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Imaging of the Intracranial Artery Dissection

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Abstract

Intracranial dissection is one of the most important cause of vascular stroke either ischemic or hemorrhagic in young and middle-aged patients. The diagnosis of intracranial dissection is clinically significant as it can cause an ischemic stroke or might be responsible of a disabling sub-arachnoid hemorrhage. Computed Tomography Angiography (CTA) of the Willis circle as well as Magnetic Resonance Imaging (MRI) and Digital Subtraction Angiography (DSA) are the best examinations to confirm the diagnosis. We report a retrospective study including 10 patients with an intracranial dissection seen during a period of 02 years from January 2018 to December 2019. The diagnosis of intracranial dissection was confirmed by CT angiography and DSA. The posterior circulation is the most frequent in our series representing 70% of cases and 30% for the anterior circulation. The radiological signs are distributed as follows: occlusion in 03 cases (30%), stenosis in 07 cases (70%), tubular dilatation in 02 cases (20%) and dissecting aneurysm in 03cases (30%).

Keywords: Intra cranial artery dissection; Subarachnoid hemorrhagic; Stroke; Aneurysm; Transient ischemic attack

Introduction

Intracranial artery dissection is one of the most important cause of vascular stroke either ischemic or hemorrhagic in young and middle aged patients representing 14 to 20% of the ischemic strokes and 6% of hemorrhagic [1,2]. Clinical presentation is not specific because some symptoms may mislead the diagnosis such as headaches, hemicorporeal deficit or intracranial hypertension. Radiological investigations are important for the diagnosis and are based mainly on the CTA and DSA. The objective of this study is to evaluate the contribution of computed tomography angiography and DSA in the characterization and diagnosis of exclusive intra-dural dissection and to describe the imaging features.

Material and Methods

We report a retrospective study including 10 patients with an intracranial dissection divided between: 40% men and 60% females, with a mean age of 47 years, seen during a period of 02 years from January 2018 to December 2019. The patients included are only those with intracranial dissection, the cervical dissection extended to the intracranial vessels are excluded in our study. All our patients underwent neurological examinations as well as brain CT angiography and/or DSA.

Results

Intracranial dissections were frequent in young subjects with an average age of 47 years with a predominance of women (60%). Clinical signs that patients experienced by frequency were: headache, intracranial hypertension syndrome, loss of consciousness and hemicorporeal deficit. All patients have performed non enhanced brain CT followed by an arterial injection acquisition and arteriography. The hemorrhagic form is the most frequent in our series in 07 patients (70%) compared to the ischemic form found only in 03 cases (30%).

The posterior circulation is the most frequent in our series representing 70% of cases and 30% for the anterior circulation. The radiological signs found are as follows: occlusion in 03cases (30%) (Figures 1 and 2). Stenosis in 07 cases (70%) (Figures 2-4), dissecting aneurysm in 04 cases (40%) (Figures 1, 5 and 6). And tubular dilatation in 02 cases (20%). Images of dilated stenosis achieving a “pearl and string” appearance (Figures 3 and 4).

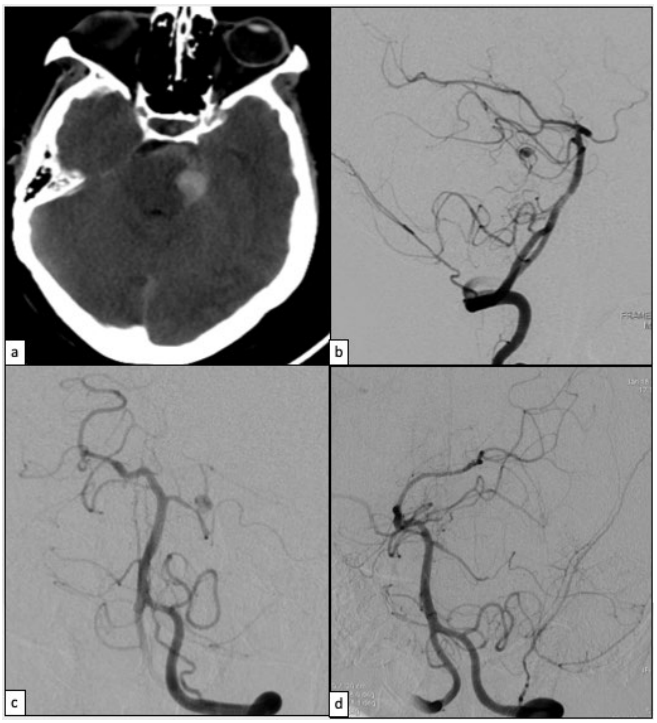


Figure 1: 49 years old patient with severe headache. **a:** Non enhanced axial brain CT: peri-mesencephalic and left sylvian fissure subarachnoid hemorrhage. **(b, c):** Brain angiography in frontal (b) and lateral (c) views: caliber disparity with aneurysm of the left superior cerebral artery. **d:** Brain angiography control 03 months later: disappearance of the aneurysm and the left superior cerebellar artery.

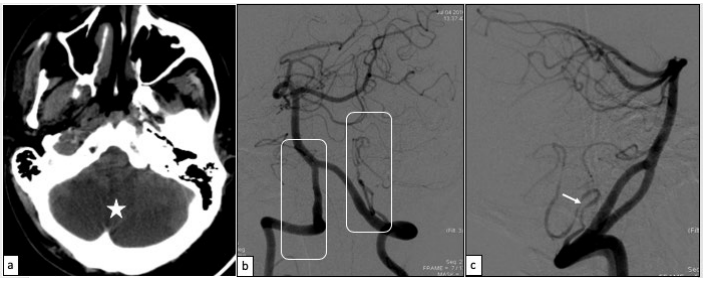


Figure 2: 60 years old patient, loss of consciousness. **(a):** Non enhanced brain CT: bilateral stroke of the PICA territories. **(b, c):** Brain angiographyin frontal (b) and lateral (c): long stenosis of the left PICA and total occlusion of the right PICA.

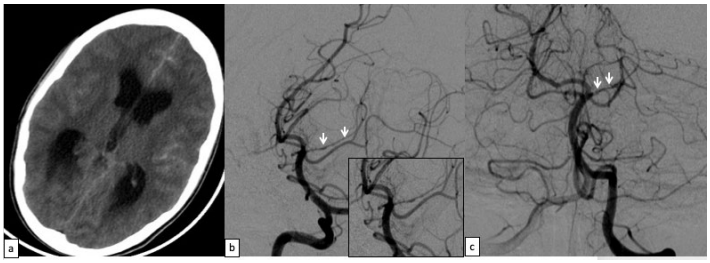


Figure 3: 16 years old patient, with intracranial hypertension. **(b, c):** Non enhanced brain CT: massive subarachnoid hemorrhage, brain angiography in lateral (b) and frontal (c) views: irregular and lobulated appearance of the arterial surface of the left posterior cerebral artery with reduction of its caliber.

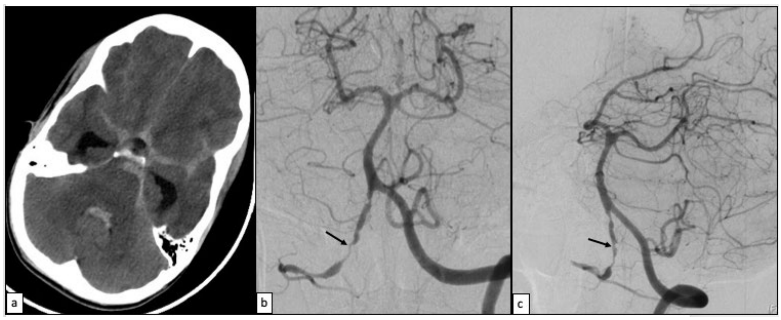
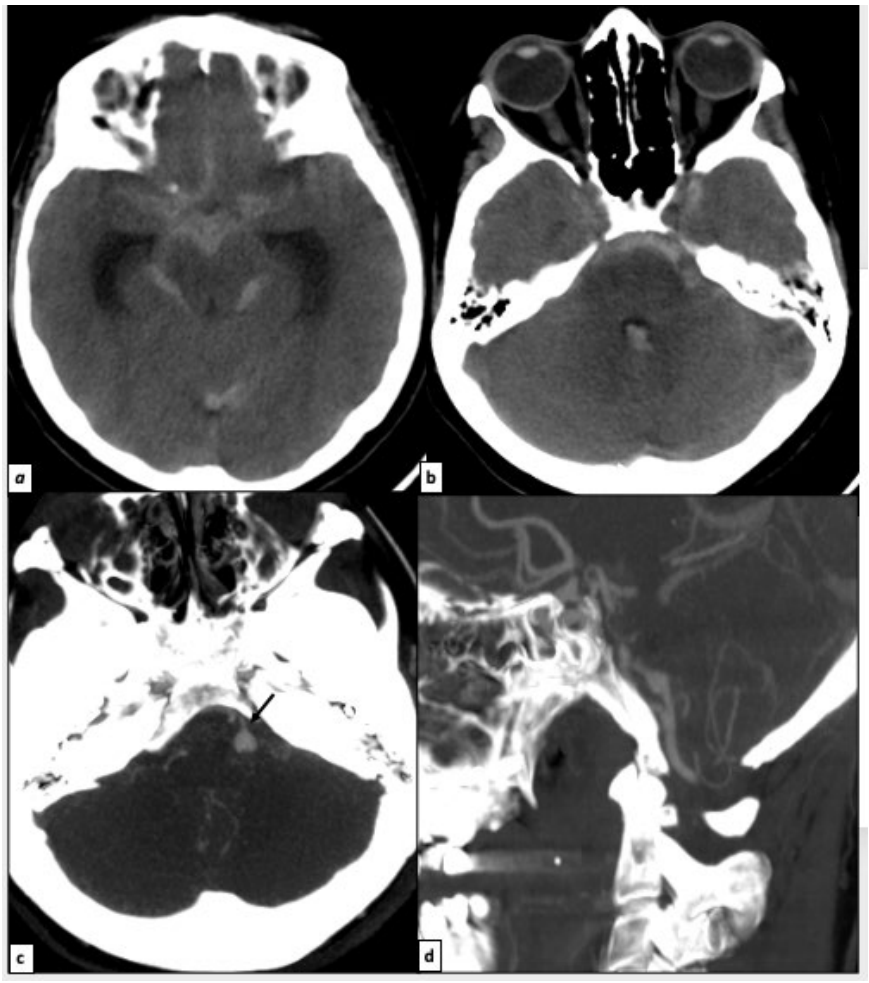


Figure 4: 45 years old patient. **(a):** Non enhanced brain CT: spontaneous subarachnoid hemorrhage. **(b, c):** Brain angiography showing a succession of stenoses and tubular dilations of the V4 segment of the right vertebral artery.

The association of a fusiform or irregular aneurysmal dilation associated with a segmental stenosis in (03 cases 30%) (Figures 5, 6). No case of intimal flap was identified.



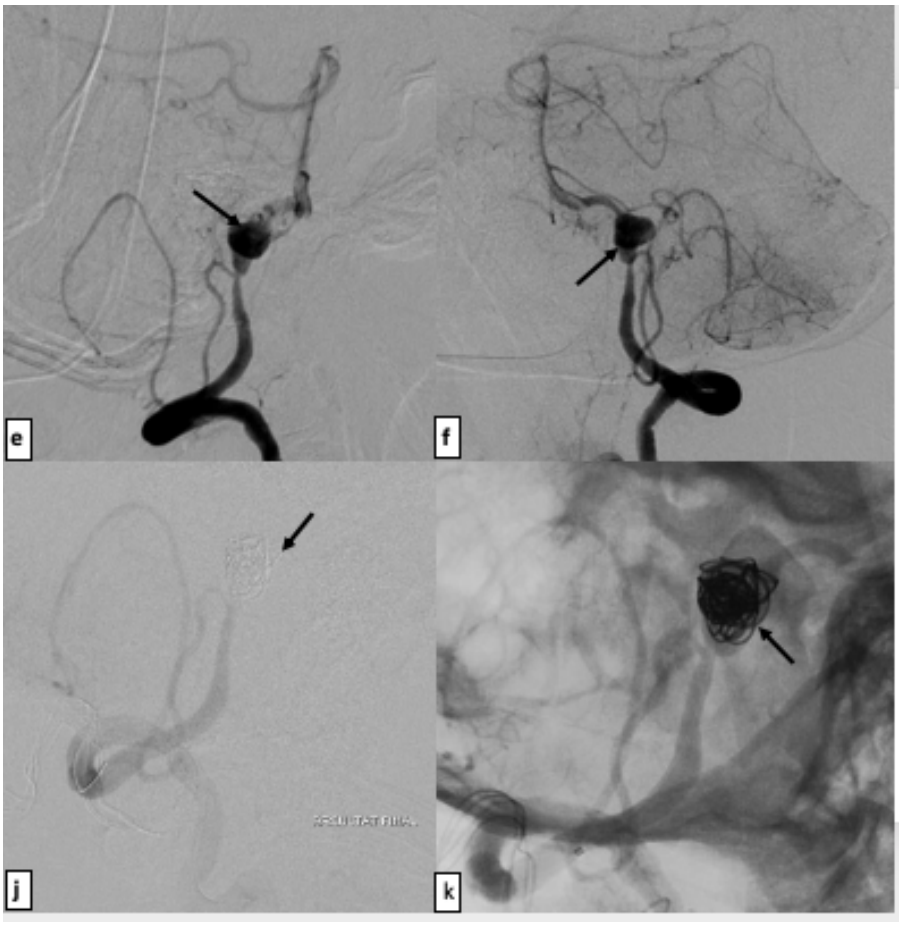


Figure 5: 45 years old patient with a loss of consciousness. **(a, b):** Axial non enhanced brain CT: peri mesencephalic and bilateral sylvien fissures subarachnoid hemorrhage and hydrocephalus. **(c, d):** Brain CT angiography in arterial phase in axial section (b) and sagittal section (c): narrowing of the left vertebral artery after the emergence of Posterior Inferior Cerebellar Artery (PICA) with individualization of a dissecting aneurysm. **(e, f, j, k):** Brain arteriography confirms the presence of a dissection of the vertebral artery, prior to the birth of PICA, complicated by a dissecting aneurysm. **(f, j):** Endovascular treatment in the same patient: embolization of the artery carrying the aneurysm by coils with extinction of the aneurysm and preserving the PICA.

Two patients benefited from endovascular treatment based on the occlusion of the aneurysm with coils (Figure 5), and occlusion the parent artery of aneurysm with ethylene vinyl alcohol copolymer (Figure 6). One other patient showed a spontaneous regression and disappearance of the aneurysm spontaneously.

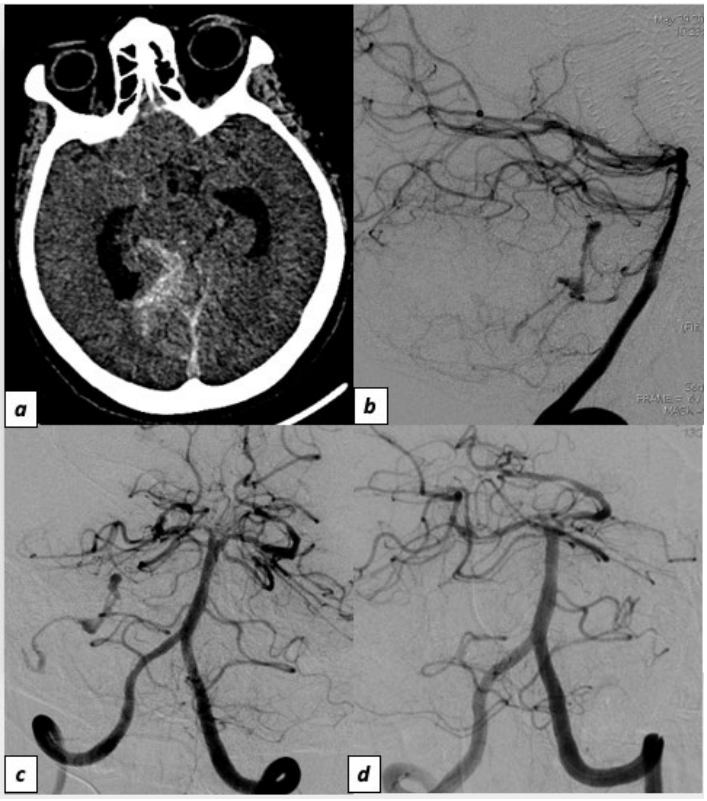


Figure 6: 72 years old patient, loss of consciousness. **(a):** Non enhanced brain CT: subarachnoid hemorrhage. (**b, c):** Brain angiographyin lateral (b) and frontal (c): stenosis of the right AICAwith aneurysm of the homolateral artery. **(d):** Endovascular treatment in the same patient: embolization of the artery carrying the aneurysm by onyx with extinction of the aneurysm.

Discussion

Exclusive intra cranial dissections are rare and less frequent than cervical dissection. They represent 2, 6 to 3 per 100,000 inhabitants per year [3]. They are more frequent in young population with an average age of 50 years. The clinical signs are variable and nonspecific. Intra cranial dissections has been lately more identified and the diagnosis became easier thanks to the development of less invasive investigation modalities.

Dissections are characterized by the leakage of blood within the wall of the artery, most often through an intimal breach. There are two types of dissections: a sub-intimal dissection and a sub-adventitial dissection (Figure 7). The histological structure of the intracranial arteries is different from the extra cranial arteries (thinner wall, disappearance of the external elastic limiting, thinning of the adventitia and of the media on the intracranial vessels), probably explaining the greater frequency of sub-adventitial intracranial dissections and the higher risk of hemorrhagic complications [4]. Risk factors for intracranial artery dissections are unknown. In the few studies including both patients with cervical artery dissection and patients with intracranial artery dissection, the distribution of vascular risk factors did not differ between the two groups [5]. Sometimes, a history of cranio-cervical trauma, fibro muscular dysplasia or genetic causes (Loeys-Dietz Syndrome, Marfan's Disease, Ehlers danlos Syndrome) were identified, but most of time there are no risk factors.

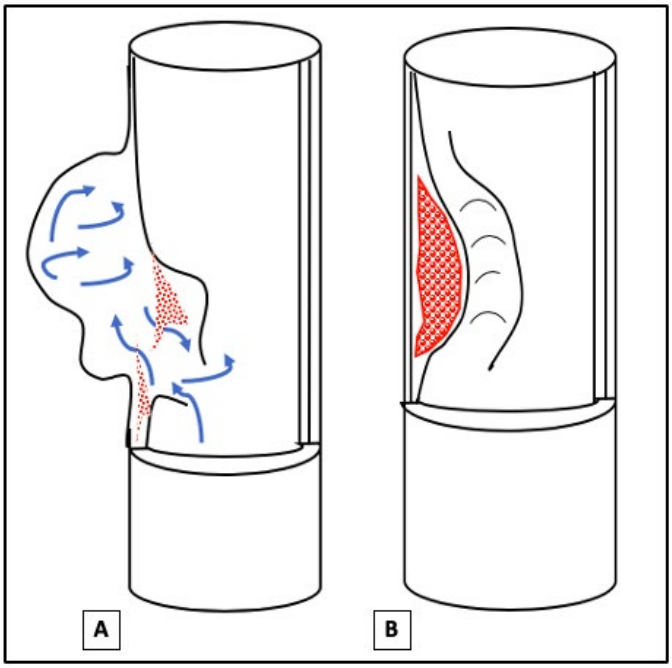


Figure 7: Arterial dissection anatomy. Schematic sagittal views of common patterns of injury. **A:** Sub-adventitial dissection: Extension of the arterial wall defect into the sub adventitial space leads to weakening and aneurysmal dilation of the adventitia. Arrows indicate turbulent flow of blood within the aneurysm. Note that adventitial tissue is present and forms the wall of the dissecting aneurysm. **B:** Sub-intimal dissection: intramural hematoma. Hemorrhage within the media with associated reduction in the cessel lumen. Modified.

Clinical symptoms are not specific, the two main revealing presentations are subarachnoid hemorrhage and cerebral ischemia. Intracranial dissections represent 50 to 60% of all the causes of subarachnoid hemorrhages. 30% to 78% of patients with dissection of the intracranial artery have cerebral ischemia (ischemic stroke or transient ischemic attack), without subarachnoid hemorrhage. About 80% of patients with intracranial artery dissection have prodromal headache, before subarachnoid hemorrhage or cerebral ischemia, and subsequently subarachnoid hemorrhage occurs within 3 days of onset of headache in 96 % patients [6].

The radiological diagnosis of intra cranial dissections is difficult given the small size of the intra cranial arteries

thus the analysis is difficult [7]. Pathognomonic radiological findings dissection includes an intimal flap, a wall hematoma, an aneurysmal dilatation, stenosis, occlusion and association with a focal stenosis. These signs are rarely found at the cranial level and more frequent at the arteries of the neck, butthe association of a fusiform or irregular aneurysmal dilation associated with a segmental stenosis is very suggestive of intracranial artery dissection [3,8]. Intramural hematoma can be demonstrated on T1 weighted Magnetic Resonance Imaging (MRI) as an area of increased signal intensity, while there is a thick ring-like or a railroad-like enhancement corresponding to the double lumen on contrast MRI or on angiography as seen in.

The presence of an intimal flap, with or without double lumen, is a subtle sign, which is mainly observed in the proximal arterial segments, and which is probably best detected by DSA. The mural hematoma generally leads to a regular crescent-shaped thickening of the arterial wall with a widening of the external diameter of the dissected artery and often a reduced and eccentric arterial lumen. It has been identified in more than 50% of patients in the literature [8]. Some studies have reported that aneurysmal dilations were more common in dissection of the intracranial artery with subarachnoid hemorrhage than in dissection of intracranial artery without subarachnoid hemorrhage [8,9]. A segmental stenosis or an occlusion found in 45 to 70% of cases of intracranial dissection. The stenosis can be manifested by images of dilated stenosis achieving a “pearl and string” appearance. It can also appear as a long and irregular stenosis with proximal dilatation and a resonance of the contrast in angiography.

In case of stenosis without dilatation it is difficult to distinguish between a dissection, atheromatous stenosis or a vasospasm. The occlusion can be total with the absence of visualization of an artery. This occlusion can appear early or during the evolution of the intra cranial dissection. In our series the radiological signs found are as follows: occlusion in 03 cases (30%), stenosis in 07 cases (70%), dissecting aneurysm in 04 cases (40%) and tubular dilatation in one case (20%). No case of intimal flap was identified.

Patients with intracranial artery dissection with subarachnoid hemorrhage are usually treated with surgical or endovascular procedures because the risk of rebreeding within the first days after the event representing up to 40% of patients. Patients with intracranial artery dissection without subarachnoid hemorrhage have been treated medically, and offered acute stroke treatment and long-term prevention of ischaemic stroke. Endovascular treatment is undertaken only in patients with recurrent ischaemic symptoms despite receiving optimum medical treatment. Sometimes, endovascular treatment is undertaken if the dissecting aneurysm has increased in size, to prevent rupture, or more rarely to reduce signs of brainstem compression.

Endovascular or surgical treatment aim to reduce blood flow in the dissected artery. We distinguish 02 techniques the first one is based on the parent artery occlusion with a risk of cerebral infarction. The second one consists in the occlusion of the aneurysm with clips or coils. As for saccular intracranial aneurysms, endovascular treatment is currently more frequently undertaken than is surgical treatment in most patients with intracranial artery dissection with subarachnoid hemorrhage. In patients with intracranial artery dissection without subarachnoid hemorrhage and no signs of cerebral ischemia, or in rare cases when both subarachnoid hemorrhage and cerebral ischemia are present, no antithrombotic treatment, but close monitoring has been proposed to detect a change in size or shape. The endovascular or surgical treatment of the aneurysm can be discussed if developed [10,11].

In our series two patients benefited from endovascular treatment based on the occlusion of the aneurysm with coil and occlusion the parent artery dissection with ethylene vinyl alcohol copolymer. One other patient showed a spontaneous regression and disappearance of the aneurysm spontaneously.

Conclusion

Cerebral arterial dissection is a serious disease characterized by acute onset. Diagnosis of intracranial artery dissection is often difficult because of non-specific clinical presentation. DSA, CTA and MR can be used in diagnosis with vascular and parenchyma imaging and also comparison between basic and follow-up imaging. These modalities show different findings in connection with cerebral arterial dissection. The typical description is the presence of a pearl-and-string sign, sometimes associated with a pseudo aneurysm. suggestions for treatment of intracranial artery dissection are different depending on clinical presentation cerebral damage.

Conflicts of Interest

This study was not affected by any conflicts of interest pertaining to the authors or our institute.

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