES OF INJURIES

Although blood vessels can be injured by a variety of mechanisms, all traumatic injuries involve either partial or complete disruption of the vessel wall. In intracranial arteries,
strength of the arterial wall is in the internal lamina elastic, because of the presence of thinner medial and adventitial layers and the lack of a well-developed external elastic lamina. Injuries range from separation of the intima from the media with development of dissection and false lumen, to medial and adventitial perforation with pseudoaneurysm formation or to complete transection.

In dissection lesions, blood along the subintimal space tends to produce luminal narrowing, while blood extending into the subadventitial space tends to weaken the vessel and can result in a pseudoaneurysm (Fig. 5). Both can progress to vessel occlusion (3). Traumatic aneurysms of intracranial vessels may be true or false. In true aneurysms, intima and media are disrupted, and only the adventitia is intact and allowed to expand. False aneurysms are the result of full-thickness interruption of the arterial wall with formation of a perivascular hematoma, which subsequently develops a fibrous wall.

Thrombus may form on the injured intimal lining or on the static flow within a pseudoaneurysm. Stroke occurs in a significant proportion of patients with traumatic neurovascular injuries. It is considered that in intracranial vascular lesions the stroke is caused by haemodynamic compromise as a result of arterial occlusion. In contrast, in patients with traumatic cervical carotid or vertebral arterial dissection the stroke occurs most commonly as the result of distal thromboembolism (4).

When the defect in the wall of the artery is near veins or venous sinus arteriovenous fistulas can occur. The majority of posttraumatic intracranial arteriovenous fistula are carotid cavernous fistulas (CCF), resulting in a direct shunt between cavernous ICA and the cavernous sinus.

Biffi et al. devised a blunt carotid artery injury grading scale, based on the arteriographic appearance of the lesion, which is useful in proving some prognostic information (5). There is clearly an association between degrade of the lesion and stroke risk.

1. Luminal irregularity (i.e. intimal injury) or dissection with less than 25% luminal narrowing.
2. Dissection or intraluminal hematoma with more than 25% luminal narrowing, intraluminal thrombus, raised intimal flap, or haemodynamically insignificant AVF.
3. Pseudoaneurysm.
4. Occlusion.
5. Transection or hemodynamically significant AVF.

- DISSECTIONS AND PSEUDOANEURYSMS
Traumatic aneurysms comprise less than 1% of all intracranial aneurysms. However, in children, they represent up to one third of intracranial aneurysms.

Traumatic aneurysms can occur in penetrating and non-penetrating head injury. Nonpenetrating head injury is a more common cause of traumatic aneurysms than penetrating aneurysm. They usually result from rapid deceleration, causing sudden brain movement and vessel wall injury from stationary structures, by a shearing force or by direct damage caused by a skull fracture. Traumatic aneurysms of the intracranial internal carotid artery are encountered mainly in the petrous and cavernous portion, and are almost always associated with basal skull fractures crossing of the carotid canal, the foramen lacerum or the cavernous sinus. The supraclinoid segment is seldom affected, and in the absence of a basal skull fracture, an overstretching or sudden torsion of the vessel during trauma may cause the lesion (6).

Traumatic aneurysms from penetrating injuries appear most frequently on peripheral branches of the middle cerebral artery.

The natural course of this type of lesions is difficult to define because it tends to be dynamic. However, several studies suggest prompt treatment reduces related stroke and mortality rates (7).

Traumatic aneurysms typically present with subarachnoid or less commonly intracranial hemorrhage. Epistaxis, unexplained neurological deterioration and cranial nerve palsy have also been reported as a result of traumatic aneurysms.

Traumatic intracranial aneurysms have a high rate of rupture, approximately 50% within the first week after injury (4). Some of these aneurysms expand gradually and rupture in acute or delayed fashion (particularly true in nonpenetrating injury), whereas others spontaneously shrink and disappear (Fig 6, Fig. 7). Larger aneurysms tend to rupture and smaller aneurysms may resolve without treatment in several cases. However, even if the lesion shrinks initially, it can re-expand later. Traumatic aneurysms rupture in a patient with severe head trauma usually has a devastating outcome (50% mortality).

Intracranial ICA dissection causes brain ischemia and cerebral infarcts. Cerebral hypoperfusion was probably the mechanism of many of these events, in contrast to distal embolism, which is thought to be the most important mechanism of cerebral ischemia in patients with extracranial ICA dissection (8).

Laceration or transaction of a major brain artery is frequently fatal because of exsanguinations, elevation of intracranial pressure or brain herniation. Intracavernous traumatic pseudoaneurysm may present with massive epistaxis.

In the setting of penetrating or blunt head trauma, the presence of a parenchymal hematoma, subarachnoid hemorrhage or some types of skull fractures (fractures extend through the carotid canal or the region of cavernous sinus) may indicate the presence of an intracranial traumatic aneurysm (Fig. 8, Fig. 9, Fig. 10).
Angiography is considered the reference standard for diagnosing traumatic cerebrovascular injuries. Typical angiographic features include a location along the course of secondary branches (while berry aneurysms are most commonly located at the proximal bifurcations of major arteries), no visible neck, irregular contour, and delayed filling and emptying of the sac (9). However, a history of recent trauma is the main criterion for the diagnosis of traumatic intracranial aneurysms (10). The typical angiographic findings of intracranial dissection are similar to those observed with dissection of extracranial vessels: string sign, double lumen, irregular scalloped stenosis, vessel occlusion or aneurysm formation. The dynamic nature of these lesions makes repeat angiography a particular important consideration.

Treatment options include surgical clipping, endovascular occlusion of the pseudoaneurysm with preservation of the parent artery, endovascular parent artery occlusion and vascular bypass (Fig. 11, Fig. 12, Fig. 13, Fig. 14)). Surgery is difficult because these lesions commonly occur where the ICA enters the skull base.

- **TRAUMATIC VASOSPASM**

Disturbances of the cerebral circulation have been observed in patient with severe head trauma, characterized in three distinct phases: hypoperfusion, hyperemia, and vasospasm (11). Posttraumatic spasm of the large cerebral artery is one of the potential causes of cerebral ischemia, as well as increased intracranial pressure, systemic intracranial hypotension, cerebral edema, focal tissue compression from hematoma, and microvascular pathology. Posttraumatic vasospasm is associated with severe subarachnoid hemorrhage, epidural hematomas, subdural hematomas, and intracerebral hemorrhage. More than 10% of patients with posttraumatic vasospasm have no subarachnoid hemorrhage (12). Therapeutics options include hyperdynamic therapy, balloon angioplasty and intra-arterial pharmacologic treatment (13) (Fig. 15, Fig. 16, Fig. 17).

- **CAROTID CAVERNOUS FISTULA**

The majority of posttraumatic intracranial arteriovenous fistula are carotid cavernous fistulae (CCF), resulting in a direct communication between the artery and the surrounding cavernous sinus. Traumatic CCFs are almost always Barrow type A. Barrow type A CCF are high flow arteriovenous shunt which develop directly and rapidly. CCF may be caused by laceration of the intracavernous ICA or one of its small intracavernous branches by penetrating injuries or spicules of bone, or after a blunt trauma by tearing of the cavernous internal carotid artery at points where is attached to the dura matter, located between the foramen lacerum and the anterior clinoid process (proximal and distal dural rings). In some cases, the rupture of a cavernous sinus aneurysm can cause a type A fistula.

Symptoms usually are immediately evident. In some cases, symptoms may appear in a delayed fashion. The signs and symptoms are related with the pattern of venous
drainage, from cerebral ischemia by steal phenomenon to cranial nerves injury. The increased pressure in the cavernous sinus avoids the normal venous drainage into the cavernous sinus, increasing the flow in those afferent and efferent veins. In this way, when the pattern of drainage is via the ophthalmic veins the clinical manifestations are ocular (orbital pain, ophthalmoplegia, exophthalmos, chemosis, orbital bruit, glaucoma and visual loss). When cortical venous drainage is present, subarachnoid hemorrhage or intracranial hematoma may occur, as well as increased intracranial pressure or neurological impairment. The posterior drainage via IPS or SPS may be responsible for oculomotor deficits due to ischemic oculomotor nerve damage, by vascular steal phenomena, or mechanical damage by dilated vessels (1).

The typical angiographic finding in CCF is a nearly instantaneous filling of the ipsilateral cavernous sinus as contrast reaches the cavernous internal carotid artery. Most of traumatic CCF drains into the SOV (89%) and the IPS (83%). Less common are the drainage into the SPS (48%) and SMCV (32%) (Fig. 18). Retrograde intracranial venous flow is present in 9% of patients. Drainage to the contralateral cavernous sinus is also common. The arteriogram should provide the following morphological information:

- Size and location of the fistula.
- Differentiation between indirect and direct fistula.
- Visualization of the SC and the venous drainage.
- Identities of risk factors such as intercavernous pseudoaneurysms or cortical drainage.
- Identification of trauma signs such as dissection or transsection.
- Visualization of collateral circulation and identification of possible lesions.

In contrast to indirect CCF, direct CCF are high flow fistulas in which the spontaneous occlusion is uncommon. Endovascular occlusion of the fistula with preservation of the parent artery is the current preferred treatment. There are some fatal circumstances in which endovascular embolization should be performed immediately. Indications for urgent treatment include: increased intracranial pressure or presence of cerebral cortical venous hypertension, acute impairment of visual acuity, increased intraocular pressure and worsening proptosis, intracerebral hemorrhage, presence of a large CS varix, epistaxis and sphenoid sinus aneurysm (4).

Treatments alternatives include manual compression, transvenous CS embolization, transarterial CS embolization, and parent vessel occlusion.

- Manual compression: It may be the choice management in cases with low-flow fistulas.
- Transarterial embolization: Several embolic materials are used for transarterial embolization of direct CCF, including detachable balloons, coils, coils or balloons in combination with liquid embolics.

The rate of successful occlusion of direct CCF with detachable ballons is 90% with preservation of the parent artery in 60-80% of cases. The failures or the recurrences of the lesion are mainly due to technical difficulties involved in navigating the balloon through the shunt, or to its rupture when in contact with the osseous spicules of the sellar region produced by fracture (14).

Coil embolization of traumatic CCF is used when the defect in the wall of the ICA is small with the subsequent impossibility to occlusion the fistula with ballon, and in cases of CCF due to a ruptured cavernous segment aneurysm (Fig. 19, Fig. 20). Usually transarterial coil embolizations require the use of assisted techniques performed using protection ballons or supporting stents (Fig. 21, Fig. 22).

Also has been reported the use of covered stent to transarterial embolization of direct CCF, with potential risk of arterial dissection and stent thrombosis, and the recently use of onyx (15), a new embolic material usually used to endovascular treatment of AVM, for both transvenous and transarterial embolization.

- Transvenous CS embolization: Due to its anatomical topography, short length, straight course and attachment to the dura along the petroclival fissure, the IPS approach represents the most preferred access route to the CS, followed by the transfacial approach through the SOV (1) (Fig. 23). The embolic agents usually used are detachable platinum coils. Transvenous embolization with coils alone seems to be more effective than transarterial embolization with coils alone, because tighter packing of the cavernous sinus is possible. Stenting may be used for occluding traumatic CCF in combination with coils to support coil placement and keep coils within the CS (16).

In order to prevent venous infarction, selective occlusion is the importance in cases where the normal venous drainage via the Sylvian vein or the SPPS needs to be maintained.

The most important complications of this route are perforation of the venous wall during navigation of the microcatheter, perforation of CS and coil migration into internal carotid artery. Arterial ischemic complications are less frequent with transvenous approach.

- Parent vessel occlusion: When it is not possible to repair the fistula by endovascular treatment, parent vessel sacrifice is required.
Fig. 1: Bouthillier’s classification. It divides the ICA in seven segments: C1, cervical segment; C2, petrous segment; C3, lacerum segment; C4, cavernous segment; C5, clinoid segment; C6, ophthalmic segment; C7, communicating segment.

Fig. 2: Anatomy illustration of the CS.
Fig. 3: The complex anatomy of the CS and its communicating venous structures.
Fig. 4: Illustration of the posterior communicating artery.
A. Subintimal dissection.

B. Subintimal hematoma with dissecting aneurysm.

C. Subadventitial dissection with pseudoaneurysm (false aneurysm).

D. Intimal tear with embolic formation.

**Fig. 5:** Mechanisms of damage to the arterial wall
**Fig. 6:** CASE 1. This 48 year-old woman was admitted to our hospital after being involved in a traffic accident. Her Glasgow Coma Scale (GCS) score was 6 on admission with left exoftalmos and quemosis. A, CT scan showed obvious subarachnoid hemorrhage, frontal intracerebral contusion and depressed right frontal bone fracture. B-D, CTA coronal planar reconstruction demonstrates a large pseudoaneurysm adjacent to the supraclinoid ICA. Axial CTA demonstrates the pseudoaneurysm adjacent to the lateral wall of the supraclinoid ICA and dilatation to the left SOV.
Fig. 7: CASE 1. A-D, Cerebral angiography shows the presence of an irregular narrowing of the supraclinoid portion of the right ICA secondary to dissection. There are no evidence of pseudoaneurysm showed in CTA, suggesting thrombosis (C-D, arrow). E, Lateral view angiography of the left vertebral artery demonstrates significant narrowing of the basilar artery due to vasospasm.
**Fig. 8:** CASE 2. This 32 year-old man was assaulted, presenting a stab wounds through the right orbit. CT scan shows ethmoidal bone fracture, intracerebral hematoma crossing the midline, subarachnoid hemorrhage and intraventricular hemorrhage.

**Fig. 9:** CASE 2. A-B, right ICA angiography and 3D angiography show a narrowing of the supraclinoid ICA and A1 segment of the ACA. Well visible is also a pseudoaneurysm in A1-A2 ACA. Note the presence of a fenestration in CMA. C, left ICA angiography shows displacement of the ACA due to intracranial hematoma and segmentary vasospasm.
Fig. 10: CASE 2. A-D, Contrast CT and sagittal plannar reconstruction, assessment five days after the admission, demonstrate rim enhancement surrounding the intracranial hematoma, suggesting a late cerebral abscess.
Fig. 11: CASE 3. Sixty year-old male admitted in our hospital after a traffic accident. He presented multiple facial fractures and right subdural hematoma Twenty days after the accident he presented III CN palsy. A-C, CT scan shows an hyperdense mass occupying the sphenoidal sinus with osseus remodeling. D-E, RM after contrast injection showing evident enhancement of the mass suggesting a cavernous pseudoaneurysm in sphenoidal sinus.
Fig. 12: CASE 3. A-B, Lateral and PA views of the left ICA angiogram demonstrate a giant pseudoaneurysm of the lateral wall of the cavernous ICA. C, Illustration of cavernous ICA pseudoaneurysm occupying sphenoidal sinus. D, Occlusion test (D). E-G, Occlusion of the left ICA, deploying coils in cavernous ICA (F) and final result demonstrating no filling of the pseudoaneurysm.
Fig. 13: CASE 4. This 57 year-old man was transferred to our hospital after trauma from a kick. His GCS score was 7 on admission. A-C, CT scan shows subarachnoid hemorrhage, and temporal and clivus fractures. D-E, Lateral and PA views of the right ICA arteriography reveal traumatic dissection and pseudoaneurysm on the supraclinoid ICA and posterior communicating artery. Note intraluminal thrombus in the supraclinoid ICA. F, Several ischemic lesions were seen in control CT as a result of multiple embolisms.
**Fig. 14:** CASE 4. Angiographic control three days later. A-B, Right ICA 3D angiography demonstrates pseudoaneurysms growth. C, Left ICA angiography shows severe spasm in the supraclinoid ICA. D-E, Endovascular treatment with stent deployed covering the pseudoaneurysm and posterior catheterization of pseudoaneurysmal sac. F, Final result reveals complete occlusion of the aneurysm.
Fig. 15: CASE 5. This 19 year-old boy was admitted to our hospital after a motorcycle accident. His GCS score was 7. He presented bilateral CN III palsy. CT scan showed subarachnoid and intraventricular hemorrhage and base skull fracture. A-B, PA and lateral views of right ICA reveal segmentary vasospasms in supraclinoid ICA, A1 and M1 segments, and cerebral posterior artery (B, arrow). C-D, PA and lateral views of left ICA show segmentary vasoepasm in supraclinoid ICA, A1 and M1 segments.
Fig. 16: CASE 5. A-C, Advance the microwire and inflate the balloon into the target vessel. B-D, cerebral angiography of the left and the right ICA after the treatment of M1 vasospasm with balloon angioplasty.
Fig. 17: CASE 5. Ten days after the injury, the patient presented a new subarachnoid hemorrhage. A-B, Second angiogram reveals a pseudoaneurysm in posterior communicating artery. C, Selective angiography shows the pseudoaneurysm. D-F, Occlusion of the aneurysm with coils.
Fig. 18: ICA arteriography shows direct CCF with its draining veins
**Fig. 19:** CASE 6. This 35 year-old female presented massive epistaxis during transsphenoidal surgery. A-B, PA and lateral right ICA angiography shows direct CCF and cavernous pseudoaneurysm.

**Fig. 20:** CASE 6. C-D, Control angiogram after embolization of the pseudoaneurysm with coils and subsequent occlusion of the CCF.
Fig. 21: CASE 1. A-C, PA and lateral views left ICA show direct CCF with opacification of intracranial arteries. Multiple venous drainages are observed (RFV, right facial vein; LFV, left facial vein). D, Selective left external carotid artery demonstrates no arterial supply to the CCF.
Fig. 22: CASE 1. First endovascular treatment of the CCF. A-C, Transarterial embolization of the CCF with coils, in combination with the use of balloon in ICA to support coils. D, Subtotal occlusion of the fistula at the end of the procedure.
**Fig. 23:** CASE 1. A, Cerebral angiography demonstrates recurrence of the CCF. B, Transvenous embolization via IPS (b, arrow) placing the coils into the CS. C, Decreased arteriovenous shunting after the procedure. Note the venous drainage into the SOV and IOV. D, Transfemoral placement of a microcatheter via the internal jugular vein, facial vein and SOV into the CS. E, Packing of coils. D, Final result shows subtotal occlusion of the fistula.
Conclusion

The radiologist can play a vital role in the diagnostic, follow up and treatment of these injuries.

An understanding of injury mechanism, clinical presentation, and recognition of associated injuries facilitates the early diagnosis.

The knowledge of the spectrum of vascular injuries, their evolution and possible associated complications enable the radiologist to determine the adequate endovascular approach.

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