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Supplementary webappendix

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Supplementary Material

Inclusion criteria for intracranial artery dissection patients in published series are listed in Supplementary Panel 1. While some studies included all types of intracranial artery dissection,[1-5,19-24] several studies have focused exclusively on vertebrobasilar intracranial artery dissection[6-15,27-35] intracranial artery dissection with subarachnoid haemorrhage[15,16,25,2632,37-45] or intracranial artery dissection without subarachnoid haemorrhage.[7,17,18,46,47] Some studies restricted the analysis to patients treated with a certain interventional approach.[7,9,13,16,22,24,28,30-34,37,40,42,43]

All studies were retrospective and based on less than 400 patients, and 37 of 50 studies (74·0%), comprising 2,592 out of 2,973 patients (87·2%) were conducted in Asia. In the main manuscript we are referring primarily to larger studies including 40 or more intracranial artery dissection patients, the smaller studies being reported in the supplementary appendix.

Supplementary Panel 1: Clinical criteria of included studies

Author, journal, year	Country	Inclusion period	Criteria
Studies including more than 40 patients			
Yamaura, Neuropathology 2000[1]	Japan	1995-1996	SAH, brain ischaemia or headache
Mizutani, J Neurosurg 2011[2]	Japan	1985-2008	IAD
Ono, Stroke 2013[3]	Japan	1980-2000	IAD
Kwak, Neurointerv 2011[4*]	Korea	2000-2007	CeAD and IAD
Metso, Stroke 2007[5]	Finland	1994-2004	CeAD with intracranial involvement and IAD
Ahn, Radiology 2012[6†]	Korea	2001-2010	symptomatic intracranial vertebrobasilar artery dissection
Kim, Stroke 2011[7†]	Korea	2001-2008	symptomatic vertebrobasilar artery IAD and aneurysm with endovascular treatment
Matsukawa, Cerebrovasc Dis 2012[8‡]	Japan	2003-2012	vertebral artery IAD
Kashiwasaki, Neuroradiology 2013[9]	Japan	1998-2011	vertebral artery IAD without PICA involvement treated with endovascular internal trapping
Takemoto, Acta Neurochir 2010[10]	Japan	1995-2007	symptomatic vertebral artery IAD
Shin, Eur Neurol 2014[11§]	South Korea, USA	2002-2010	symptomatic extra- and intracranial vertebral artery dissections
Nakazawa, Neurorad J 2011[12]	Japan	2004-2010	vertebral artery IAD
Jin, AJNR 2009[13*]	Korea	1997-2007	vertebrobasilar artery IAD and aneurysm with endovascular treatment
Zhao, Eur Radiol 2014 [14||]	China	2000-2011	Vertebral artery IAD and endovascular treatment
Nakajima, Acta Neurochir 2010[15]	Japan	2003-2006	SAH symptomatic vertebral artery IAD
Zhao, Plos One 2013[16||]	China	200-2011	SAH vertebral artery IAD treated with stent(s)-assisted coiling
Kim, Neurology 2011[7†]	Korea	2001-2008	symptomatic unruptured vertebrobasilar artery IAD
Kai, Neurosurgery 2011[17]	Japan	2003-2009	non-SAH symptomatic vertebral artery IAD
Matsukawa, JNNP 2014[18‡]	Japan	2003-2013	unruptured vertebral artery IAD with conservative treatment
Studies including 20-40 patients			
Lasjaunias, Childs Nerv Syst 2005[19]	France	not specified	intracranial aneurysm in children under 15 years
Mohammadian, Neurol Res Int 2013[20]	Iran	2008-2012	symptomatic IAD
Li, J Clin Neurosc 2011[21#]	Australia	2003-2008	symptomatic IAD
So, Clin Neurol Neurosurg 2014[22#]	Australia	2003-2011	IAD with endovascular treatment
Pelkonen, Acta Radiol 2004[23]	Finland	1982-2002	CeAD and IAD
Wakhloo, Stroke 2008[24]	USA	1996-2007	IAD and aneurysm with endovascular treatment
Anxionnat, Neurosurgery 2003[25]	France	1985-2000	SAH IAD
Wong, Surg Neurol Int 2010[26]	Hong Kong	2005-2010	SAH IAD

Rabinov, AJNR 2003[27]	USA	1992-2002	vertebrobasilar artery IAD
Hosoya, Stroke 1999[28]	Japan	1996-1997	vertebral artery IAD without PICA involvement treated with endovascular internal trapping
Lee, Yonsei Med 2007[29]	Korea	1992-2005	symptomatic vertebral artery IAD
Park, AJNR 2009[30†,§]	Korea	2001-2007	symptomatic vertebrobasilar artery IAD and aneurysm with stent-only therapy
Taha, Turk Neurosurg 2010[31]	Japan	1997-2006	vertebral artery IAD with endovascular treatment
Albuquerque, Neurosurg Focus 2005[32]	USA	1997-2005	symptomatic vertebral artery IAD with aneurysm and endovascular treatment
Lv, AJNR 2010[33]	China	2001-2007	symptomatic vertebral artery-PICA IAD with endovascular treatment
Yoon, Acta Neurochir 2010[34]	Korea	2003-2008	vertebrobasilar artery IAD with stent-alone treatment
Kim, AJNR 2008[35§]	Korea	2001-2007	symptomatic basilar artery IAD
Nam, JNIS 2014[36]	Korea	2005-2014	vertebrobasilar artery IAD with endovascular treatment
Endo, J Neurosurg 2013[37]	Japan	2006-2011	SAH vertebral artery IAD with endovascular treatment
Ramgren, Neurorad 2005[38]	Sweden	1993-2003	SAH vertebrobasilar artery IAD
Yuki, J Neurosurg 2005[39]	USA	1994-2003	SAH vertebrobasilar artery IAD
Shibukawa, Hiroshima J Med Sci 2009[40]	Japan	1998-2008	SAH vertebral artery IAD with endovascular treatment
Lee, Acta Neurochir 2010[41]	Korea	2001-2009	symptomatic ruptured and unruptured aneurysms in vertebral artery IAD
Sugiu, Neurorad 2005[42]	Japan	1992-2002	SAH vertebral artery IAD with surgical or endovascular treatment
Kurata, AJNR 2001[43]	Japan	1996-1999	SAH vertebral artery IAD with endovascular treatment
Yamada, J Neurosurg 2004[44]	Japan	1990-2000	SAH vertebral artery IAD with conservative treatment
Zhao, Acta Neurochir 2007[45]	France	1989-2006	SAH vertebrobasilar IAD
Arauz, Eur J Neurol 2013[46]	Mexico	1990-2011	symptomatic intra- and extracranial vertebral artery dissection with acute infarction, who did not undergo surgical or endovascular treatment
Naito, Neurosurg 2002[47]	Japan	1988-2001	vertebral artery IAD initially without SAH
Han, Eur Radiol 2014[48]	Korea	2012-2013	clinical symptoms and CTA suspicious of vertebrobasilar IAD
Oran, Diagn Interv Radiol 2009[49]	Turkey	2002-2008	SAH posterior circulation, non- vertebral artery IAD

SAH=subarachnoid haemorrhage. IAD=intracranial artery dissection. CeAD=cervical arterial dissection. PICA=posterior inferior cerebellar artery.
*, †, ‡, §, ||, # these series partly overlap

Supplementary Table 1: Clinical and radiological characteristics of IAD patients in published series including 20 to 40 patients

Paper	N	Origin	Imaging performed	Age, yrs (range)	Men	Location	Presenting symptoms
All types							
Lasjaunias[19] Childs Nerv Syst 2005	33	France Radiology	not specified	5·7 (8 days- 15 years) *SAH: 4·7* *non-SAH: 6·3*	57·6% *SAH: 46·2%* *non-SAH: 65·0%*	48·5% anterior; 51·1% posterior circulation *SAH: 38·5%/61·5%; non-SAH 55·0%/ 45·0%* ICA (10), ACoA (1), MCA (5), BA (6), VA (6), PICA (3), SCA (1), PCA (1)	SAH (13, 39·4%) headache alone (11, 33·3%) non-haemorrhagic deficit (8, 24·2%) asymptomatic (1, 3·0%)
Mohammadian[20] Neurol Res Int 2013	30	Iran Neurology Neurosurgery	DSA in all	50 (25-77) *SAH: 51* *non-SAH: 47*	53·3% *SAH: 52·0%* *non-SAH: 60·0%*	43·3% anterior; 56·7% posterior circulation *SAH: 44·0%/56·0%; non-SAH 40·0%/60·0%* ICA (5), MCA (5), ACA (3), VA (12), PCA (5)	SAH (25, 83·3%) cerebral infarction (2, 6·7%)
Li[21]* J Clin Neurosc 2011	25	Australia Neurology Neurosurgery Radiology	MRA, CTA, DSA	*SAH: 50* *non-SAH: 39*	40·0% *SAH: 38·5%* *non-SAH: 41·7%*	44·0% anterior; 56·0% posterior circulation *SAH: 23·1%/76·9%; non-SAH 66·7%/33·3%* ICA (11), VA (12), BA (2)	SAH (13, 52·0%) cerebral ischaemia (12, 48·0%)
So[22]* Clin Neurol Neurosurg 2014	23	Australia Neurology Radiology	DSA in all	47 (13-70) *SAH: 43* *non-SAH: 54*	43·5% *SAH: 31·3%* *non-SAH: 71·4%*	4·3% anterior; 95·7% posterior circulation *SAH: 6·3%/93·8%; non-SAH 0%/100%* ICA (1), VA (12), BA (3), PICA (4), AICA (1), PCA (2)	SAH (16, 69·6%) headache (3, 13·0%) cerebral ischaemia (2, 8·7%) asymptomatic (2, 8·7%)
Pelkonen[23] Acta Radiol 2004	22	Finland Radiology	DSA, MRA	43 (1-62)[§]	67·9%[§]	54·5% anterior; 45·5% posterior circulation *SAH: 0%/100%; non-SAH 70·6%/29·4%* ICA (9), MCA (3), VA (7), BA (1), PICA (1), PCA (1) 1 (4·8%) IAD in the VA was bilateral	SAH (5, 22·7%) *SAH and ischaemia (1, 4·5%)*
Wakhloo[24] Stroke 2008	20	USA Neurosurgery Radiology	DSA in all, MRI in some	51 (29-72) *SAH: 34* *non-SAH: 54*	30·0% *SAH: 66·7%* *non-SAH: 23·5%*	70·0% anterior; 30·0% posterior circulation *SAH: 33·3%/66·7%; non-SAH 76·5%/23·5%* ICA (9), MCA (4), VA (3), BA (4)	SAH (3, 15·0%) headache (5, 25·0%) *[presence of stroke not clearly specified]*
SAH IAD							
Anxionnat[25] Neurosurgery 2003	27	France Neurology Neurosurgery	DSA in all	48 (16-74)	48·1%	3·7% anterior; 96·3% posterior circulation ACA (1), VA (19), BA (2), PICA (2), SCA (2), PCA (2) 1 (3·7%) IAD in the VA was bilateral	SAH (27, 100%)
Wong[26] Surg Neurol Int 2010	23	Hong Kong Radiology	not specified	56	43·5%	34·8% anterior; 65·2% posterior circulation ICA (4), ACA (3), MCA (1), VA (15), PCA (1)	SAH (23, 100%)
Vertebrobasilar IAD							
Rabinov[27] AJNR 2003	34[‖]	USA Neurosurgery Radiology	not specified	51 (17-87) *SAH: 52* *non-SAH: 46*	64·7% *SAH: 65·5%* *non-SAH: 60·0%*	only vertebrobasilar IAD included	SAH (29, 85·3%) *SAH and stroke (5, 17·2%)* headache (2, 5·9%)

							stroke (3, 8·8%)
Hosoya[28] Stroke 1999	31	Japan Neurosurgery Radiology	DSA and MRI in all	55 (25-82)	64·5%	only vertebral IAD included 14 (45·2%) IADs were bilateral	SAH (3, 9·7%) headache alone (1, 3·2%) cerebral ischaemia (15, 48·4%)
Lee[29] Yonsei Med J 2007	28	Korea Neurosurgery Radiology	CT and MRI in all, MRA in some	52 (34-74) *SAH: 51* *non-SAH: 52*	50·0% *SAH: 39·1%* *non-SAH: 100%*	only vertebral IAD included	SAH (23, 82·1%) cerebral ischaemia (5, 17·9%)
Park[30†,‡] AJNR 2009	27	Korea Neurosurgery Radiology	CT/CTA, MRI/MRA, DSA	46 (24-71) *SAH: 48* *non-SAH: 44*	63·0% *SAH: 54·5%* *non-SAH: 68·8%*	only vertebrobasilar IAD included	SAH (11, 40·7%) headache alone (10, 37·0%) cerebral ischaemia (5, 18·5%) mass effect (1, 3·7%)
Taha[31] Turk Neurosurg 2010	25	Japan Neurosurgery Radiology	DSA, MRA, CTA	50 (38-66) *SAH: 52* *non-SAH: 48*	77·8% *SAH: 76·5%* *non-SAH: 100%*	only vertebral IAD included	SAH (17, 68·0%) headache (1, 4·0%) cerebral ischaemia (1, 4·0%)
Albuquerque[32] Neurosurg Focus 2003	23	USA Neurosurgery	DSA in all	49 (35-72)	43·5%	only vertebrobasilar IAD included	SAH (17, 73·9%) cerebral ischaemia (2, 8·7%) headache (3, 13·0%)
Lv[33] AJNR 2010	22	China Neurosurgery	DSA in all, CT and MR in some	43 (12-59) *SAH: 44* *non-SAH: 41*	68·2% *SAH: 62·5%* *non-SAH: 83·3%*	only vertebral IAD included	SAH (16, 72·7%) headache (2, 9·1%) cerebral ischaemia (4, 18·2%)
Yoon[34] Acta Neurochir 2010	22	Korea Neurosurgery	DSA, CTA, MRI in all	50 (24-65)	50%	only vertebrobasilar IAD included 2 (9·1%) IADs were bilateral VA (23), BA (1)	SAH (6, 27·3%) cerebral ischaemia (2, 9·1%) headache (5, 22·7%) asymptomatic (3, 13·6%)
Kim[35†] AJNR 2008	21	Korea Neurosurgery Radiology	CT/CTA, MRI/MRA, DSA	53 (24-78)	57·1%	only basilar IAD included	SAH (10, 47·6%) cerebral ischaemia (10, 47·6%) mass effect (1, 4·8%)
Nam[36] JNIS 2014	26	Korea Neurosurgery Radiology	DSA and CTA in all haemorrhagic patients, MRI in non-haemorrhagic patients	54 (34-70)	50·0%	only vertebrobasilar IAD included	SAH (14, 53·8%)
SAH vertebrobasilar IAD							
Endo[37] J Neurosurg 2013	38	Japan Neurosurgery Radiology	DSA in all	53 (30-82)	50·0%	only vertebral IAD included	SAH (38, 100%)
Ramgren[38] Neurorad 2005	29	Sweden Radiology	DSA in all	55 (41-73)	34·5%	only vertebrobasilar IAD included VA (23), BA (4), PICA (2)	SAH (29, 100%)
Yuki[39] J Neurosurg	29	USA Radiology	DSA in all	45	44·8%	only vertebrobasilar IAD included VA (27), BA (2)	SAH (29, 100%)

2005							
Shibukawa[40] Hiroshima J Med Sci 2009	26	Japan Neurosurgery	DSA in all	52 (36-74)	61·5%	only vertebral IAD included	SAH (26, 100%)
Lee[41] Acta Neurochir 2010	25	Korea Neurosurgery Radiology	DSA in all	45 (22-66)	56·0%	only vertebral IAD included	SAH (25, 100%)
Sugiu[42] Neurorad 2005	25	Japan Neurosurgery	not reported	55 (37-73)	68·0%	only vertebral IAD included 0 IADs were bilateral	SAH (25, 100%)
Kurata[43] AJNR 2001	24	Japan Neurosurgery	DSA in all	54 (35-71)	79·2%	only vertebral IAD included	SAH (24, 100%)
Yamada[44] J Neurosurg 2004	24	Japan Neurosurgery	DSA in most	51 (35-71)	66·7%	only vertebral IAD included	SAH (24, 100%)
Zhao[45] Acta Neurochir 2007	21	France Radiology	DSA in all	43 (6-67)	52·4%	only vertebrobasilar IAD included 2 (9·5%) IADs were bilateral VA (16), BA (5)	SAH (21, 100%)
Non-SAH vertebrobasilar IAD							
Arauz[46] Eur J Neurol 2013	27	Mexico Neurology	DSA (62%), CTA (22%), MRA (16%)[#]	40	66·7%	only vertebrobasilar non-SAH IAD included	ischaemic stroke (27, 100%)
Naito[47] Neurosurg 2002	21	Japan Neurosurgery	DSA in all	50 (33-68)	61·9%	only vertebral IAD non-SAH at first presentation included	cerebral ischaemia (11, 52·4%) headache or neckpain (9, 42·9%) vertigo (1, 4·8%)
Han[48] Eur Radiol 2014	33	Korea Neurology Radiology	MRI /MRA and CTA in all, DSA in most	51 (29-77)	84·8%	only vertebrobasilar IAD included 2 (6%) bilateral IAD VA (25), BA (1), PICA (9)	cerebral ischaemia (28, 84·9%) local symptoms (5, 15·2%)
SAH posterior circulation non-vertebral artery IAD							
Oran[49] Diagn Interv Radiol 2009	23	Turkey Radiology	DSA in all, MRA (1)	52 (36-65)	30·4%	only posterior circulation SAH included BA (6), PCA (7), PICA (5), SCA (5)	SAH (23, 100%)

DSA=digital subtraction angiography. SAH=subarachnoid haemorrhage. ICA=internal carotid artery. MCA=middle cerebral artery. ACA=anterior cerebral artery. VA=vertebral artery. BA=basilar artery. PICA=posterior inferior cerebellar artery. PCoA=posterior communicating artery. SCA=superior cerebellar artery. PCA= posterior cerebral artery. IAD=intracranial artery dissection.

[*], [†] these series partly overlap

[‡] series overlaps partly with Ahn, Radiology 2012; Kim, Stroke 2011; Kim, Neurology 2011

[§] numbers and percentages only reported for all patients with intra- and extracranial dissection (N=136), and not for subgroup of patients with IAD (N=22)

|| one trauma patient excluded
percentages only reported for all patients with intra- and extracranial dissection (N=110), and not for subgroup of patients with IAD (N=27)

Supplementary Table 2: Radiological criteria used for diagnosis of IAD in published series including more than 40 patients

Paper	Radiological criteria IAD
Studies including more than 40 patients	
Yamaura[1] Neuropathology 2000	- tapered narrowing (string sign) or occlusion - aneurysmal outpouching (fusiform dilation) - intimal flap* - retention of contrast media into false lumen - combination of these findings like pearl and string sign
Mizutani[2] J Neurosurg 2011	- irregular stenosis - segmental stenosis and aneurysm formation (pearl and string) - irregular fusiform or aneurysmal dilation - double lumen* - occlusion - absence of markedly dilated lesions with significant elongation and tortuosity of the parent vessels - serial geometric change (on repeated angiographies)
Ono[3] Stroke 2013	- typical pearl and string or double lumen sign at a non-branching site of the intracranial cerebral arteries on DSA - fusiform dilation with retention of contrast medium or angiographic steno-occlusive lesions accompanied by intramural haemorrhage detected on MRI at the same region on DSA*
Kwak[4] Neurointerv 2011	- double lumen* - luminal narrowing >30% - gradual tapering ending in total occlusion - aneurysmal pattern
Metso[5] Stroke 2007	- intramural haematoma* - intima flap or double lumen* - long filiform stenosis - occlusion that recanalisedsed into a long filiform stenosis (if in ICA, occlusion > 2 cm above carotid bifurcation) - rat tail-shaped or flame-like occlusion - fusiform aneurysm with arterial wall irregularity and no notable atherosclerosis, causing SAH
Ahn [6] Radiology 2012	- aneurysmal dilation of the intracranial vertebrobasilar arterial trunk - pearl-and-string sign (aneurysmal dilation alternating with stenosis) - tapered steno-occlusion - intramural haematoma* - intimal flap* - double lumen*
Kim[7] Stroke 2011	- aneurysmal dilation of the intracranial vertebrobasilar arterial trunk - pearl-and-string sign
Matsukawa[8] Cerebrovasc Dis 2012	- intramural haematoma on fat suppression T1-weighted MRI or MRA* - intimal flap* - double lumen*

	- string sign (smoothly tapered steno-occlusive lesion) - pearl-and-string sign in the absence of atherosclerotic lesions - aneurysmal dilation of the vertebral artery trunk not located at an arterial branching point and associated with sudden headache or posterior ischaemic symptoms
Kashiwasaki[9] Neuroradiology 2013	- not specified
Takemoto[10] Acta Neurochir 2010	- pearl-and-string sign - string sign - tapered occlusion - intimal flap* - intramural haematoma on T1 weighted image*
Shin[11] Eur Neurol 2014	- on DSA: double lumen (false lumen or intimal flap)*, stenosis involving an irregular long or short segment (pearl-and-string sign), occlusion of either the complete artery or a segment, or a pseudoaneurysm associated with a narrowed arterial lumen - on CTA: a narrowed centric or eccentric lumen surrounded by crescent-shaped mural thickening (i.e. acute thrombus sign in false lumen) and an associated increase in the external diameter*, an abrupt or tapered occlusive lumen with mural thickening and an associated increase in the external diameter, or an aneurysmally dilated lumen or a dilated and narrowed lumen with or without crescent-shaped mural thickening or an intimal flap - on MRA: long stenotic segments consistent with the 'string sign', tapered stenosis or occlusion, pseudoaneurysm, intimal flap formation and luminal irregularity*, or the presence of a wall haematoma*
Nakazawa[12] Neurorad J 2011	- intimal flap or double lumen on angiogram* - intimal flap or double lumen on CTA* - haematoma in the arterial wall on T1-weighted image of MRI* - dilation and stenosis, retention of contrast media, string sign, pearl sign, tapered occlusion on angiogram - dilation and stenosis on MRA or CTA - intimal flap or double lumen on MRI, MRA; Enhanced volume T1WI* - fusiform dilation of parent artery on angiogram, MRA, CTA - chronological changes of the radiological findings and no cause except for dissection
Jin[13] AJNR 2009	- not specified
Zhao[14] Eur Radiol 2014	- not specified
Nakajima[15] Acta Neurochir 2010	- not specified
Zhao[16] Plos One 2013	- fusiform dilation without stenosis in the affected vertebral trunk portion - irregular lateral dilation without stenosis in the affected vertebral trunk portion - fusiform or irregular dilation with stenosis ("pearl and string sign")
Kim[50] Neurology 2011	- intramural haematoma on fat-suppression T1-weighted MR or MR angiogram source images* - intimal flap*

	- double lumen sign*
	- string sign (smoothly tapered steno-occlusive lesion) or pearl-and-string sign, without any atherosclerotic change of the involved artery
	- fusiform or irregular aneurysmal dilation of arterial trunk not located at arterial branching point, but associated with sudden onset of severe pulsatile headache or posterior ischaemic symptoms
Kai[17] Neurosurgery 2011	- long stenotic segments exhibiting the ‘‘string sign’’
	- tapered stenosis or occlusion
	- pseudoaneurysm
	- intimal flap formation*
	- luminal irregularity
	- crescent-shaped high signal intensity within a vessel wall (mural haematoma or double lumen)*
Matsukawa[18] JNNP 2014	Major criteria
	- ‘Double lumen’ or ‘intimal flap’ demonstrated on either DSA, MRI, MRA, CTA or duplex ultrasonography*
	- ‘Pearl and string sign’ or ‘string sign’ demonstrated on DSA
	- Pathological confirmation on arterial dissection*
	Minor criteria
	- ‘Pearl sign’ or ‘tapered occlusion’ demonstrated on DSA
	- ‘Pearl and string sign’, ‘string sign’ or ‘tapered occlusion’ demonstrated on MRA
	- ‘Hyperintense intramural signal’ (corresponding to intramural haematoma) demonstrated on T1-weighted MRI*
	Additional criteria
	- Change in arterial shape demonstrated on either DSA, MRI, MRA, CTA or duplex ultrasonography
	- No other causes of arterial abnormalities
	Definite dissection
	□Presence of one or more major criteria, or presence of one or more minor criteria and both of two additional criteria
	Probable dissection
	□Presence of one or more minor criteria

DSA=digital subtraction angiography
* these criteria fulfil the proposed radiological criteria for a definite intracranial artery dissection (Panel 2)

Supplementary Table 3: Treatment and outcome of IAD patients in published series including 20 to 40 patients

Paper	N	Treatment	Follow-up	Death	Good functional outcome†	Symptomatic recurrences or complications
All types						
Lasjaunias[19] Childs Nerv Syst 2005	33	*SAH:* 76·9% surgical or endovascular treatment 15·4% no treatment 7·7% unknown *non-SAH* 45·0% surgical or endovascular treatment 10·0% medical treatment with aspirin 15·0% no treatment 30·0% unknown	not specified	11·1% (2/18) *SAH: 22·2%* *non-SAH: 0%*	not reported	16·7% SAH *SAH: 23·1% rebleeding*
Mohammadian[20] Neurol Res Int 2013	30	*SAH:* 100% endovascular treatment *non-SAH* 100% surgical or endovascular treatment	17 months (6-33 months)	3·3% *SAH: 4·0%* *non-SAH: 0%*	mRS≤3 93·3% *SAH: 92%* *non-SAH: 100%*	0% SAH 20·0% cerebral ischaemia (peri-procedural)
Li[21*] J Clin Neurosc 2011	25	*SAH:* 76·9% surgical or endovascular treatment 23·1% unknown *non-SAH* 66·7% medical treatment with aspirin 16·7% anticoagulation treatment 16·7% surgical or endovascular treatment	90 days	12·0% *SAH: 7·7%* *non-SAH: 16·7%*	mRS ≤3 64·0% *SAH: 46·2%* *non-SAH: 83·3%*	0% SAH 0% cerebral ischaemia
So[22*] Clin Neurol Neurosurg 2014	23	*SAH:* 100% endovascular treatment *non-SAH* 100% endovascular treatment	16 months[§] (0-82 months)	17·4% *SAH: 25·0%* *non-SAH: 0%*	mRS≤1 65·2%	17·4% SAH *SAH: 25·0% rebleeding pre-treatment* 13·0% cerebral ischaemia *SAH: 12·5% cerebral ischaemia (peri-procedural)* *non-SAH: 14·3% cerebral ischaemia (peri-procedural)*
Pelkonen[23] Acta Radiol 2004	22	not reported	not reported	not reported	not reported	not reported
Wakhloo[24] Stroke 2008	20	*SAH:* 100% endovascular treatment *non-SAH* 100% endovascular treatment	20·6 months (3-156 months)	0% *SAH: 0%* *non-SAH: 0%*	not specified 65% asymptomatic	5·0% SAH *non-SAH: 5·9% SAH due to wire perforation* 10·0% cerebral ischaemia *non-SAH: 11·8% cerebral ischaemia*
SAH IAD						

Anxionnat[25] Neurosurgery 2003	27	63·0% endovascular treatment 37·0% treated "conservatively" (no details)	not defined	14·8%	not defined 63·0%	11·1% rebleeding (at 8·3 day [1-13]) 7·4% ischaemic stroke (periprocedural)
Wong[26] Surg Neurol Int 2010	23	100% surgical or endovascular treatment	28 months (1-53 months)	8·7%	mRS≤2 60·9%	8·7% rebleeding post-treatment 4·3% ischaemic stroke (peri-procedural) 4·3% haemorrhagic stroke (peri-procedural)
Vertebrobasilar IAD						
Rabinov[27] AJNR 2003	34	*SAH:* 79·3% surgical or endovascular treatment 21·7% no treatment *non-SAH:* 100% surgical or endovascular treatment 100% endovascular treatment	not reported	20·6% *SAH: 24·1%* *non-SAH: 0%*	mRS≤1 53·3% *SAH: 48·0%* *non-SAH: 80·0%*	13·3% SAH *SAH: 6·9% rebleeding* 2·9% cerebral ischaemia due to extension of contralateral IAD *SAH: 3·4% cerebral ischaemia*
Hosoya[28] Stroke 1999	31	80·6% antihypertensives and/or oral anticoagulants 9·7% endovascular treatment 9·7% surgical treatment	not reported	0%	"good recovery" 74·1%	not reported
Lee[29] Yonsei Med J 2007	28	100% surgical or endovascular treatment	6 months	3·6% *SAH: 4·3%* *non-SAH: 0%*	GOS 4-5 98·3% *SAH: 87·0%* *non-SAH: 100%*	0 % SAH 4·8% postoperative infection of cerebrospinal fluid collection
Park[30†,‡] AJNR 2009	27	100% endovascular treatment	28 months (7-50 months)	3·7% *SAH: 9·1%* *non-SAH: 0%*	mRS≤1 77·8% *SAH: 63·6%* *non-SAH: 87·5%*	3·7% SAH *SAH:9·1% rebleeding post-treatment*
Taha[31] Turk Neurosurg 2010	25	100% endovascular treatment	61·8 months (13- 120 months)	12·0% *SAH: 23·5%* *non-SAH: 0%*	GOS 4-5 84·0% *SAH: 23·5%* *non-SAH: 100%*	4·0% SAH *SAH: 5·9% rebleeding post-treatment* 17·6% cerebral infarction *5·9% asymptomatic cerebral infarction*
Albuquerque[32] Neurosurg Focus 2003	23	100% endovascular treatment	14·6 months (1-49 months)	8·7% *SAH: 11·8%* *non-SAH: 0%*	"good recovery" 90·9%	4·3% asymptomatic recurrence (no details on type, retreated) 4·3% headache due to recurrence (no details on type, treated)
Lv[33] AJNR 2010	22	100% endovascular treatment	37 months (6-84 months)	0%	GOS 5 100%	0% SAH 0% cerebral infarction
Yoon[34] Acta Neurochir 2010	22	100% endovascular treatment	16 months (1-60 months)	0%	mRS≤1 95·5% *SAH: 83·3%*	0% SAH 9·1% proximal artery dissection during procedure

					non-SAH: 100%	
Kim[35†] AJNR 2008	21	*SAH:* 30·0% antihypertensives and/or anticoagulants 70·0% endovascular treatment *non-SAH:* 63·6% antihypertensives and/or anticoagulants 36·4% endovascular treatment	21·5 months (5-50 months)	14·3% *SAH: 30·0%* *non-SAH: 0%*	mRS≤2 71·4% *SAH: 60·0%* *non-SAH: 45·5%*	19·0% SAH *SAH:40·0% rebleeding* 4·8% brain stem compression (after 3 years, treated)
Nam[36] JNIS 2014	26	100% endovascular treatment	32·6 months (0-82 months)	11·5% *SAH : 21·4%* *non-SAH : 0%*	mRS ≤1 84·6% *SAH: 71·4%* *non-SAH : 100%*	0% SAH
SAH vertebrobasilar IAD						
Endo[37] J Neurosurg 2013	38	*SAH only* 100% surgical or endovascular treatment	6 months	15·8%	mRS≤2 60·5%	*SAH only* 39·5% rebleeding pre-treatment (9 not confirmed on imaging) 2·6% ischaemic stroke post-treatment
Ramgren[38] Neurorad 2005	29	*SAH only* 51·7% endovascular treatment 48·3% treated "conservatively"	6 months	27·6%	GOS 5 55·2%	*SAH only* 31·0% rebleeding (pre-treatment, within 12 days) 6·9% cerebral ischaemia (TIA, periprocedural) 6·9% asymptomatic rupture IAD peri-procedural
Yuki[39] J Neurosurg 2005	29	*SAH only* 100% endovascular treatment	23 months[‖] (1-132 months)	17·2%	mRS≤2 65·5%	*SAH only* 6·9% rebleeding (1 before and 1 after treatment) 3·4% abcess at surgical site
Shibukawa[40] Hiroshima J Med Sci 2009	26	*SAH only* 100% endovascular treatment	not reported	19·2%	GOS 5 46·2%	*SAH only* 0% rebleeding after endovascular treatment
Lee[41] Acta Neurochir 2010	25	*SAH only* 100% endovascular treatment	44·7 months (3-93 months)	24·0%	GOS 4-5 68·0%	*SAH only* 48·0% rebleeding *44·0% rebleeding pre-treatment (mean 20·7 hours, (1-74))* *4·0% rebleeding post-treatment (day 20)* 4·0% asymptomatic ischaemia
Sugiu[42] Neurorad 2005	25	*SAH only* 100% surgical or endovascular treatment	not reported	16·0%	GOS 5 68·0%	*SAH only* 48·0% rebleeding (12 pre-treatment, 1 during treatment) *44·0% within 24 hours* 0% cerebral ischaemia
Kurata[43] AJNR 2001	24	*SAH only* 87·5% endovascular treatment 8·3% treated "conservatively" (no details)	9 months (1 week- 39 months)	33·3%	GOS 5 54·2%	*SAH only* 58·3% rebleeding pre-treatment (92·9% within first 24 hours) 8·3% ischaemic stroke

		16·7% no treatment				
Yamada[44] J Neurosurg 2004	24	*SAH only* 100% treated "conservatively" (no details), because of delayed diagnosis, bilateral VA lesion, spontaneous occlusion of VA at aneurysm, or poor clinical condition	8·3 years	66·7%	GOS 5 33·3%	*SAH only* 45·8% rebleeding 4·2% symptomatic vasospasm
Zhao[45] Acta Neurochir 2007	21	*SAH only* 66·7% surgical or endovascular treatment 33·3% treated "conservatively" (no details)	13·8 months	23·8%	"good recovery" 57·1%	*SAH only* 14·3% rebleeding 4·8% recurrent dissection (16 months) 14·3% cerebral ischaemia
Non-SAH vertebrobasilar IAD						
Arauz[46] Eur J Neurol 2013	27	*non-SAH only* 59·3% with anticoagulants 40·7% with antiplatelet agents	46·4 months[#]	*non-SAH only* not reported	mRS≤2 *non-SAH only* 70·4%	*non-SAH only* no SAH no recurrence in this group
Naito[47] Neurosurgery 2002	21	*non-SAH only* 42·9% endovascular treatment due to enlargement of aneurysm (2), large aneurysm (2), progression of dissection (1), persistent double lumen (1) 57·1% treated "conservatively" (no details)	14 months (4 days- 88 months)	*non-SAH only* 0%	"good recovery" 85·7%	*non-SAH only* 14·3% SAH (mean 17 months, (1 day- 51 months)) 0% cerebral ischaemia
Han[48] Eur Radiol 2014	33	*Non-SAH only* 93·9% medical treatment 6·0% coil embolisation	3 months	*non-SAH only* not reported	not reported	not reported
SAH posterior circulation non-vertebral artery IAD						
Oran[49] Diagn Interv Radiol 2009	23	*SAH only* 100% endovascular treatment 91·3% endovascular treatment in acute phase	25·5 months (20 days- 84 months)	*SAH only* 4·3%	GOS 5 47·8%	*SAH only* 4·3% rebleeding post-treatment 21·7% cerebral ischaemia 8·7% TIA

SAH=subarachnoid haemorrhage. mRS=modified Rankin scale score. IAD=intracranial artery dissection. GOS= Glasgow Outcome Scale. TIA=transient ischaemic attack.

[*, †] these series partly overlap

[‡] series overlaps partly with Ahn, Radiology 2012; Kim, Stroke 2011; Kim, Neurology 2011

[§] mean follow-up only reported for 19 survivors

[||] mean follow-up only reported for 18 survivors who were not lost to follow-up

[#] mean follow-up only reported for all patients with intra- and extracranial dissection (N=110), and not for subgroup of patients with IAD (N=27)