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Childhood Basilar Artery Occlusion: A Report of 5 Cases and Review of the Literature

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Abstract

Basilar artery occlusion has poor outcome in adults; little is known regarding outcomes in children. Whether intra-arterial treatments improve adult outcomes is controversial. Safety and efficacy of intra-arterial treatments in children are unknown. We report 5 cases of basilar artery occlusion and review published cases. We estimated National Institute of Health Stroke Scale (NIHSS) and modified Rankin Score (mRS) of published cases, compared scores between non-intra-arterial treatments and intra-arterial treatments groups, and examined the correlation between NIHSS and mRS. Of our cases, 4 had good outcomes and 1 died. Of 63 published cases, 45 had no intra-arterial treatments and 18 had intra-arterial treatments. In the non-intra-arterial treatments group 24 had good outcomes. In the intra-arterial treatments group 13 had good outcomes. There was strong correlation between the NIHSS and the mRS. Children with basilar artery occlusion have better outcomes than adults. Certain children with basilar artery occlusion may be treated conservatively. A registry for childhood basilar artery occlusion is urgently needed.

Keywords

basilar artery occlusion; intra-arterial treatment; National Institute of Health Stroke Scale; modified Rankin Score; thrombolysis

Arterial ischemic strokes are uncommon in children and basilar artery occlusive strokes are particularly uncommon. In adults, basilar artery occlusion has a grim outcome. The rate of

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Author Contributions

MC reviewed the identified cases from the institution and compiled the clinical information, collected data from the cases reported in the literature, estimated the National Institute of Health Stroke Scale and the modified Rankin Scores, and compiled the initial draft. WDL designed the study and provided the overall supervision, contributed towards the review of the literature, performed the statistical analysis, and revised the manuscript.

Ethical Approval

The study was approved by the Nationwide Children's Hospital Institutional Review Board.

Declaration of Conflicting Interests

MC has nothing to disclose. WDL is an editorial board member of the *Journal of Child Neurology* and *Pediatric Neurology*. He receives institutional support from the Research Institute of Nationwide Children's Hospital and has received support from the NINDS, but no such funds were expended for this study.

death or severe disability in adults approaches 80%,^{1,2} but in children much less is known regarding the outcome. Advocates of intra-arterial treatments, such as intra-arterial thrombolysis, thrombectomy, and angioplasty, argue that intra-arterial treatment improves outcomes in adults.^{3,4} Recent case reports describing intra-arterial treatments in children with basilar artery occlusion have noted good outcomes. In these case reports the justification for intra-arterial treatments has been the poor outcome of basilar artery occlusion with conservative management. The lack of outcome data in childhood basilar artery occlusion makes it difficult to judge the efficacy of intra-arterial treatments, and there are challenges and risks to performing intra-arterial treatments in children with basilar artery occlusion.

Acute thrombolytic therapy is associated with a number of complications including intracerebral or systemic hemorrhage that can lead to death or severe morbidity. In the Basilar Artery International Cooperation Study,⁵ symptomatic intracranial hemorrhage was more common in patients treated with intra-arterial thrombolysis (14%) and intravenous thrombolysis (6%) than in patients treated with antithrombotic therapy (1%) such as antiplatelet/systemic anticoagulation. Other complications such as reocclusion and secondary embolization can occur with ineffective thrombolysis or embolism of the lysed clot. Not all complications are life-threatening, but they can lead to prolonged hospital stay, delayed rehabilitation, and increased morbidity.⁶

No studies directly compare the outcomes of children with basilar artery occlusion who have been treated with intra-arterial treatments and those who have not been treated with intra-arterial interventions in children. The rarity of basilar artery occlusion poses a considerable obstacle to performing a clinical trial. As a result, most physicians who treat childhood stroke depend on the knowledge developed from case reports and series. To address this need, we report 5 children who developed basilar artery occlusion at a single academic medical center and compare their findings with those of cases previously reported in the literature who were treated either conservatively or with intra-arterial treatments.

Method

We identified 5 children who had basilar artery occlusion at the Nationwide Children's Hospital between 2002 and 2012. Two cases were identified retrospectively before 2006 and 3 cases were identified from a prospective stroke registry. In all cases the diagnosis of basilar artery occlusion was confirmed by inspection of the radiological images. The clinical presentations and outcomes were obtained from the medical records and the last documented clinical examination of the subject. Based on the admission records we estimated an initial National Institute of Health Stroke Scale (NIHSS) for our 5 cases. A recent study demonstrated that a pediatric NIHSS can be estimated from the medical record.⁷ If there were multiple examinations close in time, we reported the most severe score. We used the last available outpatient clinic assessment on our own cases to estimate a modified Rankin Score (mRS). Previous studies have used chart review to estimate an mRS.^{8,9}

To identify published cases from the literature we performed a search of Medline for the years 1962 to 2012 that included the terms *basilar artery*, *basilar artery occlusion*,

thrombolytic therapy, angioplasty, child, embolectomy, thrombectomy, thrombolysis, arterial occlusive diseases, thrombosis, arterial occlusion, fibrinolytic agents, streptokinase, and children 0 to 18. We excluded patients who were older than 18 years of age and patients whose published reports provided insufficient clinical or radiological information to adequately document a basilar artery occlusion. As with our own cases we estimated an NIHSS and mRS from the clinical information provided in the case reports.

Since the total number of cases from our series and previous publications was small, we used descriptive statistics and histograms to compare the initial NIHSS and the mRS for cases managed conservatively versus cases that underwent intra-arterial treatments. We used Spearman's rho with a 2-tailed level of significance at $P < .05$ to examine the correlation between the NIHSS and the mRS. Statistical analysis was performed with SPSS (IBM-SPSS, Armonk, NY).

This study was approved by the Nationwide Children's Hospital Institutional Review Board.

Case Reports

Case 1

A 2-year-old boy presented with alerted mental status and vomiting for 2 days, and a gradually deteriorating mental status. He was admitted to a local hospital and treated with intravenous fluids for dehydration. No clinical improvement was noted, prompting a computed tomography (CT) brain scan, which showed acute infarcts in the cerebellum. On initial evaluation at our institution approximately 24 hours after the onset of symptoms he was awake, irritable, but easily consolable. He had dysarthria, but no facial asymmetry. He had minimal movements of the left extremities with diminished tone, and spontaneous movements on the right with decreased strength. A magnetic resonance image (MRI) of the brain showed acute infarcts within both cerebellar hemispheres (Figure 1) and the left cerebral peduncle, and an older infarct in the right thalamus. A magnetic resonance angiogram (MRA) showed basilar artery occlusion (Figure 2). Intra-arterial thrombolysis was not considered since the patient was well outside the window for intervention. He was treated with heparin for anticoagulation that was transitioned to enoxaparin. Cardiac echocardiogram and thrombophilia evaluation were negative.

CT angiography of the head and neck 2 weeks after the initial presentation showed improved flow through the basilar trunk with near complete resolution of the thrombus and reestablishment of flow. No definite dissection was noted, but multiple areas of subtle stenosis were noted in the posterior circulation vessels; the anterior circulation was normal. Fibro-muscular dysplasia was suspected, but the areas of stenosis were subtle and the renal angiogram was normal.

He received inpatient rehabilitation for 3 weeks after the initial 10 days on the inpatient neurology service, and was discharged home on enoxaparin. By 5 months following his initial presentation, he had no focal motor or coordination deficits except for speech, which was difficult to understand at times. A repeat CT angiogram at 6 months showed improved appearance of the basilar trunk with no change in the appearance of multiple areas of

stenosis noted in the posterior circulation previously. He was transitioned to aspirin. At 7 months follow-up, he was started on levetiracetam for atypical absence seizures.

Case 2

A 6-year-old boy was hospitalized for a 2-month course of increasing intermittent dizziness, headache, and associated vomiting. He was initially thought to have migraine. On the day of admission he became progressively unresponsive. A CT brain scan showed acute stroke in the right cerebellar hemisphere and vermis with effacement of the fourth ventricle and hydrocephalus. He underwent an emergency posterior fossa decompression. Twenty-four hours after admission, MRI brain scan, MRA head and neck, and conventional catheter angiography showed complete basilar artery occlusion, a large right cerebellar infarct, and a left vertebral artery dissection (Figures 3 and 4). Anticoagulation was not considered initially because of the large infarct. He was started on aspirin 24 hours after the surgery to reduce further thrombosis and embolization. Aspirin was eventually transitioned to enoxaparin after 2 weeks. Thrombophilia workup was negative. He developed posterior fossa syndrome with cerebellar mutism during the course of his stay, but rapidly recovered toward baseline. CT angiogram at 2 months showed a persistent basilar artery thrombus localized to the midportion with partial recanalization and persistent nonocclusive aneurysmal dissection of the V3 segment of the left vertebral artery. After 2 weeks of acute inpatient stay, he had 16 days of inpatient rehabilitation. He was discharged home on enoxaparin.

On neurology follow-up at 6 months after the initial presentation, he had no focal deficits except for a very subtle finding of slower and less rhythmic alternating movements on the right. He was maintained on enoxaparin with plans for follow-up imaging to monitor the dissection and eventual transition to aspirin.

Case 3

An 8-year-old previously healthy boy presented with 4 days of headache, double vision, and a left sixth cranial nerve palsy. A brain MRI scan and a MRA showed a small left pontine stroke (Figure 5), mild enhancement of the cisternal segment of the left sixth cranial nerve, and basilar artery occlusion (Figure 6). His neurological examination was notable only for a partial left sixth cranial nerve palsy, and the remainder of his examination was normal. A conventional catheter angiogram showed basilar artery occlusion with no evidence of dissection. He was started on aspirin. An evaluation that included cerebrospinal fluid examination identified an antinuclear antibody with speckled/nucleolar pattern that was positive at a 1:640 titer, an elevated anti-streptolysin O (ASO) of 999 (Ref: 0–195 IU/ml), anti-DNAse B of 898 (Ref: 0–250 IU/ml), low C4 of 8 mg/dL (Ref: 16–59 mg/dL), and a throat culture positive for group A Strep. A thrombophilia evaluation and a cardiac echocardiogram were normal. The positive antinuclear antibody (ANA) result suggested an underlying autoimmune disorder, but the catheter angiogram showed no features of vasculitis. The streptococcal infection was treated with antibiotics and he received a 3-day course of intravenous methylprednisolone and then transitioned to oral prednisolone. He was started on hydroxychloroquine and mycophenolate for the suspicion of lupus. Acute inpatient stay consisted of 5 days; he did not require inpatient rehabilitation. His

neurological exam 1 month after his initial presentation was notable only for the partial left sixth cranial nerve palsy.

Case 4

A 7-year-old boy presented with altered mental status after complaining of headache, dizziness, nausea, and vomiting for 2 days. He was initially thought to have meningoencephalitis at the local hospital. On initial evaluation at our institution approximately 12 hours after the onset of symptoms, he was aphasic with facial weakness, bilateral upper and lower extremity weakness, bilateral hyperactive reflexes with bilateral clonus at the ankles, and extensor toe signs bilaterally. A brain MRI scan showed a subacute infarct of the right medial temporal lobe and an acute infarct of the central anterior pons. An MRA and conventional angiogram showed a hypoplastic right vertebral artery with dissection and basilar artery occlusion. He was treated with aspirin. His neurological status improved in the next 24 hours. He was more alert and was able to follow commands, his facial movements were symmetric, and he had improved strength all 4 extremities. Thrombophilia evaluation as well as a transthoracic and transesophageal echocardiograms were normal. He received 3 weeks of inpatient rehabilitation after 10 days of initial neurology inpatient stay. At the time of discharge, 1 month after the initial presentation, he had only ataxia and dysarthria.

At 3 months after presentation, a repeat MRI and MRA showed gliosis in the right medial temporal lobe and central pons, persistent thrombosis of the right vertebral artery, and marked attenuation of flow through the basilar artery. At 3.5 years after his presentation, his mRS was 1 and a repeat MRI/MRA showed persistent occlusion of the right vertebral and the midbasilar artery with the posterior cerebral arteries supplied mainly by the posterior communicating arteries. He was continued on aspirin until 5 years after his stroke.

Case 5

A 14-year-old girl was struck in the head during a sporting event. There was no history of loss of consciousness. Her initial Glasgow Coma Scale was 12 and improved to 15 after approximately 30 minutes. She developed a generalized tonic-clonic seizure in the emergency room. A CT brain scan showed a depressed frontal skull fracture extending through the left orbital roof, a hemorrhagic contusion of the left frontal lobe, and a subarachnoid hemorrhage along the anterior falx. A CT neck scan showed no fracture. At 24 hours after the injury she had no neurological deficits. At 36 hours after presentation she had a cardiorespiratory arrest with respiratory failure and was intubated. The pupils were fixed and dilated, and brainstem reflexes were absent. A repeat CT brain showed diffuse brain edema, and basilar cistern subarachnoid hemorrhage. The patient was treated with intracranial pressure monitoring, pentobarbital, and hypothermia. A repeat CT brain showed increased subarachnoid hemorrhage, and severe brain edema. Conventional angiogram showed right vertebral artery dissection with basilar artery occlusion and absence of intracerebral bilateral carotid flow beyond the level of the middle cerebral arteries. She was declared brain dead based on the neurological exam and absence of cerebral blood flow.

Results

In our small series, there were delays in the diagnosis of a stroke so that the window for intra-arterial treatments had closed or in one case the patient lost all brainstem function in a catastrophic presentation before the diagnosis of basilar artery occlusion was recognized (Table 1). Four patients were treated with antiplatelet agents or heparin; these 4 had good outcomes with mRS scores of 1. Three of our cases had vertebral artery dissection as the identified cause of the basilar artery occlusion while 1 case had a suspected autoimmune vasculitis. One case had no identified cause (case 1), but dissection was suspected.

In our review of the literature from 2002 to 2012 we identified 63 cases of basilar artery occlusion in children. Of these, 45 were treated with anticoagulation, antiplatelet agents, intravenous thrombolysis, or no treatment at all. Twenty-four of these 45 had good outcomes with estimated mRS scores of 0–2, 15 had poor outcomes with mRS scores of 3–5, and 6 died. If we add our cases to the existing published cases, 28 of 50 have mRS scores of 0–2, 15 had mRS scores of 3–5, and 7 died (Figure 7). The median value of the mRS for the conservatively treated group was 2 with a range of 0–6. Since 2000, there have been 18 published cases that were treated with intra-arterial treatments; of these 13 had good outcomes with mRS scores of 0–2, 4 had mRS scores of 3–5, none died, and outcome couldn't be assessed in 1. The median value of the mRS for the intra-arterial treatment group was 1 with a range of 0–5. The mRS scores would suggest that intra-arterial treatment results in better outcomes since no cases with intra-arterial treatments died and the proportion of good outcomes is higher than in the more conservatively treated cases.

If one includes the estimated NIHSS as an initial measure of severity, the comparison becomes more complicated. The median estimated NIHSS score was 21.5 (range 1–36) in the conservatively treated group and a median of 31 (range of 6–36) in the intra-arterial treatment group. The histogram of the estimated NIHSS, however, shows that there are far more cases with NIHSS 30 or greater in the conservatively treated group than the intra-arterial treatment group. (Figure 8). When one pools all the cases, intra-arterial treatment group and the non-intra-arterial treatments group, initial NIHSS was highly correlated with the mRS with a correlation coefficient of 0.534, $P < .001$,¹⁰ so one might anticipate that the conservatively treated cases with a high NIHSS might have poorer outcomes. A high NIHSS score, however, does not invariably predict poor outcome, and some cases may do well even with conservative treatment (Figure 9).

Discussion

This study illustrates that children with basilar artery occlusion may have proportionately better outcomes than adults regardless of intra-arterial treatments. Recent case reports of intra-arterial treatments in childhood basilar artery occlusion have noted that basilar artery occlusion has poor outcomes,^{11–13} but the limited available evidence suggests that the outcomes of basilar artery occlusion are more nuanced. The prognosis of basilar artery occlusion in adults is poor, with approximately 70% having poor outcomes in multiple series and 1 large registry.^{1,14–15} In comparison, if one pools our cases with the published cases, 41 of 68 reported cases have a good outcome, which is a higher proportion of good

outcomes. The difference in the mRS scores would appear to suggest that intra-arterial treatments results in better outcomes since no cases with intra-arterial treatments died and the proportion of good outcomes is higher than in the more conservatively treated cases. This observation should be treated with some caution since the intra-arterial treatment group had fewer cases with an NIHSS > 30 when compared with the conservatively treated group.

Our findings suggest that treatment in children who present with basilar artery occlusion may be selected, that is, that intra-arterial treatments need not be performed in all cases. Children with a low initial NIHSS might be conservatively managed with anticoagulation and observed to determine if there is a clinical deterioration. Consistent with our results, a recent case series¹⁶ found that children with basilar artery occlusion had a mortality rate of 8% and severe neurological deficits occurred in less than one third of cases receiving conservative treatment. In the adult literature, the evidence is equivocal that intra-arterial treatments is the most effective option. A review of previously published series found no superiority of intra-arterial treatments versus intravenous thrombolysis.¹⁷ In a large European registry, intra-arterial treatments was not superior to intravenous thrombolysis in improving outcome. A recent study in adults indicated that endovascular therapy is not superior to standard treatment with intravenous thrombolysis in patients with acute ischemic stroke.¹⁸ At present, intravenous thrombolysis in a child should be considered experimental, and a clinical trial is under way to determine safety and efficacy of tissue plasminogen activator in children (TIPS).¹⁹ There is uncertainty whether intra-arterial treatments should be pursued if basilar artery occlusion is diagnosed well after the clinical presentation. Stroke is an uncommon disease in children that frequently is associated with delayed diagnosis, as illustrated by our cases. Several of the case reports of intra-arterial treatments in childhood basilar artery occlusion described treatment occurring many hours after the clinical presentation. The available evidence in childhood basilar artery occlusion does not answer this question, but some direction might be gleaned from the adult experience. In the large registry noted previously, the risk of poorer outcome increased when recanalization (both intravenous thrombolysis and intra-arterial treatments) were initiated more than 6 hours after the onset of the basilar artery occlusion.²⁰

There are limitations to our report. It is difficult to generalize the findings of our own series because of the small number of cases. In the examination of previous cases we estimated NIHSS and mRS scores from the published reports. While it is possible to estimate NIHSS and mRS from chart reviews, as noted previously, our estimates depend on the accuracy of the case descriptions. The mRS is one of the most widely used measures of stroke outcome in adults, but there are uncertainties regarding the interobserver reliability of the mRS²¹ that can add to variability in this outcome measure. We show a high level of correlation between the NIHSS and mRS; however, the scatterplot of these values shows that the predictive value of a high NIHSS score has limitations. Lastly, we cannot eliminate the possibility of publication bias that may have skewed reports toward cases that had better outcomes, particularly with the intra-arterial treatments reports. A similar publication bias was noted with reports of intravenous thrombolysis for childhood ischemic stroke.²²

Despite these limitations, our series taken together with previously published cases suggest that basilar artery occlusion in children is a very different disorder than what occurs in

adults. The collected cases suggest that there are specific children with radiologically confirmed basilar artery occlusion who may be managed with non-intra-arterial treatment therapies coupled with careful clinical observation. Our report raises a number of questions regarding the best management of childhood basilar artery occlusion, for example, what is the range of treatment options; if intra-arterial treatments are contemplated, whether there is a window of opportunity as in adults. A randomized clinical trial might answer these questions, but given the low incidence of basilar artery occlusion in children, such a trial is unlikely to be accomplished. As has been suggested by Fink et al,¹³ the next best alternative may be a registry for children with basilar artery occlusion to provide us with a body of information to begin to address these questions.

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Figure 1. Magnetic resonance imaging (MRI) with diffusion sequence for case 1. White and black arrow pointing to the restricted diffusion in the right and left cerebellar hemispheres, respectively, indicating acute infarct.

Figure 2.

Magnetic resonance angiogram (MRA) for case 1. Large white arrows pointing to vertebral arteries, small white arrows showing no flow in distal basilar artery indicating distal basilar artery occlusion, black arrow heads indicating middle cerebral arteries, and white arrow heads pointing to anterior cerebral arteries.

Figure 3. Magnetic resonance imaging (MRI) with diffusion sequence for case 2. White arrow pointing to the right cerebellar hemisphere with restricted diffusion and black arrow pointing to the cerebellar vermis with restricted diffusion indicating acute infarct.

Figure 4. Conventional angiogram for case 2. Large black arrow pointing to aneurysmal dissection of the V3 segment of the left vertebral artery, small black arrow pointing to left posterior inferior cerebellar artery, and white arrow showing no flow into the basilar artery indicating complete basilar artery occlusion.

Figure 5. Magnetic resonance imaging (MRI) with diffusion sequence for case 3. White arrow pointing to restricted diffusion in left anterior pons indicating acute infarct.

Figure 6. Conventional angiogram after contrast injection into left vertebral artery (VA) for case 3. Large black arrow—left VA; small black arrow—large left posterior inferior cerebellar artery (PICA); white large arrow—right VA after reflux of contrast from the left VA; small white arrow—right PICA after reflux of contrast from the left VA; and black arrow head—showing no flow into the proximal basilar artery, indicating basilar artery occlusion.

Figure 7. Histogram depicting modified Rankin Scores sorted by group. Group 1, no intra-arterial interventions; group 2, intra-arterial interventions. Abbreviations: IAT, intra-arterial treatment.

Figure 8.

Histogram depicting National Institute of Health Stroke Scale scores sorted by group. Group 1, no intra-arterial interventions; group 2, intra-arterial interventions.

Figure 9. Scatterplot of National Institute of Health (NIH) Stroke Scale versus modified Rankin Score (MRS). The bolder circles indicate multiple cases per data point.

Table 1

Five Cases From Nationwide Children's Hospital: Clinical Features, Imaging, NIHSS and mRS Scores, Etiology, and Treatment.

Age in Year	Sex	Clinical Features	CT	MRI	MRA/Conventional Angiogram/CT Angiogram	NIHSS	mRS	Etiology/Treatment
2	M	Vomiting and altered mental status. On exam, dysarthria and weakness, left > right.	Bilateral cerebellar infarcts	Acute infarcts bilateral cerebellar hemispheres and left cerebral peduncle	MRA—basilar artery thrombosis with occlusion. CT angiogram—BAO, multifocal areas of stenosis in posterior circulation. No dissection.	13	1	Unknown Initial heparin, transitioned to enoxaparin and then to aspirin.
6	M	2 months of progressive headache, dizziness, and vomiting. On exam, comatose.	Infarct right cerebellum and vermis, effacement of fourth ventricle and hydrocephalus.	Acute infarct of right cerebellum and the vermis.	MRA and conventional angiogram—complete basilar artery occlusion with left vertebral artery dissection.	35	1	Vertebral artery dissection Initial aspirin then transitioned to enoxaparin.
8	M	Headache, double vision and left sixth cranial nerve palsy On exam, left sixth cranial nerve palsy.		Small left pontine infarct, mild enhancement of the cisternal segment of left sixth cranial nerve.	MRA and conventional angiogram—basilar artery occlusion. No dissection.	1	1	Possible immune mediated; aspirin
7	M	2 days of headache, dizziness, vomiting followed by altered mental status. On exam, aphasia, facial weakness, extremity weakness, right > left.		Acute central pontine infarct and subacute right medial temporal lobe infarct.	MRA and conventional angiogram—basilar artery occlusion, hypoplastic right vertebral artery with dissection.	16	1	Vertebral artery dissection. Aspirin
14	F	Trauma during sporting event. On exam, Initial GCS 13 → 15; at 36 hrs comatose.	Depressed skull fracture with a small subarachnoid hemorrhage; repeat CT with worsening subarachnoid hemorrhage, diffuse brain swelling		Conventional angiogram—right vertebral artery dissection with basilar artery occlusion.	36	Death	Vertebral artery dissection.

Abbreviations: BAO, basilar artery occlusion; CT, computed tomography; GCS, Glasgow Coma Scale; MRA, magnetic resonance angiogram; MRI, magnetic resonance imaging; mRS, modified Rankin Score; NIHSS, National Institute of Health Stroke Scale.

Table 2

No Intra-arterial Intervention Group.

Study	Age in Years	Sex	Conventional Angiogram/MRA/TCD/Pathology	Intervention	Given or Estimated NIHSS	mRS
1 Fowler ²⁴	18 months	F	Pathology: postmortem BAO—distal portion	No	31	Death
2 Dooley and Smith ²⁵	6	M	Conventional angiogram BAO—distal portion	No	9	1
3 Ouvrier and Hopkins ²⁶	9	M	Conventional angiogram BAO—distal portion	Phenindione	10	2
4 Ouvrier and Hopkins ²⁶	14	M	Conventional angiogram BAO	No	19	3
5 Moscow and Newton ²⁷	10	M	Conventional angiogram BAO	No	*	4
6 Latchaw et al ²⁸	7	M	Conventional angiogram BAO	No	24	5
7 Latchaw et al ²⁸	11	M	Conventional angiogram BAO, left VA occlusion	No	12	2
8 Marsden ²⁹	3	F	Pathology: Postmortem BAO. Giant cell arteritis	No	34	Death
9 Sakata et al ³⁰	10	M	Pathology: Postmortem BA and left VA thrombosis.	No	36	Death
10 Singer et al ³¹	6	M	Conventional angiogram BAO	No	10	2
11 Fraser and Zimble ³²	6	M	Conventional angiogram BAO	No	16	4
12 Golden et al ³³	13	M	Conventional angiogram BAO	No	36	5
13 Ackerman et al ³⁴	10	M	Conventional angiogram BAO—complete	No	36	4
14 Zimmerman et al ³⁵	7	M	Conventional angiogram BAO—distal portion	Aspirin	6	0
15 Thompson et al ³⁶	15	M	Conventional angiogram BAO—midportion	No	*	2
16 Mori et al ³⁷	13	M	Conventional angiogram BAO—distal portion	Aspirin	11	2
17 Matsuishi et al ³⁸	4	M	Conventional angiogram BAO—proximal portion	No	6	1
18 Jain et al ³⁹	16	F	Conventional angiogram BAO—proximal portion	No	34	4

Study	Age in Years	Sex	Conventional Angiogram/MRA/TCD/Pathology	Intervention	Given or Estimated NIHSS	mRS
19 Echenne et al ⁴⁰	9	M	Conventional angiogram BAO—distal portion	Aspirin and dipyridamole	10	2
20 Lewis and Berman ⁴¹	16	M	Conventional angiogram BAO, right VA dissection	Aspirin	11	1
21 Biller et al ⁴²	18	F	Pathology: BA and bilateral VA occlusion	No	34	Death
22 Ross et al ⁴³	23 months	M	Conventional angiogram BAO—distal portion, left VA dissection	Heparin	8	1
23 Mares et al ⁴⁴	7	M	Conventional angiogram BAO	Heparin	34	Death
24 Chatkupt et al ⁴⁵	8	M	Conventional angiogram BAO	No	34	5
25 Awada et al ⁴⁶	15	M	Conventional angiogram BAO	No	34	5
26 Kwon et al ⁴⁷	16	F	Conventional angiogram BAO—multifocal	Oral anticoagulant	8	1
27 Schols et al ⁴⁸	10	F	Conventional angiogram BA dissection with thrombosis	Heparin Oral anticoagulant	34	1
28 Nakatomi et al ⁴⁹	7	M	Conventional angiogram BAO	No	34	1
29 Way et al ⁵⁰	16	F	Conventional angiogram BAO—embolic	Heparin Warfarin	30	1
30 Verdu et al ⁵¹	9	M	Conventional angiogram BAO	Aspirin Enoxaparin Acenocoumarol	34	1
31 Hasan et al ⁵²	7	M	Conventional angiogram BAO—distal portion, right VA dissection	Heparin Coumadin	14	0
32 Rosman et al ²³	6	M	Conventional angiogram BAO	No	34	3
33 Rosman et al ²³	4	M	MRA—BAO	Heparin Warfarin	34	3
34 Juneja et al ⁵³	10	F	MRA—BAO and left VA occlusion	Enoxaparin Warfarin	34	3
35 Hudaoglu et al ⁵⁴	10	M	Conventional angiogram BAO—distal portion	Heparin Aspirin	14	3
36 Gordon and Bertrayer ⁵⁵	9	M	Conventional angiogram BAO—distal portion, embolic	Aspirin	12	2
37 Palfi et al ⁵⁶	17	M	BAO on TCD	No	34	2
38 Cheng et al ⁵⁷	8	F	Conventional angiogram BAO, left VA dissection	Heparin Warfarin Aspirin	34	4

Study	Age in Years	Sex	Conventional Angiogram/MRA/TCD/Pathology	Intervention	Given or Estimated NIHSS	mRS
39 Golubovi et al ⁵⁸	14	F	Conventional angiogram BAO, left VA occlusion	Heparin	34	3
40 Loevner et al ⁵⁹	7	M	Conventional angiogram Giant aneurysm of distal BA with clot within.	No	6	1
41 Navero et al ⁶⁰	4	F	Conventional angiogram BAO and VA dissection	No	34	Death
42 Ishikawa et al ⁶¹	11	M	Conventional angiogram BAO	Ticlopidine	4	0
43 Camacho et al ⁶²	3	F	Conventional angiogram BAO	Aspirin	4	0
44 Marietta et al ⁶³	6	M	Conventional angiogram BAO, left VA dissection	Enoxaparin	2	0
45 Heil et al ⁶⁴	16	F	MRA—BAO	IVT—outside the therapeutic window	8	0

Abbreviations: BAO, basilar artery occlusion; IVT, intravenous thrombolysis; MRA, magnetic resonance angiogram; mRS, modified Rankin Score; NIHSS, National Institute of Health Stroke Scale; TCD, transcranial doppler; VA, vertebral artery;

* , unknown.

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Table 3

Intra-arterial Intervention Group.

Study	Age in Years	Sex	Conventional Angiogram/MRA/ TCD/Pathology	Intervention	Given or Estimated NIHSS	mRS
1 Sungarian and Duncan ⁶⁵	9	M	Conventional angiogram BAO ⁵ —midportion, right VA dissection	Intra-arterial thrombolysis within 6 hrs	18	0
2 Sungarian and Duncan ⁶⁵	10	F	Conventional angiogram BAO—midportion, bilateral VA pseudoaneurysm	Intra-arterial thrombolysis within 5 hrs	22	1
3 Ming-Hsiang Chen et al ⁶⁶	15	M	CT angiogram—BAO	Intra-arterial thrombolysis within 6 hrs	25	0
4 Yoshikawa ⁶⁷	8	M	Conventional angiogram—BAO	Intra-arterial thrombolysis	19	0
5 Zaidat et al ⁶⁸	16	M	Conventional angiogram BAO—midportion	Thrombectomy and angioplasty	35	2
6 Komiya et al ⁶⁹	13	M	Conventional angiogram Proximal BAO with dissection	Stenting at 4.5 to 5.5 hrs	34	5
7 Grigoriadis et al ⁷⁰	6	M	Conventional angiogram BAO, left VA dissection	Intra-arterial thrombolysis, thrombectomy, and angioplasty at 44 hrs	34	0
8 Bhatt et al ¹¹	3	F	MRA—BAO	Intra-arterial thrombolysis at 18 hrs	35	3
9 Arnold et al ⁷¹	9	M	MRA—BAO, mid and distal portions	Intra-arterial thrombolysis at 12 hrs	22	2
10 Taneja et al ⁷²	14	F	Conventional angiogram BAO	Thrombectomy at 24 hrs	35	0
11 Janmaat et al ⁷³	4	M	Conventional angiogram BAO	Intra-arterial thrombolysis at 60 hrs	30 Initial 17	1
12 Condi et al ¹²	5	M	Conventional angiogram BAO, left VA dissection	Intra-arterial thrombolysis at 36 hrs, thrombectomy and coiling on day 7	32	1
13 Kirton et al ⁷⁴	15	M	Conventional angiogram BAO	Intra-arterial thrombolysis, thrombectomy, and angioplasty	34	1
14 Larner et al ⁷⁵	18	F	Conventional angiogram BAO distal portion	Intra-arterial thrombolysis, attempted with streptokinase, no recanalization	25	3
15 Cognard et al ⁷⁶	8	M	Conventional angiogram BAO	Intra-arterial thrombolysis and angioplasty	21	0
16 Rosman et al ²³	18	F	Conventional angiogram BAO distal portion	Intra-arterial thrombolysis	36	4
17 Fink et al ¹³	11	M	Conventional angiogram BAO distal portion	IVT and thrombectomy at 4 hrs	6	1
18 Grunwald et al ⁷⁷	16	F	MRI—BAO	Intra-arterial thrombolysis and thrombectomy	36	*

Abbreviations: BAO, basilar artery occlusion; CT, computed tomography; IVT, intravenous thrombolysis; MRA, magnetic resonance angiogram; MRI, magnetic resonance imaging; mRS, modified Rankin Score; NIHSS, National Institute of Health Stroke Scale; TCD, transcranial doppler; VA, vertebral artery.

* , unknown.

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