

## Basilar artery thrombosis in a term neonate with posterior circulation ischemic stroke: Case and controversies

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### Introduction

Perinatal stroke is a focal vascular brain injury that occurs from the fetal period to 28 days of postnatal life<sup>1,2</sup>. The most focused lifetime risk for stroke occurs in the perinatal period<sup>3</sup> with an overall incidence of up to 1 in 1,000 live births<sup>4</sup>. Neonatal arterial ischemic stroke (NAIS), an ischemic stroke involving arterial territories that presents in the term neonate, is the most common type of perinatal stroke and most often affects the anterior circulation<sup>1</sup>. Posterior circulation strokes are uncommon in neonates and presumed to occur in less than 20% of all NAIS cases<sup>4,5</sup> (Table 1). Although thromboembolic occlusion is a presumed mechanism, thrombus detection at diagnosis is uncommon and evidence of residual thrombus in the basilar artery extremely rare.

We describe the case of a term neonate who presented with acute symptomatic seizures and was found to have basilar artery thrombosis with multifocal ischemic strokes in the posterior circulation, leading to complex management considerations.

### Case description:

Baby M was born to a healthy 25-year-old mother with no complications or exposures during pregnancy. He was delivered at 38+5 weeks gestation via vacuum-assisted vaginal delivery after prolonged fetal heart rate decelerations. Birth weight was 2520 g. APGARS were 4,7,8 at 1 minute, 5 minutes, and 10 minutes respectively. Initial resuscitation measures included positive pressure ventilation (PPV) and continuous positive airway pressure (CPAP), with transition to room air by four minutes of life. At 3 hours of life, he had recurrent apneas associated with eye fluttering, right arm posturing, and right leg cycling which responded clinically to phenobarbital 20 mg/kg. On initial examination he was encephalopathic requiring ventilatory support and significantly hypotonic with minimal spontaneous

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Srivastava - Basilar Artery Thrombosis

movement. Reflexes were symmetric. He had a large cephalohematoma and was thrombocytopenic with platelets of  $49 \times 10^9/L$ . Blood glucose was 1.1 mmol/L, requiring ongoing infusions of 20% dextrose for glycemic control. Dopamine was required for persistent hypotension. Workup for adrenal insufficiency as a cause for hypoglycemia identified left adrenal hemorrhage. EEG showed subclinical seizures in the form of epileptiform discharges isolated to the left posterior head region with no clear clinical correlate. Multiple medications were required to control seizures and included phenobarbital (20 mg/kg), levetiracetam (60 mg/kg), and fosphenytoin (20 mg/kg).

### Neuroimaging and management considerations:

Magnetic resonance imaging (MRI) revealed an occlusion at the tip of the basilar artery and multiple discrete areas of acute focal infarction in the left posterior cerebral artery (PCA) territory, bilateral thalami, midbrain, bilateral cerebellum, and right occipital region (Figures 1 and 2). There was no evidence of vertebral dissection. Echocardiogram showed functionally bicuspid aortic valve with no abnormal shunting and no thrombus.

After discussion with hematology and interventional radiology, endovascular thrombectomy was not attempted due to multiple concerns including unknown age of stroke, risk of hemorrhage, and absence of safety evidence. Anticoagulation was deferred in context of thrombocytopenia and the cephalohematoma. Repeat neuroimaging 3 days and 8 days later showed no further ischemic injury and no thrombus propagation.

### Discussion:

This complex case identified basilar artery thrombosis in a neonate with arterial ischemic stroke, which is an extremely rare occurrence in perinatal stroke literature<sup>6,7</sup> and clinical practice. The baby presented with early refractory seizures that resolved within days, which is an expected pattern of acute symptomatic seizures in the context of NAIS. His encephalopathy was initially attributed to multiple anti-seizure medications with likely contributions from his bi-thalamic injury. Hypoglycemia has been associated with posterior circulation NAIS and often demonstrates a similar pattern of occipital injury<sup>4</sup> though its role as an independent risk factor is uncertain<sup>8,9</sup>.

The presence of cerebellar infarcts suggested that the thrombus began more proximally and propagated to the basilar tip. In the absence of cardiac disease, the placenta was felt to be the most likely distal thromboembolic source.

### Treatment controversies

# pediatric stroke

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## Case Report

- Anticoagulation in neonates with thrombosis has been shown to be safe and well-tolerated<sup>10,11</sup>, but was deferred in this case for thrombocytopenia and significant hemorrhage risk. In absence of anticoagulation, repeat neuroimaging is essential to rule out clot propagation<sup>12</sup>. Had repeat neuroimaging shows clot propagation in this case, our recommendation would have been to start anticoagulation as guided by our hematology colleagues.
- Mechanical thrombectomy in NAIS is reported in isolated cases<sup>7,13</sup>. A similar case of distal basilar artery occlusion was treated with thrombectomy at 14 hours of life<sup>7</sup>, after which the basilar artery recanalized but needed nimodipine for vasospasm, and the left vertebral artery occluded due to repeat catheterization requiring treated with anticoagulation. To avoid such iatrogenic complications and in the absence of evidence showing benefit, we did not pursue thrombectomy and opted for conservative management instead.
- Placental thromboembolism may be present for days or weeks prior to delivery, with resulting large vessel occlusion occurring anytime within that period. Thus, interventional therapies in NAIS are limited by the inability to determine stroke onset. Neonates additionally have small arteries that may be more susceptible to secondary dissection or hemorrhage.
- Hypoglycemia in this case may have contributed to occipital lobe injury and may also have been an indication of critical illness. Although this baby's basilar artery occlusion is the primary etiology for his strokes, glycemic control is a key aspect of both primary and secondary ischemic injury<sup>8</sup>.

## Case conclusion

By 6 weeks of age, Baby M weaned off all anti-epileptic medication with no recurrence of seizures. He remained hypotonic but active, requiring NG feeds for poor suck.

Posterior artery strokes overall have good long-term neurodevelopmental outcome but higher risks of visual field deficits<sup>5</sup>. Baby M's bilateral thalamic ischemic injuries further increase his risk of cognitive dysfunction and epileptic encephalopathy, such as continuous spike and wave in sleep (CSWS)<sup>14</sup>. Risks of neonatal thrombectomy are substantial and undefined with the theoretical benefit undetermined, requiring critical analysis of this acute intervention in such a vulnerable population.

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Srivastava - Basilar Artery Thrombosis

*Pediatr Stroke. 2023;5: 29-38*

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## FIGURES & TABLES

**Table 1:** Prevalence of posterior circulation strokes in select original research studies of neonatal arterial ischemic stroke (NAIS). N, number of cases in study.

Author, year	N posterior circulation strokes	N all NAIS	% Posterior circulation strokes
Govaert et al 2009 <sup>6</sup>	4	54	7%
Kirton et al 2011 <sup>15</sup>	22	248	9%
Dunbar et al 2020 <sup>4</sup>	16	116	14%
Van der Aa et al 2013 <sup>5</sup>	13	85	15%

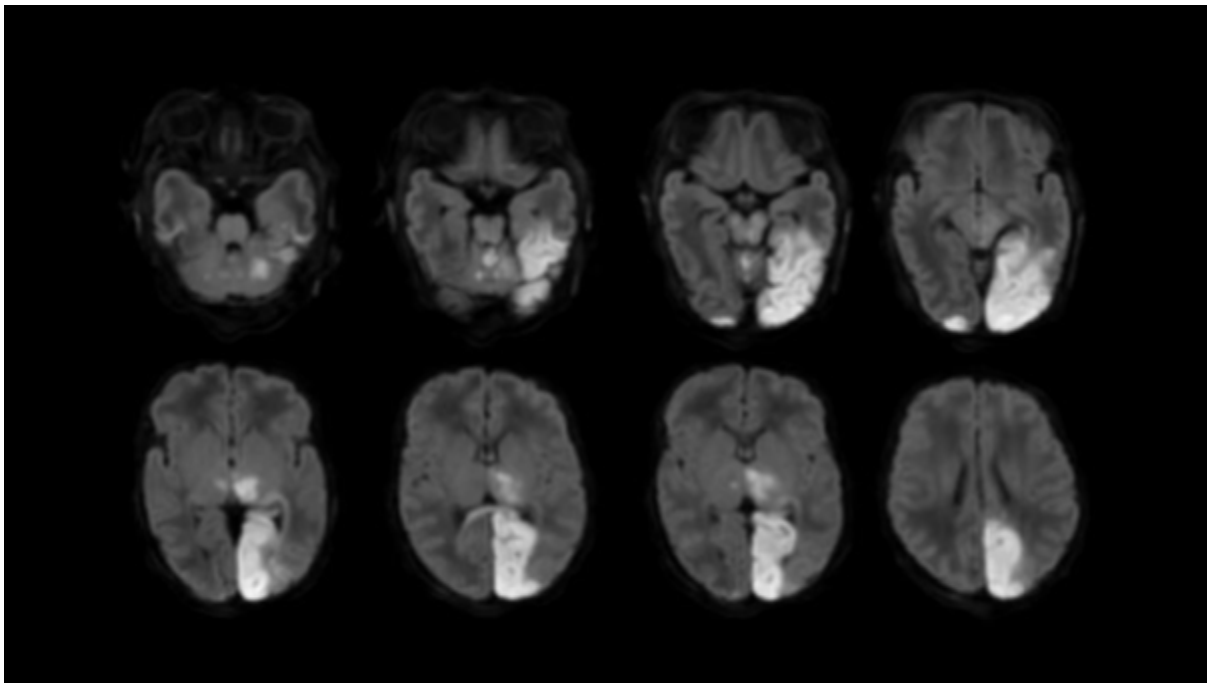
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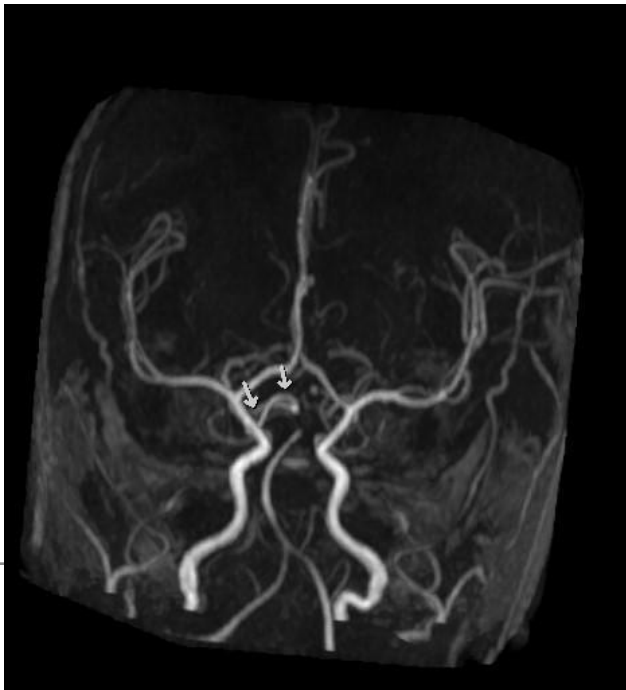
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**Figure 1: Representative MRI sequences showing acute ischemia.** Axial DWI showing areas of restricted diffusion in the left occipital lobe (left PCA territory). Multifocal patchy areas of restricted diffusion in the bilateral thalami, left corticospinal tract, midbrain, superior cerebellar vermis, and bilateral cerebellar hemispheres pertaining to the areas of posterior circulation. Focal area of restricted diffusion noted in the right occipital region.



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**Figure 2: Angiographic evidence of basilar artery occlusion.** Time-of-flight MRA shows occlusion at distal basilar artery proximal to the bifurcation (white arrows). The left P1 PCA and superior cerebellar artery are not visualized. P2 segment of left PCA reconstitutes through the anterior circulation but appears attenuated. There is reconstitution of the right PCA through anterior circulation but is also attenuated.