

# Lateral Medullary Syndrome Due to Left Vertebral Artery Occlusion in a Boy Postflexion Neck Injury

Abdulla Alawadhi, MD<sup>1</sup> , Christine Saint-Martin, MDCM, MSc<sup>2,3</sup>,  
 Christine Sabapathy, MD, MSc, FRCP<sup>4</sup>, Guillaume Sebire, MD, PhD<sup>1</sup>, and  
 Michael Shevell, MDCM, FRCP<sup>1</sup>

## Abstract

Lateral medullary syndrome is rare in pediatrics. It is characterized by neurological deficits due to an ischemic lesion in the lateral medulla. The authors describe a 17-year-old boy who developed lateral medullary syndrome in the context of a hyperflexion neck injury while diving in shallow water with traumatic vascular injury. He had “crossed” neurological deficits above and below the neck. His magnetic resonance angiography showed intra- and extracranial left vertebral artery occlusion and his magnetic resonance imaging showed signal abnormality involving the left lateral medulla and inferomedial cerebellum in keeping with an infarct secondary to left vertebral artery and left posterior inferior cerebellar artery occlusion. Good neurological recovery was observed on heparin therapy started after surgical treatment of traumatic injury. To our knowledge, this is the first reported case of lateral medullary syndrome in a pediatric population related to a flexion neck injury. The authors emphasize the importance of a high level of suspicion for accurate diagnosis.

## Keywords

Wallenberg syndrome, posterior inferior cerebellar artery, dissection, trauma, stroke, pediatrics

Received October 21, 2018. Received revised June 03, 2019. Accepted for publication July 14, 2019.

Pediatric ischemic strokes have a frequency of 1.8 to 3.3 per 100 000 children per year with less than 8% involving the posterior circulation.<sup>1</sup> Posterior circulation strokes can be a complication of an injury to the neck.<sup>2</sup> Lateral medullary syndrome, Wallenberg syndrome, or posterior inferior cerebellar artery syndrome are not uncommon in adults but are rare in children. It is a known stroke syndrome characterized by neurological deficits due to an ischemic lesion in the lateral medulla.<sup>3,4</sup> The anatomical parts involved are the spinothalamic tracts, sympathetic fibers, inferior cerebellar peduncle, nucleus ambiguus, nucleus and tract of cranial nerve V, and the inferior vestibular nucleus. Involvement of these structures result in loss of pain in the contralateral body, contralateral hemiplegia, ipsilateral Horner syndrome, ataxia, nausea, vomiting, decreased sensation over the ipsilateral face, nystagmus, diplopia, dysphagia, dysphonia, and vertigo. The vascular territory is supplied by the posterior inferior cerebellar artery, a branch of the vertebral artery.<sup>2,5</sup> Lateral medullary syndrome is often incomplete. Vertebral artery injuries have been described in penetrating injuries to the neck, chiropractic

manipulation, yoga, and sustained physiologic movements. The incidence of traumatically induced vertebral artery occlusion associated with nonpenetrating cervical spine fractures and/or dislocations was 17.2% in an adult study.<sup>6</sup> The most common

<sup>1</sup> Division of Pediatric Neurology, Departments of Pediatrics & Neurology/Neurosurgery, The Montreal Children's Hospital, McGill University, Montreal, Québec, Canada

<sup>2</sup> Department of Medical Imaging, The Montreal Children's Hospital, McGill University, Montreal, Québec, Canada

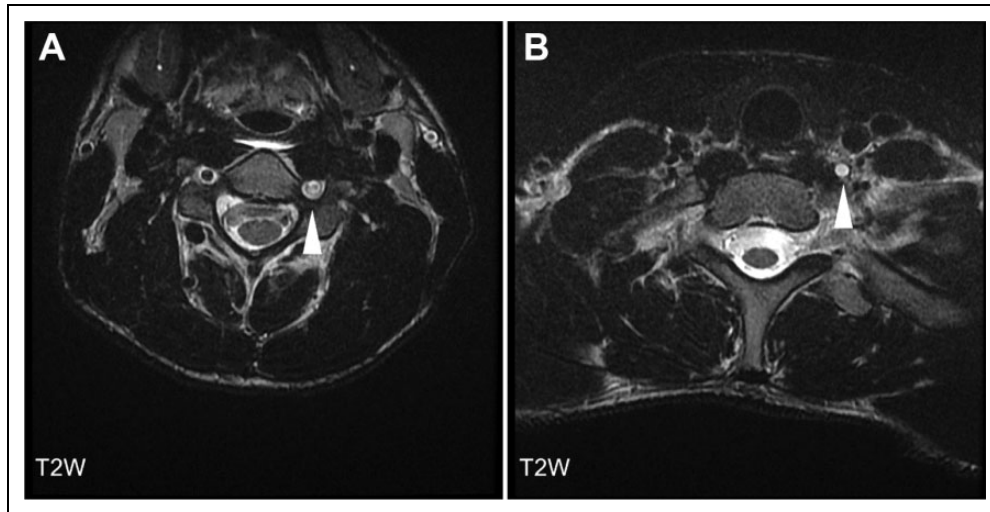
<sup>3</sup> Department of Radiology, The Montreal Children's Hospital, McGill University, Montreal, Québec, Canada

<sup>4</sup> Division of Pediatric Hematology/Oncology, Department of Pediatrics, The Montreal Children's Hospital, McGill University, Montréal, Québec, Canada

## Corresponding Author:

Abdulla Alawadhi, Division of Pediatric Neurology, Departments of Pediatrics & Neurology/Neurosurgery, The Montreal Children's Hospital, McGill University, Montreal, Quebec, Canada.  
 Email: [dr\\_abdulla@live.com](mailto:dr_abdulla@live.com)





**Figure 1.** Before spine surgery (day 0) showing loss of flow void with elevated T2 signal within the left vertebral artery from the level of C2-C3 (A) to T1 (B) within the left vertebral artery (arrowhead).

site of traumatic injury being the second part of the vertebral artery (C6-C7), where it passes through the foramen transversarium in adults, and V3 and upper V2 (around C2) in children.<sup>7,8</sup> Recent reports in adult studies have mentioned that flexion injuries are the most common mechanism of injury with incidence between 28% and 75%.<sup>6</sup>

The authors report a 17-year-old boy who developed lateral medullary syndrome in the context of a hyperflexion injury to the neck while diving in shallow water with traumatic vascular injury. To our knowledge, this is the first such report of lateral medullary syndrome in a pediatric population related to a flexion neck injury. The authors emphasize the importance of a high level of suspicion in cases of spine and neck injuries for accurate diagnosis.

## Methods

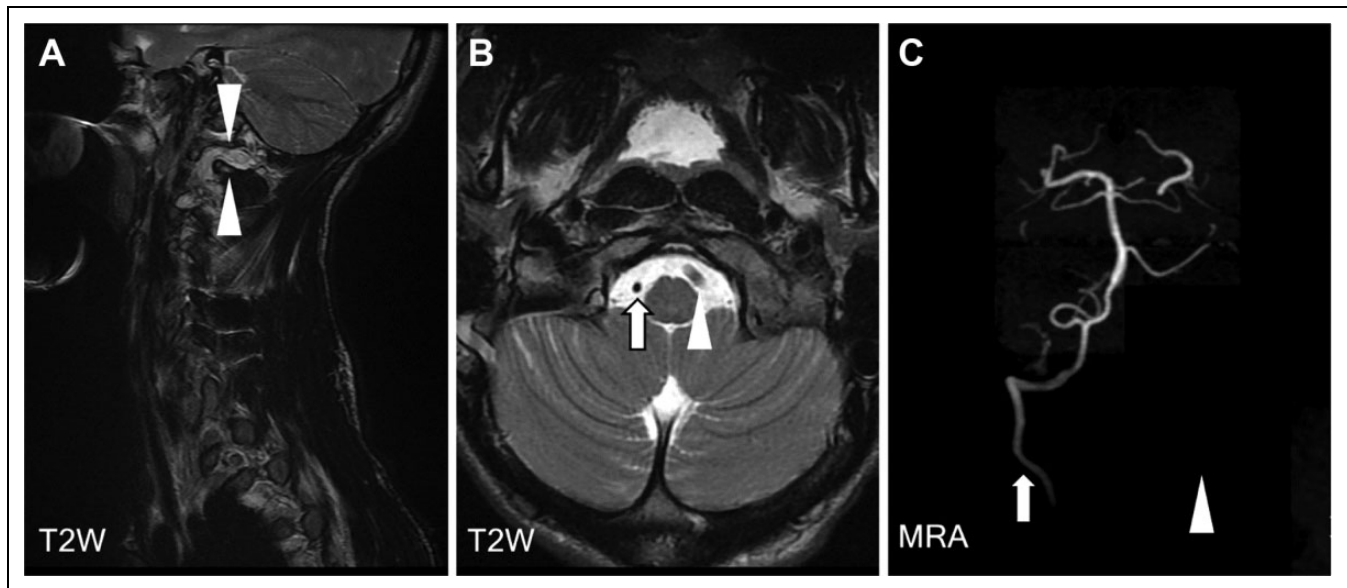
Case report and systematic review of the literature of all cases of lateral medullary syndrome in the pediatric population. Search was completed in PubMed with the key words “lateral medullary syndrome,” “Wallenberg syndrome,” “pediatrics,” and “children.” No language or year restriction applied. Age below 18 years old. Data extraction was done by the first author (A.A.). It included year of publication, age at presentation, gender, the initial presentation, imaging, treatment, and outcomes.

## Case Report

The patient is a 17-year-old boy who was previously healthy with a negative family history of stroke or thrombophilia. He presented to the hospital as a traumatic case of flexion neck injury after diving into shallow water. At an outside hospital, he was diagnosed with a C5 fracture. Immobilization was undertaken, and he was transferred to our center. On arrival, he was found to have cervical spine tenderness with overt weakness of the right upper extremity. There were no obvious signs of trauma to the head. His Glasgow Coma Scale was 15/15. His

pupils were equal and reactive to light and there was no obvious facial asymmetry. He was hemodynamically stable. Neck and spine magnetic resonance imaging (MRI) at day 0 (D0), a few hours after presentation, showed a burst fracture at C5 with associated retropulsion and perched facets, and a lesion in the right hemicord at the C4 to C5 level in keeping with contusion. In addition, there was loss of flow void in the left vertebral artery concerning for possible thrombosis/dissection (see Figure 1). Thick axial slices covering V1 (=C6 and below) segment showed variable signal and caliber of the left vertebral artery (one cut with flow void, one cut with complete obstruction and hyperintense thrombus, a few cuts with decreased caliber); all the other segments (V2, V3, V4) were occluded, replaced by hyperintense T1 signal from obstructive thrombus. Note that no magnetic resonance angiography was done on the initial imaging, and V1 was not imaged.

After the initial spine imaging, he was intubated and went directly to the operating theater. He underwent an open reduction of C4 to C5 with posterior spinal fusion of C4 to C6. He was diagnosed to have a C5 fracture with C4 to C5 bilateral facet joint dislocation. Magnetic resonance imaging brain and cervical spine and magnetic resonance angiography of the circle of Willis post-surgery (D1) showed an improved cervical spine alignment to near anatomical alignment and residual T2 signal changes in the right hemicord over C4 and C5. In addition, there was an absence of flow within the intracranial segments of the left vertebral artery, corresponding to T1 signal and loss of flow void on T2-weighted sequence in keeping with extensive thrombosis of the left vertebral artery including its intracranial segment. There was an absence of flow and an acute obstructive endoluminal thrombus extending from V1 (partial) to V4 evident. Important to mention that the brain MRI was limited in part due to orthodontic braces, but otherwise was normal (see Figure 2). The patient was not started on antiplatelet or anticoagulation given the imminent need for spinal surgery. Heparin was started at 24 hours following surgery due to



**Figure 2.** Magnetic resonance imaging (MRI) after spine surgery (day 1) showing persistent unchanged absence of T2 flow void of the left vertebral artery (arrowheads) in sagittal view (A), and axial view (B) including the intracranial vertebral artery segment. B, Reduced flow void in the intracranial segment on the left vertebral artery (arrowhead) compared with the right vertebral artery. Also, loss of flow signal seen on magnetic resonance angiography (MRA) sequence (C) in the left vertebral artery (arrowhead) with present flow in the right vertebral artery (arrow).

concerns about expansion of small epidural hematoma at the C5 vertebral body, and the neurosurgical team felt it was safest to wait 24 hours prior to initiating anticoagulation to ensure the bleed was not progressing and to ensure no further operative procedures would be needed. During his stay in the ICU, there was no headache, diplopia, or vomiting.

On examination at day 2 (after 24 hours post-surgery), the patient was not encephalopathic and had good speech production. Mild dysarthria and hoarseness of voice were observed. His pupils were equal, round, and reactive to light. He had subjective tingling in the V2 and V3 distribution of left trigeminal nerve, reduced corneal reflex on the left, and mild left facial weakness. There was left gaze-evoked nystagmus and dysmetria on the left upper and lower extremities. He had weak palatal elevation on the left and a weak gag reflex. The uvula was deviated to the right. His tongue was midline. Shoulder elevation was normal; however, his strength was reduced in the right upper extremity, in the C5 innervated muscles. Sensory examination showed reduced sensation to pain in the right upper and lower extremities. Strength in the left upper extremity and bilateral lower extremities was normal. Reflexes were normal except reduced in the right upper extremity. Plantar responses were both down going. The exact time of onset of these symptoms was not clear to the patient but he felt them after the surgery.

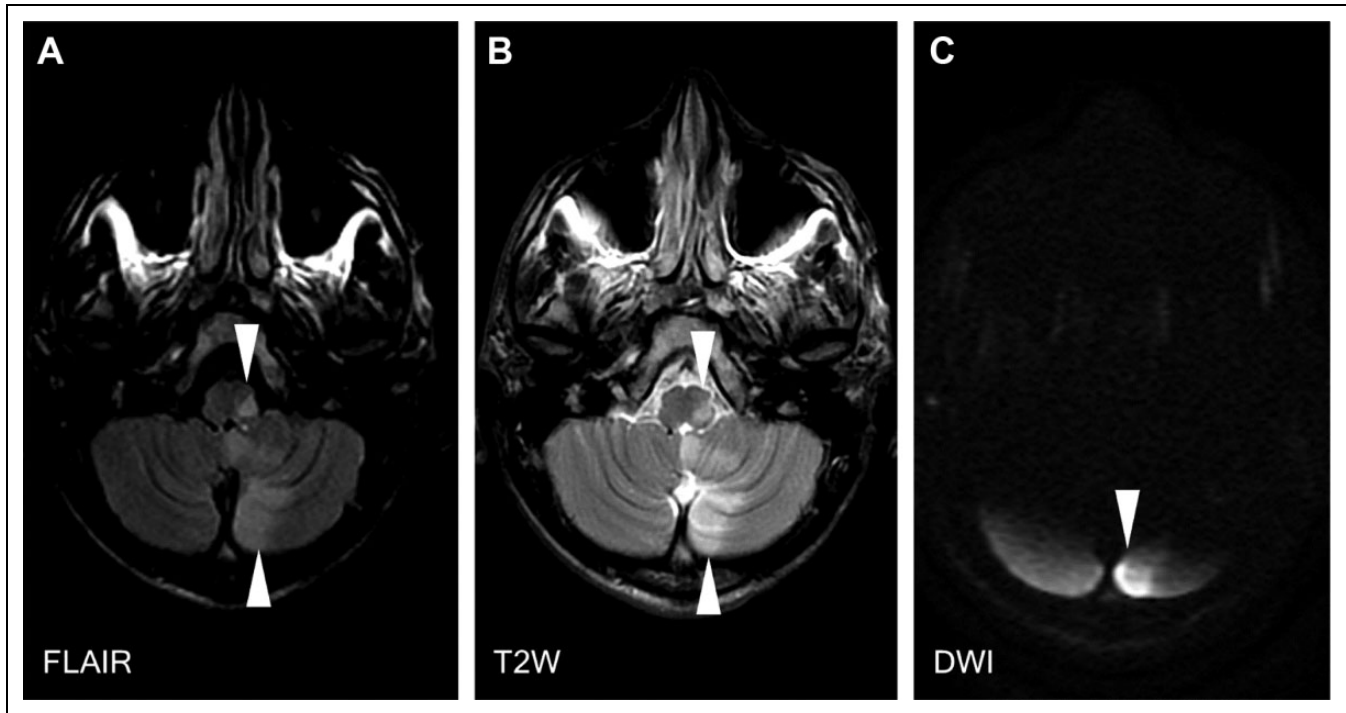
Given the onset of a likely embolic stroke and the visualization of the arterial thrombus on imaging, it was felt that anticoagulation with heparin would be most beneficial. The patient was started on intravenous unfractionated heparin around 30 hours post-surgery. Later that day, the patient developed persistent recurrent hiccups that responded to chlorpromazine and

resolved in one day. His brain MRI at day 2 (post neurological symptoms) showed interval development of T2W/fluid-attenuated inversion recovery signal abnormality within the left lateral medulla and inferomedial aspect of the cerebellum in keeping with infarcts secondary to the left vertebral artery thrombosis and occlusion of the left posterior inferior cerebellar artery (see Figure 3). He was shifted to enoxaparin 6 days post the event. During that time, his persistent dysphagia prompted a gastrostomy tube placement prior to transfer to a rehabilitation center. The left vertebral artery remained occluded on imaging. After 3 months of enoxaparin treatment, the patient developed a postoperative hematoma after a nerve graft surgery and switched to aspirin. Pregabalin was started for neuropathic pain. On 10 months follow-up, all the symptoms recovered except for a slight hemianesthesia.

## Discussion

The authors report a 17-year-old healthy boy who presented with left lateral medullary syndrome after a flexion neck injury while diving in shallow water. Few cases of lateral medullary syndrome have been described in the pediatric population, and none have been yet described following a “flexion” neck injury.

The probable mechanism in our case was thrombosis or occlusion secondary to flexion neck trauma with possible dissection as an etiology. Local thrombus formation is promoted by luminal stenosis and the release of thrombogenic factors by intimal damage leading to a narrowed lumen and therefore reduced blood flow. The other possibility would be an embolic event from the original thrombus.<sup>9</sup> Endothelial injury, tearing,



**Figure 3.** Magnetic resonance imaging (MRI) 2 days after surgery showing interval development of T2/fluid-attenuated inversion recovery signal abnormality within the left lateral aspect of the medulla, left dentate nucleus, and inferior medial portion of the left cerebellum (A and B). The cerebellar abnormality is correlated with a posterior inferior cerebellar artery (PICA) distribution while the medullary finding likely relates to the known vertebral artery thrombosis. Unfortunately, due to artifact created by dental hardware there is obscuration of this area on the diffusion-weighted imaging (C) and apparent diffusion coefficient “ADC” (not shown); only the most inferior medial aspect of the posterior cerebellum is spared of the artifact and demonstrates restricted diffusion in keeping with an infarction (arrowhead).

and separation between vessel layers will cause exposure to collagen and in turn will activate tissue and von Willebrand factors that will lead to thrombus formation resulting in occlusion or artery-to-artery embolism.<sup>10</sup>

Vertebral artery dissection is often preceded by neck trauma. However, other predisposing etiologies include genetic connective tissue disorders predispose to eventual dissection such as Ehler-Danlos syndrome, fibromuscular dysplasia,  $\alpha$ 1 antitrypsin deficiency, angioliomatosis, lentiginosis, hyperhomocysteinemia, and Turner syndrome, which accounts for a very small proportion of cases. Our patient didn't have signs or symptoms of these disorders. Acute infections have been also associated with cervicocerebral dissection. Bone anomalies (Klippel-Feil anomaly, odontoid aplasia, atlanto-axial subluxation, and congenital arcuate foramen) can also cause vertebral artery occlusion such as that seen in rotational vertebral artery syndrome or “Bow hunter syndrome.”<sup>11</sup> A study done by Taneichi et al<sup>6</sup> (in adults) mentioned that the incidence of traumatically induced nonpenetrating vertebral artery occlusion was around 17.2% and is a complication in 20% of cervical spinal fractures and or dislocation. Blood flow restoration potential was higher in compressive injuries (suggesting a probable vasospasm or minor artery dissection leading to a reversible occlusion) than in distractive injuries. Also, vertebral artery occlusion was rarely symptomatic because of

sufficient collateral blood supply through not only the contralateral vertebral artery but also the circle of Willis.<sup>6</sup>

Given lateral medullary syndrome is not that common in pediatrics as opposed to adults screening tools such as the McGovern or the Denver could be used to assist in minimizing unnecessary radiation and determining which high-risk patients are in need of vascular imaging.<sup>12</sup> According to adult data, the most frequent presenting symptoms of lateral medullary syndrome are sensory in origin (61% face, 79% body), dizziness/vertigo or imbalance (88%), Horner syndrome (88%), nystagmus (70%), nausea/vomiting (64%), dysphagia (61%), and hoarseness (39%).<sup>13,14</sup> The clinical presentation varies and depends on the precise location of the ischemia affecting the lateral medulla and the inferior medial cerebellum in the territories supplied by the vertebral artery and posterior inferior cerebellar artery. In our case, the patient had both an extracranial and intracranial thrombus. It has been reported that intracranial dissections are more common in males, while extracranial dissections are more common in females.<sup>9</sup> Also, our patient developed hiccups which was found in 25% of adult patients with lateral medullary syndrome.<sup>10,15</sup>

The authors found 15 cases of lateral medullary syndrome reported in the pediatric population (Table 1). Ten cases were diagnosed by MRI and 2 with changes in arteriogram (radiograph with contrast). Age was between 6 and 15 years old with

**Table 1.** Pediatric Cases of Lateral Medullary Syndrome.

#	References	Case	Sex	History/Etiology	Diagnosis
1	Monteventi et al <sup>16</sup>	8-year-old 9-year-old 13-year-old	M M M	Lyme brucellosis Lyme brucellosis Lyme brucellosis	MRI: stroke in Rt PICA territory MRI: Rt cerebellum (Rt PICA territory), Rt and Lt VA stenosis. Proximal Basilar Artery stenosis MRI: Lt PICA territory Lt VA stenosis near PICA emergence
2	Allen and Jungbluth <sup>17</sup>		M	Lyme brucellosis	MRI: infarcts in a vertebrobasilar distribution, affecting medulla, pons, and cerebellum. MRA demonstrated vessel irregularity or “beading” in the Circle of Willis, suggesting vasculitis
3	Ehresmann et al <sup>18</sup>	7-year-old	M	Minor trauma	MRI: a small infarct on the lateral posterior left part of the medulla oblongata. MRA normal.
4	Kibe et al <sup>4</sup>	9-year-old	M	Hereditary dysfibrinogenemia after bread eating game? neck hyperextension or rotation	MRI: Lt dorsolateral aspect of the medulla oblongata
5	Sharafaddinzadeh et al <sup>19</sup>	15 years	M	Celiac disease: spontaneous	MRA revealed absence of flow in the Rt VA and stenotic lesion proximal of the Lt VA Brain MRI showed signal abnormality in the Rt cerebellum and medulla
6	Ng et al <sup>13</sup>	10-year-old	F	Atrial septal defect repair at age 7 years, Recurrent sinusitis and otitis media, skull base osteomyelitis ( <i>Streptococcus milleri</i> )	MRI demonstrated Rt medullary lesion, Rt petrous apex enhancement, Rt sphenoidal, and maxillary sinusitis with increased meningeal enhancement post-gadolinium. T1 images showing right clivus signal changes suggestive of base of skull osteomyelitis
7	Toelle et al <sup>3</sup>	9-year-old	F	Swung on a rope looking to the sky with neck extension	MRI: lesion of the Lt dorsolateral medulla
8	Serrano et al <sup>20</sup>	10-year-old	F	Embolism post lightning strike	ND
9	Martinez et al <sup>21</sup>	ND	ND	Post radiofrequency treatment of tachyarrhythmias	ND
10	Kovacs et al <sup>2</sup>	6-year-old	M	Post varicella	MRI: lesion of the Rt medulla
11	Klein et al <sup>22</sup>	8-year-old	M	Minor fall on the Rt face	Arteriogram: Reflux down the Lt VA showed a sharp cutoff 6 mm from its junction with the basilar artery. There was no filling of the Lt PICA
12	Isler <sup>23</sup>	12-year-old	F	Spontaneous? Unknown	Arteriography disclosed subtotal VA occlusion at the commencement of the basilar artery, with normal filling of the PICA
13	Richwien and Unger <sup>24</sup>	13-year-old	M	Congenital heart disease? emboli	ND

Abbreviations: F, female; FU, follow up; IV, intravenous; Lt, left; M, male; MRA, magnetic resonance angiography; MRI, magnetic resonance imaging; ND, no details; PICA, posterior inferior cerebellar artery; Rt, right; Y, year; VA, vertebral artery.

11 of 15 patients being male. Two patients had minor antecedent trauma, 2 had neck extension events, 3 with possible embolisms (struck by lightning, congenital heart disease, and radiofrequency ablation procedure), and others were in the context of osteomyelitis, post-varicella, neuroborreliosis, or celiac disease. The initial presenting symptoms were vertigo or dizziness (10/15), headaches (7/15), vomiting (6/15), motor or sensory abnormalities (6/15), nausea (3/11), ataxia (3/15), swallowing difficulties (2/15), speech changes (1/15), neck pain (1/15), and photophobia (1/15). Aspirin was used in 4 cases, anticoagulation in one case, others received fluids, antibiotics, gluten restriction, or surgery as treatments depending on the associated condition. Neurological outcome was favorable in 4 patients having full recovery in 3 to 12 months. Others had persistent mild deficits including mild ataxia, mild hemiparesis, sensory deficits, nystagmus, and vertigo. One

died with eventual respiratory failure as a complication from aspiration pneumonia.

Regarding medical treatment, the decision was to go with anticoagulation based on the clinical signs and symptoms of lateral medullary syndrome instead of aspirin. In post dissection treatment of posterior circulation strokes, a pediatric study showed that 23% of the cases were treated with antiplatelets, 23% with anticoagulation, 14% with both, and 40% with either. The use of medical intervention (anticoagulation or antiplatelet agents) seemed to be increasing over time.<sup>8</sup> In an adult study, done by Markus et al,<sup>25</sup> on 250 participants with extracranial carotid and vertebral dissection who were randomized to antiplatelet or anticoagulation with 1-year follow-up, the result showed that the number of recurrent strokes was low (2.4%), and there was no difference between treatment groups in outcome events or the rate of recanalization.

The American College of Chest Physicians suggested for the treatment of arterial ischemic stroke that are nonsickle cell related to use anticoagulation with LMWH/unfractionated heparin or aspirin as an initial therapy until the dissection or the embolic causes are excluded then used aspirin daily for 2 years. If the arterial ischemic stroke was secondary to a dissection or a cardiac emboli to use LMWH or vitamin K antagonists for at least 6 weeks with ongoing treatment depending on the follow-up imaging.<sup>26</sup> Perioperative decision of anticoagulants and antiplatelet agents is based on a compromise between the risk of hemorrhage induced by maintaining these agents and the risk of further thrombosis if they are discontinued. In our patient, anticoagulation was started about 24 hours after the spinal surgery as this surgery could be considered a major bleeding risk and the patient already had evidence of an epidural hematoma.

A study done by Hasan et al<sup>27</sup> on pediatric vertebral artery dissection of 30 cases mentioned that 15 cases were treated with only antiplatelet, 8 cases with anticoagulation followed with antiplatelets, or 7 cases followed without supplemental antiplatelet therapy. Full neurological recovery occurred in 80% of the patients who received antiplatelet therapy alone compared with 27% of those who received anticoagulation therapy with or without antiplatelet therapy. They concluded that the role of different therapies for children presenting with symptoms related to vertebral artery dissection is very unclear. A randomized multicenter prospective trial in children should be considered to determine whether antiplatelet and/or anticoagulation therapies are appropriate for children presenting with VAD. In another study done in children with posterior circulation dissection: 1 (2%) of 45 cases died before 1990 and none after 1990. Among survivors (over 5-year follow-up), 37% had complete recovery, 33% had mild deficits, 8% with moderate deficits, and 6% with severe deficits. None had recurrent dissection, but 15% had recurrent ischemia. They presumed that anticoagulation possibly reduces a patient's risk of a thromboembolic event after a dissection.<sup>8</sup> In general, the prognosis is better in children than adults with dissection and in extracranial rather than intracranial dissection. Moreover, the highest dissection recurrence rate was in intracranial arterial dissection 5 weeks to 14 months post initial diagnosis.<sup>28</sup>

Our case demonstrates that lateral medullary syndrome can occur postflexion injury in the pediatric population. The etiology relates probably to a vertebral artery dissection. The presence of crossed neurologic findings above and below the neck in the context of neck injury is an important diagnostic clue that should prompt imaging study focusing on the brain stem and the posterior fossa vascular structures. However, a high level of suspicion should also be kept in mind in asymptomatic patients, and screening tools could be used to assist in minimizing unnecessary radiation and determining which high-risk patients are in need of vascular imaging. There is as yet no definite standard treatment for vertebral artery dissection. Decisions regarding need and type of treatment are often made by the treating team balancing the benefits of anticoagulation the risks of bleeding. Most of ischemic lesions are a consequence of a thrombotic or

embolic phenomenon. Therefore, a course of anticoagulation might be considered if there is no intracranial hemorrhage or major systemic hemorrhage. In intracranial dissection, more caution is required as they may develop a subarachnoid hemorrhage. Good neurological recovery on anticoagulation or antiplatelets therapy started soon after surgical treatment of traumatic injuries has been observed in this case and in the literature.

### Authors' Note

This link [http://n.neurology.org/content/90/15\\_Supplement/P6.217](http://n.neurology.org/content/90/15_Supplement/P6.217) was a resident poster presentation at the AAN "American academy of neurology conference".

### Acknowledgments

The authors thank Dr S. Abish, Dr F. Bhanji, and Dr R. Al-Sharhan for their help.

### Author contributions

All authors participated in gathering the data, designing the article, and discussing and editing the manuscript.


### Declaration of Conflicting Interests

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

### Funding

The authors received no financial support for the research, authorship, and/or publication of this article.

### ORCID iD

Abdulla Alawadhi  <https://orcid.org/0000-0001-7014-3056>

### Ethical Approval

Consent was taken from the patient.

### References

1. Rollins N, Pride GL, Plumb PA, Dowling MM. Brainstem strokes in children: an 11-year series from a tertiary pediatric center. *PNU Pediatr Neurol*. 2013;49(6):458-464.
2. Kovacs SO, Kuban K, Strand R. Lateral medullary syndrome following varicella infection. *Am J Dis Child (1960)*. 1993; 147(8):823-825.
3. Toelle SP, Albisetti M, Holzmann D, Steinlin M. A 9-year-old girl who swung on a rope. *Lancet*. 1997;350(9090):1516.
4. Kibe T, Ikeya M, Yokochi K, Okumura N. Lateral medullary syndrome in a boy with hereditary dysfibrinogenemia. *Brain Dev*. 2012;34(10):857-860.
5. Sacco RL, Freddo L, Bello JA, Odel JG, Onesti ST, Mohr JP. Wallenberg's lateral medullary syndrome. Clinical-magnetic resonance imaging correlations. *Arch Neurol*. 1993;50(6): 609-614.
6. Taneichi H, Suda K, Kajino T, Kaneda K. Traumatically induced vertebral artery occlusion associated with cervical spine injuries: prospective study using magnetic resonance angiography. *Spine*. 2005;30(17):1955-1962.

7. deSouza RM, Crocker MJ, Haliasos N, Rennie A, Saxena A. Blunt traumatic vertebral artery injury: a clinical review. *Eur Spine J*. 2011;20(9):1405-1416.
8. Fullerton HJ, Johnston SC, Smith WS. Arterial dissection and stroke in children. *Neurology*. 2001;57(7):1155-1160.
9. Thanvi B, Munshi SK, Dawson SL, Robinson TG. Carotid and vertebral artery dissection syndromes. *Postgrad Med J*. 2005; 81(956):383-388.
10. Andrade A, Yau I, Moharir M. Current concepts in pediatric stroke. *Indian J Pediatr*. 2015;82(2):179-188.
11. Greiner HM, Abruzzo TA, Kabbouche M, Leach JL, Zuccarello M. Rotational vertebral artery occlusion in a child with multiple strokes: a case-based update. *Childs Nerv Syst*. 2010;26(12): 1669-1674.
12. Herbert JP, Venkataraman SS, Turkmani AH, et al. Pediatric blunt cerebrovascular injury: the McGovern screening score. *J Neurosurg Pediatr*. 2018;21(6):639-649.
13. Ng J, Connolly DJ, Rittey CD, Mordekar SR. Skull base osteomyelitis leading to lateral medullary syndrome in a child. *Eur J Paediatr Neurol*. 2007;11(2):111-114.
14. Kim JS, Choi-Kwon S. Sensory sequelae of medullary infarction: differences between lateral and medial medullary syndrome. *Stroke*. 1999;30(12):2697-2703.
15. Kim JS. Pure lateral medullary infarction: clinical-radiological correlation of 130 acute, consecutive patients. *Brain*. 2003; 126(pt 8):1864-1872.
16. Monteventi O, Steinlin M, Regényi M, RouletPerez E, Weber P, Fluss J. Pediatric stroke related to Lyme neuroborreliosis: data from the Swiss NeuroPaediatric Stroke Registry and literature review. *Eur J Paediatr Neurol*. 2018;22(1): 113-121.
17. Allen NM, Jungbluth H. Lyme neuroborreliosis: a potentially preventable cause of stroke. *J Pediatr*. 2016;170:334-e331.
18. Ehresmann AM, Van HC, Merlini L, Fluss J. Wallenberg syndrome: an exceptional cause of acute vertigo in children. *Neuropediatrics*. 2016;47(1):61-63.
19. Sharafaddinzadeh N, Tirdad R, Pipelzadeh M, Ali AA. Wallenberg syndrome as a sole presentation of celiac disease. *Neurosciences (Riyadh)*. 2008;13(2):179-181.
20. Serrano ML, Gari M, Castro P, Garzo C, Maroto C, Maroto E. Wallenberg syndrome in childhood secondary to the striking by lightning. *Rev Neurol*. 1995;23(120):414-416.
21. Martinez JL, Monedero CM, Alvaro EM, et al. Radiofrequency treatment of tachyarrhythmias in children and adolescents. *Rev Esp Cardiol*. 1995;48(12):820-827.
22. Klein RA, Snyder RD, Schwarz HJ. Lateral medullary syndrome in a child. Arteriographic confirmation of vertebral artery occlusion. *JAMA*. 1976;235(9):940-941.
23. Isler W. *Acute Hemiplegias and Hemisyndromes in Childhood*. EH Burrows (trans), *Clinics in Developmental Medicine No 41/42, Spastics International Medical Publications*. Philadelphia, PA: JB Lippincott Co; 1971.
24. Richwien R, Unger G. Study of a case of Wallenberg's syndrome in childhood in addition to observations on cerebral vascular processes in children [in German]. *Monatsschr Kinderheilkd*. 1966;114(8):442-443.
25. Markus HS, Levi C, King A, Madigan J, Norris J, Cervical Artery Dissection in Stroke Study Investigators. Antiplatelet therapy vs anticoagulation therapy in cervical artery dissection: the Cervical Artery Dissection in Stroke Study (CADISS) randomized clinical trial final results. *JAMA Neurol*. 2019;76(6):657-664.
26. Monagle P, Chalmers E, Chan A, et al. Antithrombotic therapy in neonates and children: American college of chest physicians evidence-based clinical practice guidelines (8th edition). *Chest*. 2008;133(6):887S-968S.
27. Hasan I, Wapnick S, Tenner MS, Couldwell WT. Vertebral artery dissection in children: a comprehensive review. *Pediatr Neurosurg*. 2002;37(4):168-177.
28. Schievink WI, Mokri B, Piepgras DG. Spontaneous dissections of cervicocephalic arteries in childhood and adolescence. *Neurology*. 1994;44(9):1607-1612.