Intracranial Arterial Dissection: Analysis and Results in a Series of 69 Cases

**Diseccion Arterial Intracraneana: Estudios y Resultados en Serie de 69 Casos**

Alejandra Jaume¹
Mariana Romero¹
Matías Negrotto²
Roberto Crosa¹
Leonardo Moreno³

**ABSTRACT**

**Introduction:** Intracranial dissections (ID) were once considered to be rare. Nevertheless, they cause 5 to 20% of ischemic strokes in children and young adults. Even nowadays in Uruguay, angiography is not frequently performed in patients with evidence of ischemic vascular disorders. Consequently, the available data may underestimate its real prevalence. **Materials and methods:** Cohort study including all patients who were diagnosed with arterial dissection, using digital angiography, in a period of 10 years, from August 2000 to August 2010. A total of 69 patients, ages 2 to 69, with and without history of trauma were included. The clinical presentation was subarachnoid hemorrhage (SAH) and / or brain ischemia. The average follow-up was 2 years. **Results:** From 69 cases, 20 were children under 15 year-old, and 49 were over 15 year-old. Patients presented with ischaemic stroke in 35 cases, and hemorrhagic stroke in 34. Thirty seven patients (54%) presented arterial dissections of the posterior circulation. It should be noted that the clinical presentation for posterior fossa dissections was mostly subarachnoid hemorrhage (SAH) (67%). On the contrary, the anterior circulation showed a large majority of ischemic strokes (72%) compared with SAH (28%). Subsequently, 31 dissecting aneurysm were diagnosed and treated, using different endovascular techniques. Of the 31 cases which were treated 4 died (14%) after treatment related to the underlying pathology, 18 (56%) had good evolution of symptoms, and 10 (30%) developed some kind of neurologic impairment. **Conclusions:** Endovascular treatment is the treatment of choice for dissecting aneurysms, being an effective and safe treatment. In cases of dissection without SAH or dissecting aneurysms, treatment of choice is medical treatment with anticoagulation.

**Key words:** Internal carotid artery dissection; Fibromuscular dysplasia; Subarachnoid hemorrhage; Stroke.

**INTRODUCTION**

Intracranial dissections (ID) were once considered to be rare. Nevertheless, they cause 5 to 20% of ischemic strokes in children and young adults¹. Even nowadays in Uruguay, angiography is not frequently performed in patients with ischemic vascular disorders. Thus, available data may well underestimate its real prevalence.

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¹Department of Neurosurgery, Hospital de Clínicas de Montevideo, University of the Republic, Uruguay
²Endovascular Neurosurgery Center, CEDIVA, Montevideo, Uruguay.
³Department of Quantitative Methods, CCEE, University of the Republic, Uruguay
Dissection is defined as an injury to the arterial wall that determines a tear in the wall, separating its layers, and disrupting together forming a true and a false lumen. The topography of the vascular dissection is related, in most cases, with the passage of the vessel through the dura mater. Thus, usually the most frequent dissections are in the posterior circulation, between V3 and V4 (between extradural and intradural segment of the vertebral artery), and in the anterior-level C4-C5 segment (between the cavernous and clinoid segments internal carotid artery). That is, between a mobile and fixed segment of an artery.

**Materials and Methods**

Cohort study including all patients who were diagnosed with arterial dissection, using a digital-substraction angiography, in a period of 10 years from August 2000 to August 2010. A total of 69 patients of both sexes, ages 2 to 69, with and without history of trauma were included. The clinical presentation was subarachnoid hemorrhage (SAH) and/or brain ischemia. Most of the patients were studied with an average follow-up of 2 years.

In some of the patients studied at our center, arterial dissection had been previously diagnosed by Magnetic Resonance Imaging (MRI) and was subsequently confirmed by digital angiography. Treatment was decided taking into account the clinical presentation and angiographic findings.

Dissecting aneurysms are heterogeneous, depending on the size of the neck, as well as the flow on the aneurysm. Most patients were followed an average of two years.

The graphics were done with ggplot2 package which is available in the language R (the R project for statistical computing), with the particular use of rpart randomForest packages.

**Results**

Thirty per cent of the patients were younger than 15 years old, while 70% were between 15 and 69 year-old. Mean age of presentation for intracranial arterial dissection in children was 9.5 years in our series, while in adults was 40.3 years.

Stroke presented as ischemic in 35 cases (51%) while hemorrhagic presentation was observed in 34 cases (49%). Out of 69 patients, 37 (54%) presented arterial dissection of the posterior circulation, most of them in the V4 segment of the vertebral artery (22). According to frequency, this localization was followed by segments P1-P2 and P2-3 of the posterior cerebral artery (5), basilar artery (5) and Posterior Inferior Cerebellar Artery (PICA) (5); 32 patients (46%) presented dissections of the anterior circulation, most of them in the carotid artery above its intracavernous portion (C4) (24), followed in a descending order of frequency, by the anterior cerebral artery (5) and the Middle Cerebral Artery (3).

It should be noted that the clinical presentation for posterior fossa dissections was mostly SAH (67%, i.e., 25 out of 37 patients), far higher than ischemic strokes (12 cases, 33%). On the contrary, the anterior circulation showed a large majority of ischemic strokes (23 cases, 72%) compared to SAH (9 cases, 28%).

Of the studied sample, 31 dissecting aneurysms were found. Out of 34 patients presenting with SAH, dissecting aneurysms were found in 27 of them (86%). From P1 to P3, dissecting aneurysms associated with SAH were consistently found. As for the causes of the dissection, a history of trauma appeared frequently in posterior circulation dissections (74%). On the other hand, for anterior circulation dissections the number of patients with a history of trauma was approximately equal to that of patients with no clear cause.

In our sample, most spontaneous dissecting aneurysms were associated with dysplasia.

The topography of vascular dissection is related in most cases, to the passage of the vessel through to the dura where increased the vascular friction is encountered, which for dissections of P1-P2 and P2-P3 segments were located at the passage through the incisurae tentoria.

Endovascular treatment was the treatment of choice for all patients having dissecting aneurysms (24 patients with acute SAH associated with acute dissecting aneurysms, 4 patients with dissecting aneurysms presenting as ischemic strokes [deferred], and 1 patient with a subocclusive subintimal dissection aneurysm).
Neck immobilization was used in patients having vertebral artery dissection. In patients with ischemic stroke and without a dissecting aneurysm, systemic anticoagulation was started.

Endovascular treatment varied according to the localization and features of the aneurysm. Occlusion of the feeding vessel with coils was the chosen treatment for most of the dissecting aneurysms cases of segment V4 of the vertebral artery, and for those of the segment P1 to P2. Stent-assisted coiling was the chosen treatment for cases of basilar artery aneurysms.

In the case of carotid dissecting aneurysms, the chosen treatment was vessel occlusion, either by stent plus coils or by coils only. In one single case, stenting was performed in a patient having subocclusion of the internal carotid artery in its supraclinoid segment.

Of the 32 cases that underwent endovascular treatment, 45% were treated with coils, 35% with stent-assisted coiling, and 20% had occlusion of the feeding vessel, and did not have functional implications for the patient. Of the total sample of endovascularly treated patients: 4 died (14%), 18 (56%) remained symptom-free with a GOS of 5, and 10 (30%) showed some degree of neurologic impairment. All 4 deceased patients were comatose at diagnosis with a SAH Hunt and Hess IV or V at the moment of diagnosis.

Of the 38 patients who underwent systemic anticoagulation for at least 3 months, 20 (62%) did not develop further neurological deficits and the initial symptoms reversed completely, while the remaining 38% maintained focal neurological deficits to varying degrees.

**Case Presentation**

**Case 1**

MB, 22-year old male, football player, who presented with intense headache and a cerebellar syndrome (Figures 1, 2, 3).
Case 2

42-year-old male, with alcohol-dependence syndrome and no history of trauma, presented with left hemianopia and a SAH (Figures 4, 5).

The patient had a good outcome with complete resolution of symptoms during the 2-year follow-up period.
Spontaneous arterial dissections are a frequent cause of strokes in young people and middle-aged adults. The idea has been increasingly accepted in the last years, establishing IC dissections as a pathology that is more often diagnosed and recognized as a cause of stroke. In patients younger than 50, arterial dissection accounts for 14 to 20% of cases. The diagnostic of arterial dissections has increased lately in line with the advancement of imaging diagnostic techniques.

One of the mechanisms that explains the pathogenesis of an acute IC dissection is the irruption of blood through a tear on the vessel wall, disrupting and its layers, generally involving the internal elastic lamina. Computerized tomography was performed, showing ischemia in the territory of the left posterior inferior cerebellar artery (PICA) (Figures 6, 7).

**Case 3**

61-year old male presented a head trauma with no loss of consciousness. Two days after the event he begins with headache and visual disturbances, followed by dysarthria and left hemiparesis, predominating in the upper limb. A computerized tomography was performed, showing ischemia in the territory of the left posterior inferior cerebellar artery (PICA) (Figures 6, 7).

**DISCUSSION**

Spontaneous arterial dissections are a frequent cause of strokes in young people and middle-aged adults. The idea has been increasingly accepted in the last years, establishing IC dissections as a pathology that is more often diagnosed and recognized as a cause of stroke. In patients younger than 50, arterial dissection accounts for 14 to 20% of cases. The diagnostic of arterial dissections has increased lately in line with the advancement of imaging diagnostic techniques.

One of the mechanisms that explains the pathogenesis of an acute IC dissection is the irruption of blood through a tear on the vessel wall, disrupting and its layers, generally involving the internal elastic lamina. The internal elastic lamina is
the strongest layer of the intracranial cerebral arteries. Under normal conditions it can withstand up to 600 mmHg, but age and hemodynamic stress weaken this layer. Likewise, a mechanical stress like head rotation may weaken the internal elastic lamella. The disruption of the internal elastic lamella does not reattach and is repaired and reinforced by the intima.

IC arterial dissections can be either subintimal or subadventitial. The presentation varies from a clinical picture of ischemic stroke to that of a subarachnoid hemorrhage (SAH) depending on the underlying lesion. When the dissection is subadventitial, it generally produces a dissecting aneurysm that ruptures and causes a hemorrhage, and when the dissection is subintimal it usually presents as an ischemic stroke because of the vessel occlusion or the production of emboli from the intimal flap. IC arterial dissections, therefore, can present as either ischemic or hemorrhagic strokes.

For a long time this type of lesion has been associated to some extent to a significant trauma that “justifies” the rupture of an IC vessel. On this account the absence of a history of trauma can lead to dismissing this clinical entity beforehand. However, we have noticed in our series that the history of trauma was more typically associated with dissections in the posterior circulation (74%), whereas in the anterior circulation the number of IC dissections with history of trauma nearly equaled the number of dissection without trauma. Therefore the absence of previous trauma does not rule out a diagnosis of IC dissection, as has been observed during daily clinical practice.

IC dissections are associated with several clinical entities that might be considered as “predisposing factors”, such as Marfan syndrome, fibromuscular dysplasia, atherosclerosis and Moyamoya syndrome; a congenital predisposition to develop this entity has also been reported.

Although aneurysms as a pathologic entity is apparently rare in children, most aneurysms found in this age group are dissecting aneurysms. In our study, the most frequent presentation for posterior circulation aneurysms was a SAH.

In our long experience of studying and treating patients of all ages with cerebrovascular diseases, in these last years we have witnessed a change in the reasoning behind the causes of strokes.

Our experience indicates that IC arterial dissections are also found in the young adult group and, more particularly, in children with ischemic stroke; mean age 9.5 years for children under 15 year-old, 40.3 for adults. The pediatric population tended to present with ischemic strokes whereas the hemorrhagic presentation was seen more often in young adults. This pathologic entity has been underestimated, particularly in the pediatric age group; since the first reports by Yamada and Colsin in 1967, and those of Fisher in 1978, a considerable debate has developed around this emergent entity that currently represents one of the main causes of strokes in children.

According to some estimates, nearly 50% of those affected by intracranial arterial dissections are under 16, therefore early diagnosis is very important in order to provide an adequate and timely treatment in line with the specific circumstances of each case.

We know that intracranial dissections generally present differently than extracranial dissections and, according to several authors, the former carries worse prognosis.

Regarding adults, a dissection can be misdiagnosed, and subsequently treated with fibrinolytics, which is a highly dangerous procedure.

Many endovascular techniques have been described for the treatment of arterial dissections, including occlusion of the feeding vessel, stenting, stent-assisted coiling, or more recently by placing flow-diverters.

Conclusions

Intracranial dissection is an underdiagnosed pathology in our environment, with a high morbidity and mortality if not treated early. Endovascular treatment is the treatment of choice for dissecting aneurysms, being an effective and safe treatment. In cases of dissection without SAH or dissecting aneurysms, treatment of choice is anticoagulation, with excellent results and very low risk.

References


CORRESPONDING AUTHOR

Alejandra Jaume, MD
Residente Neurocirugía (Quinto año), Hospital de Clínicas Universidad de la República, Montevideo, Uruguay
Camino Carrasco 6093 - CP 11500. Montevideo, Uruguay
(00598) 26010891
(00598) 99273939
E-mail: ale.jaume@hotmail.com