EXTRA AND INTRACRANIAL ARTERIAL DISSECTIONS

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The dissection is caused by the entry of blood into the arterial wall with formation of an intramural hematoma, that progresses separating the parietal layers, and may be located under the intima (sub-intimal) or between the media and adventitia (sub-adventitial).

The first usually give symptoms of obstruction of the lumen, the second produce compression of adjacent structures or extravasation of blood.
2 EPIDEMIOLOGY

• The carotid artery dissections have an annual incidence of 2.5 -3 / 100,000. The data for the vertebral territory is 1-1.5 / 100.00 inhabitants / year. (1)

• This is a disease of young people (30 to 50 years), has also been described in children and older adults (2).

• Corresponds to a 2.5 - 5% of the etiology of ischemic stroke (3).

• In patients under 60 years produce about 22% (20-25%) of Ischemic stroke (4).

• The most frequent site of dissection is the portion of extracranial vessels.

PATHOPHYSIOLOGY

SUBINTIMAL HEMORRHAGE
STENOSIS-OCCLUSION

RUPTURE INSIDE THE LUMEN - FALSE LUMEN.

ANEURYSMATIC DILATION

PSEUDO-ANEURYSM

Blood Blister Like An
Pathophysiology

Both may produce distal embolism to intracranial arteries.

Cerebral infarction due to absence of CBF

Embolism
### Distribution of cerebrovascular dissections

<table>
<thead>
<tr>
<th>Extracranial 90%</th>
<th>Intracranial 10%</th>
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<tbody>
<tr>
<td>- ICA 75 % at 2 cm distal from the bifurcation</td>
<td>- ICA supraclinoid segment, MCA (M1), and ACA (A1)</td>
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<tr>
<td>- Vertebral artery 25% segments $V_3$, $V_1$</td>
<td>- Vertebral artery $V_4$ segment</td>
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<td>ICA: internal carotid artery; V: vertebral artery; MCA: middle cerebral artery</td>
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4 Extracranial arterial dissections
Cervical ICA

Sub-intimal cervical ICA dissection
Sub-adventitial cervical ICA dissection

Extracranial arterial dissections
Cervical Vertebral Artery
Intracranial Arteries Dissections
Intradural Vertebral Artery V4
4

Intracranial Arteries Dissections
Internal Carotid Artery

BLOOD BLISTER LIKE ANEURYSM
The most common presentation is spontaneous and secondly the traumatic.

In relation to spontaneous dissections, predisposing conditions have been described, as a basis:

**Systemic Hypertension**
**Dyslipidemia**
**Diabetes Mellitus**
**Smoking**
**History of cerebral and aortic aneurysms**
**Oral contraceptives**
**History of stroke and family history of arterial dissections**

**Diseases associated with connective tissue**

As several primary arteriopathy: fibromuscular dysplasia is present in 15 to 20% of cases, has also been linked to Marfan syndrome, Ehlers-Danlos type IV, cystic medial necrosis, a 1 antitrypsin deficiency, osteogenesis imperfecta, homocysteinemia and Pseudoxanthoma elasticum.

**NOTE:** A study with electron microscopy showed abnormalities of connective tissue (collagen and elastic fibers) in 68% of patients with spontaneous dissection.
With regard to traumatic causes, numerous publications have shown the association between minor cervical trauma and strong physical efforts with cerebrovascular dissections (25 to 41% of cases).

It has been described in relation to:

cough, vomiting
delivery
weightlifting
pushing vehicles
chiropractic neck exercises
flexo-extension of the neck in rockers
prolonged use of the phone with neck flexion
cervical rotation
Extracranial Dissections

Cervical Internal Carotid Artery

The classical clinical triad of ICA dissection is:

- Ipsilateral headache
- Cerebral ischemic signs and symptoms
- Ipsilateral Claude Bernard-Horner Syndrome
CLINICAL PICTURE

Extracranial Dissections

Vertebral Artery

Occipital headache
Frequently a dorsal-lateral infarction of Medulla oblongata (Wallenberg Syndrome)
6 CLINICAL PICTURE
Intracranial Dissections
Sub-adventitial

1-Fusiform appearance-Vertebral
2-Blood Blister Like Aneurysms-ICA

1-Vertebral artery– SAH- predominant of posterior fossa
2- ICA artery– SAH related with dorsal(superior) ICA- Parasellar SAH, or more extended, and frontal Intracerebral Hemorrhage.
Ultrasound

The use of complementary techniques of Doppler and carotid duplex with transcranial Doppler have a sensitivity of 95% (1).

Is greater in cases of total occlusion or severe stenosis.

Its main disadvantages are the lack of pathognomonic findings dissection and its limitation in diagnosing an intracranial dissection.

It is now primarily used for screening and initial monitoring of patients.

There are no studies on their specificity.

The findings may show are: occlusion, stenosis, emboli, echogenic "flap", thrombus, and "tappering" (longitudinally progressive occlusion) (1).

Nuclear Magnetic Resonance

Today is the technique of choice for to study cerebrovascular dissections due to its high sensitivity and specificity, able to combine brain and vascular imaging, and it’s a noninvasive study.

Other advantages are: it allows to visualize the intramural hematoma, the expansion of the arterial wall and the relationship with the neighboring parenchyma.

Among its disadvantages provides little information on the degree of stenosis and formation of pseudoaneurysms.

The usual findings in MRI are:
- the sign of the Crescent Moon (hyperintense crescent moon in T2 sequences corresponding to intramural hematoma, around the vacuum signal flow of the blood flow)
- hyperintense vessel, poor or no visualization of the vessel and compression of the lumen.

The Angio MRI can show: decreased lumen, blood in the false lumen or the presence of a "flap".
Angiography
Despite advances in non-invasive techniques, angiography by digital subtraction remains the gold standard for diagnosis of this condition. Has a high sensitivity and specificity to diagnose multiple dissections, underlying arteriopathy (eg FMD), obstruction of distal branches by a stroke and intracranial view.

Describes the following signs: stenosis (string sign), "tappering", aneurysmatic dilation, intimal "flap", crookedness, subcranial distal stenosis, and the presence of a double lumen.
There is no prospective, randomized and controlled trial on the treatment of cerebrovascular dissections. All recommendations derived from case reports, expert opinion and small series. This is evidence of class C.

Anticoagulation
Most authors recommend treatment in patients with signs of ischemia, or brain imaging evidence of intracranial stroke. Morbidity and mortality associated with vascular dissection are due to brain infarction by cerebral embolism from the site of dissection and thrombosis in situ. There is no consensus regarding the use of anticoagulants in asymptomatic patients or only presenting with local symptoms. Given that 73% of these patients presented ischemic brain events (TIA or infarction) within the first month, it’s reasonably indicate to treat them.
Anticoagulation should be initiated as soon as possible because 82% of ischemic strokes occur in the first 7 days, but have been described to one month after the onset of symptoms, and also through the third month. Despite anticoagulation, there are new signs of brain ischemia by up to 14% of cases.

Anticoagulation is initiated with intravenous heparin taking the thromboplastin time partially activated (TTPK) to 1.5 to 2 times the baseline of the patient, and then switched to oral anticoagulant, leading an "international normalized ratio (INR) between 2 and 3.

Contraindications to anticoagulation are the general, and the presence of a large stroke clinical or radiological. In the case of cerebrovascular dissections anticoagulation is contraindicated when it is intracranial with signs of bleeding, due to high risk of vascular rupture and the presence of SAH.

We recommend a minimum of three months treatment for ischemic cases.
Antiplatelet Agents

The usefulness of antiplatelet agents, as well as anticoagulation, has not been demonstrated in this condition, with a good level of evidence. However, its use is recommended in cases where there is contraindication for anticoagulation.

Ongoing study CADISS - (UK)
Double-blind, randomized, Anticoagulants vs. Antiplatelet Agent

Thrombolysis

There is no evidence that the prognosis for both morbidity and mortality improvement, is different in the subgroup of stroke secondary to carotid dissection with the use of i.v. tissue plasminogen activator (tPA).
Surgery and Endovascular Treatment

The surgery has been limited to the following cases:

Recurrence of ischemic symptoms despite anticoagulation, dissecting aneurysm expansion, and SAH.

The techniques used are:

- proximal vessel ligation, or "Trapping"
- Associated or not to high flow "bypass"
- Wrapping with muscle tissue or silk material
- Resection of the aneurysm with arterial reconstruction or by-pass.
- Clipping - time delayed

Recently, more works are published based on the primary concept that is to treat the damaged wall of the artery with stents and / or coils, using techniques of interventional radiology in this condition.
DISSECTING ANEURYSMS
OF THE ANTERIOR CIRCULATION

* “BLOOD BLISTER LIKE” ANEURYSMS
* “CHIMAME” LIKE ANEURYSMS

FREQUENCY
0.3-1% OF THE INTRACRANIAL ANEURYSMS
(OHARA Y SUZUKI)
OUR EXPERIENCE
ETIOPATHOGENY OF THE BLOOD BLISTER LIKE ANEURYSMS

1-ACQUIRED INJURY OF THE ARTERIAL WALL
- Systemic Hypertension
- Arterial sclerosis
- Lipohialinosis
- Hypercholesterolemia
- Atherosclerosis
- atheromatous ulcer

2 - HEMODYNAMIC STRESS OF CBF OVER AREA WITH STRUCTURAL DYSFUNCTION OF COLLAGEN
DORSAL ICA- BLOOD BLISTER LIKE ANEURYSM
“BLOOD BLISTER LIKE ANEURYSM” OF ANTERIOR CEREBRAL ARTERY- A1

SURGICAL TECHNIQUE

- WRAPPING + CLIPPING
- CLIPPING
- TRAPPING + BY-PASS
- ANGIOPLASTY (ANEURYSMORRAPHY)
WRAPPING OF THE BLOOD BLISTER LIKE ANEURYSM
DIRECT CLIPPING OF THE BLOOD BLISTER LIKE ANEURYSM
-FOLLOWING THE DIRECTION OF THE ARTERY
-LATE SURGERY
-”LIGHT” CLIP
BLOOD BLISTER LIKE ANEURYSM
RIGHT ANTERIOR CEREBRAL ARTERY (A1)

BLOOD BLISTER LIKE ANEURYSM
LEFT ANTERIOR CEREBRAL ARTERY (A1)

39 YEARS OLD MALE, WITH HYPERTENSION, SUDDENLY COMPLAINTS OF SEVERE RIGHT OCCIPITAL HEADACHE, AND LATER ALL THE HEAD. SAH SIGNS, WITHOUT LOCAL SIGNS.
Reconstructive endovascular treatment of ruptured blood blister–like aneurysms of the internal carotid artery.

ICA-BBL An

Control Angio two months
An regrowth

SWS technique

Control Angio three months

Byung-Hee Lee, Byung Moon Kim, Moon Sun Park, Sung Il Park, Eun Chul Chung, Sang Hyun Suh, Chun Sik Choi, Yu Sam Won, In Kyu Yu: Reconstructive endovascular treatment of ruptured blood blister–like aneurysms of the internal carotid artery.

BLOOD BLISTER LIKE ANEURYSMS

CONCLUSIONS

- Uncommon Aneurysms (1%)

- Associated with arteriosclerosis and/or atherosclerosis, or collagen dysfunction

- Located in arterial course

- Fragile walls of neck and dome

- Treatment of Surgery, Endovascular and Medical are rapidly developing.
THANK YOU VERY MUCH !!!