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Intracranial dissection: Incidence and long term endovascular treatment results of a not so rare disease

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ABSTRACT

Objective: The objective of this communication was to study the incidence and course of ICD and the long term outcome of this severe disease. The second goal was to analyze the different endovascular treatment modalities according to their long term results.
Methods: It is a retrospective analysis of 14 patients with ICD admitted in a single center in two consecutive years, treated with endovascular procedures. Patients harbouring blister-like ruptured intracranial aneurysms were excluded from this study.
Results: In this case series, 12 patients presented with subarachnoid hemorrhage (SAH) and 2 with brain stem symptoms. Mean age was 51 years and 13 patients were female. Six patients (43%) died and 8 (57%) survived with a mRS at 0–1. Mean follow up was 21 months.
Conclusions: ICD as a cause of SAH seems to be more frequent than previously thought and usually has a severe course. It requires a high level of suspicion to diagnosis and specific endovascular treatment modalities are required for each location to ensure long term stability and change the poor prognosis.

1. Introduction

Intracranial dissection (ICD) mostly presents with SAH, contrary to extra cranial dissections that mostly lead to ischemic and thrombo-embolic events. This is due to the thinner media and adventitia of intracranial vessels as well as their lack of external elastic lamina [1,2].

The diagnosis of ICD clinically or radiologicaly is difficult and it needs a high level of suspicion, especially when the history and onset are not typical or when it presents with symptoms other than SAH. SAH due to ICD requires aggressive treatment because rebleeding risk is higher than in aneurysmal subarachnoid hemorrhage and prognosis is poorer [3,4].

We present a series of 14 patients with ICD treated in our Department of Interventional Neuroradiology between January 2008 and December 2009. We discuss the incidence of ICD, the prognosis and long term outcome and the endovascular treatment options, in review of the recent literature.

2. Materials and methods

During 2 years between January 2008 and December 2009, 14 patients with ICD were treated in the Regional Interventional Neuroradiology Center of the Central University Hospital (CHU) of Tours. Mean age was 52 years (31–75), sex-ratio was 13/1 in favor of women.

In 12 patients (86%), the dissection was revealed by SAH, in one (7%) by brainstem compression and in the last one (7%) by brainstem infarct.

Five patients (38%) had been treated for arterial hypertension.

There were 10 patients presenting with initial Glasgow Coma Scale (GCS) above 10 and 4 patients with GCS between 3 and 9 (Table 1).

All patients underwent four-vessel digital subtraction angiography (DSA) after the initial CT. Architectural changes of suspicious but not diagnostic lesions on subsequent imaging, were suggestive of dissection and imposed an indication to treat.

All SAH patients were treated within 24 h from the onset. The intervention was performed under general anesthesia directly after the diagnostic angiography was carried out. Balloon test occlusion was not performed in our institution. All procedures were

Abbreviations: SAH, subarachnoid hemorrhage; ICD, intracranial dissection; BADA, basilar artery dissecting aneurysm; DSA, digital subtraction angiography; MRI, magnetic resonance imaging; HICVBD, hemorrhagic intracranial vertebrobasilar dissection; HICD, hemorrhagic intracranial dissection; mRS, Modified Rankin Scale; GCS, Glasgow Coma Scale; CT, computed tomography; PICA, posterior-inferior cerebellar artery; AICA, anterior-inferior cerebellar artery; MCA, middle cerebral artery; ACT, activated clotting time; VICD, vertebral intracranial dissection; WFNS, World Federation of Neurosurgical Societies; CT AG, computed tomographic angiography; MR AG, magnetic resonance angiography.

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Table 1
Patient characteristics, clinical presentation, treatment and 3-month outcomes.

Patients	Age	Gender	Follow-up	GCS	S	Loc	Treatment	Immediate outcome	mRS
1	46	F	1	15	H	C2	Co	D	6
2	34	F	24	9	H	TB	Co-AO	Diplopia	1
3 (Fig. 2)	52	F	5	7	H	V4	St	A	0
4	59	F	12	15	H	TB	Co-St	A	0
5	31	F	24	15	C	TB	CoSt-St	A	0
6 (Fig. 1)	47	F	12	15	I	TB	Co-St	Hp	3
7	64	M	24	15	H	V4	Tr	A	0
8 (Fig. 3)	69	F	18	14	H	V4	Tr	A	0
9	56	F	30	14	H	V4	Tr	A	0
10	44	F	24	15	H	V4	Tr	A	0
11	47	F	12	6	H	V4	Tr	ICU comp	6
12	40	F	1	15	H	TB	AO	D	6
13	59	F	1	8	H	V4	Tr	D	6
14	75	F	1	12	H	C1	CoSt	D	6

F, female; M, male; GCS, Glasgow Coma Scale; S, symptoms of presentation; H, hemorragic; C, compressive; I, ischemic; Loc, localisation; C2, second segment of internal carotid artery; TB, basilar trunk; V4, fourth (intracranial) segment of vertebral artery; PICA, posterior inferiorcerebellarartery; C1, first segment of internal carotid artery; Co, coiling alone; AO, artery occlusion; St, stenting alone; CoSt, coiling and stenting at once; Tr, trapping; D, early death from SAH; A, asymptomatic; Hp, hemiplegia; ICU comp, intensive care unit complication.

undertaken under systemic heparinisation (activated clotting time, ACT > 2).

SAH patients harbouring blister-like aneurysms as the source of hemorrhage were excluded from our study.

In the patients with unruptured dissecting lesions, endovascular therapy was limited to those with compressive enlargement of the aneurysmal dilatation or recurrent ischemic symptoms despite the proper anticoagulation treatment.

Immediate post-treatment outcome was evaluated by the modified Rankin Scale mRS. Follow-up was conducted with clinical examination, MRI and DSA imaging and ranged from 12 to 30 months (mean during of follow-up is 21 months) for the patients who survived.

3. Results

12 ICD with SAH represented 4.2% of the whole SAH and 20% of the "non-aneurysmal" cause SAH treated in our center during the same period. 10 patients with hemorrhagic intracranial vertebrobasilar dissections (HICVBD) represented 3.5% of the whole SAH and 17% of the "non-aneurysmal" cause SAH treated in our center.

12 patients had posterior circulation ICD, seven of them involved the posterior inferior cerebellar artery (PICA) (58%), five the basilar artery and the vertebral (V4) segment above PICA. Of the five patients with basilar artery dissection, 3 were revealed by SAH and 2 by brain stem compression or ischemic symptoms (Fig. 1).

2 patients had anterior circulation ICD involving the internal carotid artery above the petrous segment and in one of those, the dissection was extending through middle cerebral artery (MCA).

Among these 14 dissections, five presented pearl and string sign in DSA, three focal (symmetric or asymmetric) dilatations with proximal or distal stenosis, two displayed regular or irregular fusiform dilatation and four showed saccular lateral wall dilatation distant of any arterial bifurcation, but none showed a stenosis alone.

Initial endovascular treatment (Table 1) involved coiling alone in four cases, covered stenting in one case (Fig. 2), coiling and stenting in two cases, parent artery trapping in six cases and proximal occlusion in one case (Fig. 3). Sufficient collateral supply by the anterior inferior cerebellar artery (AICA) or the controlateral PICA was verified before the parent artery trapping.

Four of the patients died in the immediate post-treatment period (day 1–30 post-op), one during a retreatment at 10 months and one died one year after onset, never recovering from a posterior fossa infarct. This gives an overall mortality rate of 43% in our series. Of these six patients that died, four had posterior circulation dissections and two internal carotid dissections.

Mortality of HVBICD in particular was 30% with 67% of these patients presenting an initial GCS below 9 (Table 1).

The eight patients who survived had a mRS of 0–1 at discharge and one patient was presenting with persistent occulomotor palsy (7% morbidity).

Four of the patients (29%) required retreatment. They all had posterior circulation dissections and the preservation of the affected artery was the initial choice of treatment. Three of them underwent initial coiling alone and one coiling and stenting. In regular follow-ups they presented with progress of the affected vessel lesion and retreatment with overlapping stents has proven stable in the long term for 3 of them whereas for one, proximal occlusion was necessary.

We observed the reopening of a PICA which was treated by trapping of the parent artery with no initial filling or reopacification at first angiographic control.

From these four patients with initial immediate outcome of mRS at 0–3, one died during the retreatment 10 months later due to an iatrogenic hemorrhagic dissection in the posterior cerebral artery during a stent placement in the basilar artery.

4. Discussion

The prevalence of 14 patients treated with ICD in 2 consecutive years (4.2%) in a single regional hospital is elevated compared to similar or even multicentric studies in the literature [5]. In most single center studies in the literature, ICD accounts for approximately 2.2–2.6% of non-traumatic SAH [5,6]. In many communications authors do not refer to the percentage of hemorrhagic ICD compared to the total number of SAH patients, however, their numbers allow for a similar prevalence to be extracted [2,4].

We do not have any explanation about the higher incidence of ICD in our series. The fact that in our center all patients with SAH and negative CT AG undergo a DSA control has probably led us to detect more ICDs.

Also, repeating DSA in suspicious but non-diagnostic lesions is another factor that could increase the diagnosis of ICD. DSA remains the gold standard for the diagnosis of ICD. However, CT AG and MRI AG can most of the times detect intracranial vessel abnormalities that could correspond to ICD and thus impose the indication to perform a DSA control.

Female predominance (13/1) observed in our study is not usually reported. Most studies show a male predominance of ICD