Fig. 4. a Type 3 Takahashi anomaly. A hyperplastic AChorA supplies the parieto-occipital and calcarine branches, while the PCA proper gives rise to the temporal branches. **b** Type 4 Takahashi anomaly. The PCA territory is almost completely fed by a hyperplastic AChorA arising from the ICA.

the PCOM and rarely from the terminal ICA bifurcation or the proximal ICA before the take-off of the PCOM. Duplication of the AChorA has also been reported, with either separate or a common origin from the ICA [3]. On the other hand, absence of the AChorA is exceedingly rare [8]. Finally AChorA aneurysms represent 2–4% of all intracranial aneurysms [9]. Although associations between other vascular anomalies and aneurysms have been reported, the literature has not yet revealed an increased incidence of aneurysm formation in connection with a hyperplastic AChorA.

Conclusion

With the increasing utilization of MRA for the initial evaluation of the intracranial vasculature, noninterventional radiologists must become familiar with anatomic variants that may alter the anatomical distribution of brain ischemia and the surgical or endovascular approach for the management of intracranial pathology. We report 2 cases of anatomic variants of the anterior choroidal artery identified on 3D TOF MRA, which represent examples of type 3 and 4 Takahashi hyperplastic anterior choroidal arteries.

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Subarachnoid Hemorrhage from Spontaneous Dissection of the Anterior Cerebral Artery

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Dissection of intracranial arteries usually concerns the posterior circulation [1, 2]. We report 2 cases of subarachnoid hemorrhage (SAH) related to a spontaneous dissection of the anterior cerebral artery (ACA). We discuss the pathophysiology and management of this rare entity.

Case 1

A 65-year-old woman experienced severe acute headache without associated neurological signs except a slight aphasia. She had a history of arterial hypertension and had not suffered any

Fig. 1. **a** Brain CT scan showing an SAH prevailing in the left sylvian fissure. Digital subtraction angiography (**b**) and three-dimensional digital subtraction angiography (**c**) of the left internal carotid artery disclosing a typical aspect of dissection of the A1 segment of the left ACA (arrowheads). **d** Follow-up digital subtraction angiography on day 30 showing spontaneous regression of arterial wall abnormalities (arrowhead). **e** Magnetic resonance angiography at 6 months confirming the arterial healing of the left ACA (arrowhead).

recent cranial or cervical trauma. A brain CT scan (fig. 1a) showed an SAH prevailing in the left sylvian fissure. Cerebral angiography (fig. 1b, c) disclosed a severe narrowing followed by a fusiform aneurysm of the precommunicating segment (A1) of the left ACA. Control angiography on day 12 confirmed the stability of vascular anomalies, and the patient was treated conservatively. A third angiography on day 30 (fig. 1d) showed a spontaneous regression of arterial wall abnormalities, suggesting the diagnosis of arterial dissection. The patient was discharged with a normal neurological examination. Magnetic resonance angiography (MRA) at 6 months (fig. 1e) confirmed the arterial healing.

Case 2

A 41-year-old woman, with a history of autosomal dominant polycystic kidney disease, was examined in the outpatient department because of an asymptomatic anterior communicating artery (ACoA) aneurysm discovered on systematic MRA. A cerebral angiography was planned but the patient was admitted earlier as an emergency for sudden headache followed by generalized seizures. A brain CT scan (fig. 2a) showed a diffuse SAH. The cerebral angiography (fig. 2b, c) confirmed the ACoA aneurysm at the junction of the right A1 and postcommunicating (A2) segments and also showed an irregular aspect of the A1 segment of the right ACA. The patient was operated on with the diagnosis of ruptured ACoA aneurysm associated with focal vasospasm. At surgery, the aneurysm showed no sign of rupture and was clipped. The right A1 segment was dilated and presented a dark purplish discoloration of its wall, proving the spontaneous arterial dissection, and was wrapped. On a postoperative CT scan, ischemic lesions were noticed in the right ACA territory. Two weeks later, the patient experienced neurological deterioration related to rebleeding detected on a control CT scan (fig. 2d). Conservative treatment was

decided and no other hemorrhagic or ischemic complication occurred. She was discharged with a moderate disability (left lower limb weakness). MRA at 1 year (fig. 2e) confirmed the arterial healing.

Discussion

Spontaneous dissections of cerebral arteries tend to occur most commonly in the vertebrobasilar circulation [1–3]. Anterior circulation dissections affect more frequently the internal carotid artery (ICA) and the middle cerebral artery [1, 2, 4, 5]. Intracranial artery dissection may be related to connective tissue diseases affecting the arterial wall like fibromuscular dysplasia [6] or autosomal dominant polycystic kidney disease [7] (case 2). The primary lesion is always a widespread disruption of the internal elastic lamina followed by a disruption of the media leading to the formation of a pseudolumen [3]. The stenosis or the occlusion with thrombosis of the true lumen can result in cerebral infarction [1–5, 8]. Sometimes the dissected arterial wall can rupture either directly or at the site of a pseudoaneurysm causing an SAH [1–3, 9, 10]. The arterial wall spontaneously repairs with collagen proliferation from the first week and the healing becomes effective after 4–5 weeks [3]. Sometimes the patient experiences the recurrence of infarction or hemorrhage. The risk of hemorrhagic recurrence is higher during the first month and can reach 57% of patients [3].

The pathophysiology of the association between dissection and aneurysm is unclear. As illustrated in our case 1, the initial dissection may result in the development of a fusiform aneurysm that should be named ‘aneurysmal dissection’. It can be of two types: entry-exit or entry-only [3]. As there is no opening at the bottom of the pseudolumen, the latter is more instable and more prone to rupture. Sometimes a preexisting aneurysm, because of

Fig. 2. **a** Brain CT scan showing diffuse cisternal SAH. Digital subtraction angiography (**b**) and (**c**) three-dimensional digital subtraction angiography of the left internal carotid artery confirming the ACoA aneurysm located at the right A1-A2 junction (arrowheads) and showing an irregular aspect of the A1 segment of the right ACA (arrows). The right internal carotid artery was thrombosed. **d** CT scan showing a rebleeding two weeks after surgery. **e** MRA at 1 year confirming the arterial healing of the right ACA (arrowhead).

focal flow changes or arterial wall lesions, may become the starting point of the dissection itself and should be named 'dissecting aneurysm'. This hypothesis is supported in our case 2 by the presence of an ACoA aneurysm with a right ICA occlusion, responsible for a left-to-right flow across the ACoA to the ACA.

Dissection of the ACA remains a rare occurrence. In 2003, Ohkuma et al. [11] published a series of 18 ACA dissections collected from 46 stroke centers during a 5-year period and presented their neuroradiological and clinical features. Angiographic

findings may disclose classical aspects of dissection: double lumen sign, stenosis with dilation (pearl and string sign), stenosis and occlusion. Angiographic images can change during 2 months. From a clinical point of view, three groups of patients can be delineated: ischemic, bleeding and combined groups. Ischemic dissections [3, 5, 8, 11, 12] usually correspond to patients harboring stenotic lesions of the A2 segment of the ACA. They usually have a good outcome even though ischemic symptoms may worsen during the first month. Hemorrhagic dissections [3, 6, 8, 10, 11,

Table 1. Hemorrhagic dissections of the ACA: review of the literature

Author/date	Age years	Sex	Site	Angiography	Treatment	Rebleeding	Clinical outcome	Follow-up angiography or MRA
Gherardi and Lee [14], 1967	27	F	A1	FA	C	0	1	none
Guridi et al. [15], 1993	72	F	A3	normal	C	+	1	none
Yano et al. [26], 1995	27	M	A4	FA	T	0	5	OO
Okuno et al. [8], 1996	50	M	A2	DS	T	0	5	OO
Hayashi et al. [18], 1996	36	M	A1A2	DS + DL	C	0	4	none
Otawara et al. [23], 1997	66	F	A1	FA	T	0	3	NA
Lanzino et al. [10], 1997	51	F	A2	DS	C	0	5	I
Hashimoto et al. [16], 1999	61	M	A1	DS	clipping + W	0	5	NA
Amagasaki et al. [13], 1999	47	M	A3	FA + DL	C	+	3	parent artery embolization
Nomura et al. [6], 2001	5	M	A1	S	T	0	4	OO
Mizutani et al. [3], 2001	52	M	A2	FA	T + A3A3 bypass	0	5	OO
Miyahara et al. [21], 2001	50	M	A2	DS	W	0	4	NA
Hirao et al. [19], 2001	58	F	A1	FA	T	0	4	OO
Hatayama et al. [17], 2001	50	F	A1	DS + DL	T	0	NA	OO
Sakamoto et al. [24], 2002	65	F	A2	S + DL	C	0	NA	I
Mori et al. [22], 2002	48	M	A1	DL	W	0	4	I
	43	M	A2	FA + DL	W	0	5	I
Uhl et al. [25], 2003	45	F	A2	DS	W	0	5	none
Ohkuma et al. [11], 2003	39	F	A1	DS	clipping	+	2	I
	66	F	A1	DS	T	0	2	OO
	66	F	A1	DS	T	0	4	OO
	49	F	A2	S + DL	C	0	5	I
	53	M	A3	S + DL	C	0	5	I
	40	M	A2	S + DL	C	0	5	I
	64	F	A2	DS + DL	C	0	5	I
	49	F	A2	DS	surgery?	0	5	I
	49	M	A2A3	S + DL	C	0	5	I
Leach et al. [20], 2004	39	F	A1	D + DL	T	0	4	OO
Present cases, 2006	65	F	A1	DS	C	0	5	I
	41	F	A1	D	W	+	4	I

Mean age: 49.1 $\pm$ 14.2 years; sex ratio (M:F): 13:17 (0.76). F = Female; M = male; S = stenosis; D = dilatation; DS = 'pearl and string' sign; DL = double-lumen sign; FA = fusiform aneurysm; C = conservative; T = trapping; W = wrapping. The clinical outcome was evaluated with the Glasgow Outcome Scale: 1 = died; 2 = vegetative state; 3 = severe disability; 4 = moderate disability; 5 = good recovery. OO = Operatively occluded (dissected segment trapped); I = improvement; NA = nonavailable data.

13–26] usually concern patients with dilated dissection or fusiform aneurysm of any segment of the ACA. Their angiographic features, management modalities, outcome and evolution are summarized in table 1 reviewing the available literature.

The appropriate management of arterial dissection in the anterior circulation remains controversial. Dissections of the ACA with only ischemic onset are usually treated conservatively and have a good outcome [5, 11]. The use of heparinotherapy or anti-thrombotic therapy runs a risk of hemorrhagic transformation, but some authors used these treatments with success [4, 9]. Follow-up angiograms or MRA are mandatory as dynamic changes in the arterial wall could expose the patient to delayed SAH. When SAH has occurred (table 1), the conservative treatment can be effective with a good outcome and a low rate of second rupture [10, 11, 13–15, 18, 24]. If there is a high risk of rebleeding (rebleed-

ing under conservative treatment, growing dissecting aneurysm, giant dissecting aneurysm, traumatic dissecting aneurysm or dissection associated with uncontrolled hypertension), direct treatment of the dissection might be proposed. Several surgical techniques have been attempted such as wrapping [16, 21, 22, 25], trapping with [3] or without [6, 8, 11, 17, 19, 20, 23, 26] bypass surgery or clipping of the aneurysmal bulge [11, 16]. The endovascular treatment consists in proximal occlusion, obstruction of the aneurysmal bulge or stent placement [13]. In the series of Okhuma et al. [11], the 3 bleeding cases that underwent surgery (clipping or trapping of the dissected segment) had a very poor outcome and one of them presented a postoperative rebleeding. In contrast, the other 2 bleeding cases that were managed conservatively experienced a good recovery without recurrence of bleeding.

Conclusion

These 2 case reports emphasize the difficulties of the neuro-radiological diagnosis and of the decision-making for the management of hemorrhagic dissection of the ACA. The second patient was surgically treated but, unfortunately, the wrapping of the dissected arterial wall could not prevent the recurrence of the hemorrhage. On the other hand, the follow-up showed that the dissection tends to heal spontaneously with restoration of a normal arterial lumen and that the conservative treatment was effective with a good outcome in both cases. The literature review (table 1) and our own experience seem to suggest that this therapeutic option could be safely proposed for the management of hemorrhagic dissections of the ACA.

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Increased Oxygen Extraction Demonstrated on Gradient Echo (T2*) Imaging in a Patient with Acute Ischaemic Stroke

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Magnetic resonance (MR) imaging is sensitive to the oxidative state of haemoglobin, as oxy- and deoxyhaemoglobin have different magnetic properties – the blood oxygen level dependent (BOLD) effect. Indeed, functional MR imaging techniques are sufficiently sensitive to the BOLD effect that they can be used to assess cerebrovascular reactivity with a simple breath-holding test or CO₂ challenge [1].