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Unilateral anoxic brain injury secondary to strangulation identified on conventional and arterial spin-labeled perfusion imaging

D. Dominik Prosser MD[a], *Tamara Grigsby MD*[b], *Jeffrey M. Pollock MD*[a,*]

[a] *Department of Neuroradiology, Oregon Health & Science University, 3181 Sam Jackson Rd, Portland, OR 97239, USA*

[b] *Department of General Pediatrics, Oregon Health & Science University, 700 SW Campus Drive, Portland, OR 97239, USA*

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ABSTRACT

Anoxic brain injury on magnetic resonance imaging classically demonstrates symmetric diffusion restriction involving the highly metabolic structures including the basal ganglia and cortex and global hyperperfusion on arterial spin labeling perfusion. The pattern of injury is classically diffuse and bilateral owing to global oxygen deprivation from systemic causes, most commonly cardiac arrest. In cases of suspected nonaccidental trauma presenting with a unilateral anoxic injury pattern, strangulation with temporary occlusion of a unilateral carotid artery should be considered. We present 2 cases of unilateral anoxic brain injury due to strangulation identified on magnetic resonance imaging and arterial spin labeling perfusion.

Introduction

Nonaccidental trauma (NAT) is a common cause of both traumatic injury and death in the pediatric population of the United States. Making a diagnosis can be particularly difficult, relying upon physical exam, specific imaging patterns, and a detailed history, all in the context of collaboration with multiple medical professionals across various specialties. Although the ramifications of incorrectly diagnosing NAT, including creating undue stress on the family, prolonged hospital stays, and compromising the doctor-patient relationship are considerable, the ramifications of missed cases of NAT are of far greater concern considering the long-term sequelae of NAT, both physical and psychosocial [1].

Due to the variety of ways a patient can present in cases of NAT, it is important for all involved to have broad knowledge regarding concerning findings. One such finding is unilateral anoxic brain injury due to external compression of the carotid artery, which can manifest as unilateral restricted diffusion on diffusion weighted imaging and hyperperfusion on perfusion imaging. We present 2 cases of nonaccidental

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* *Corresponding author.*
E-mail address: pollockj@ohsu.edu (J.M. Pollock).

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trauma resulting in unilateral anoxic brain injury with special emphasis on arterial spin-labeled perfusion imaging (ASL) assisting in making the diagnosis.

Case 1

Patient 1 is a 4-year-old male transferred from an outside hospital after noncontrast head computed tomography (CT) imaging revealed a small subdural hematoma. The patient presented with headache and 1 day history of stomachache with decreased appetite. The foster mother's fiancé reported the patient had slept most of the afternoon. The foster mother (maternal cousin) arrived home later and described the child being like "jello" with complaints of light hurting his eyes. As caregivers got him out of bed to take him to the hospital, the patient collapsed, striking his head on the bedframe. There was no loss of consciousness, vomiting, fever, or recent illness. One and and a half weeks prior, the child had vomited after receiving immunizations at a well child visit. Other than constipation, he was reportedly healthy. The patient had been living with his kinship foster mother, her fiancé, and their 2-year-old son for 6 months after being taken into protective custody by child welfare for neglect by polysubstance abusing parent.

The child was admitted to Pediatric Intensive Care Unit with Peds NS, Peds Trauma Surgery and Child Abuse Team consulting. In addition to scalp bruising and hematoma documented at outside hospital, physical examination was notable for bruising to both sides of the mandible, left cheek, lower back, and right buttocks. Skeletal survey demonstrated right 7th and left 6th and 7th anterior healing rib fractures. Ophthalmology diagnosed visual field cut, oculus sinister (OS), with no retinal hemorrhages.

Initial CT of the head performed at the outside facility for patient 1 demonstrated layering subdural hemorrhage on the right tentorium cerebelli and concern for right temporoparietal lobe ischemia. These findings were confirmed upon transfer to our hospital when repeat CT was performed. Also noted was 4 mm midline shift (Fig. 1).

Magnetic resonance imaging (MRI) and magnetic resonance angiography with ASL perfusion imaging performed at our institution on the day of transfer revealed unilateral restricted diffusion predominantly involving the right cerebral cortex and deep grey matter (Fig. 2A). Concomitant hyperperfusion was confirmed by ASL perfusion imaging in the regions of restricted diffusion (Fig. 2B). Evaluation of the cervical vasculature revealed patent vessels without dissection or thrombus.

Perfusion and diffusion imaging findings led the neuroradiologist to posit temporary occlusion of the carotid artery ipsilateral to the affected brain via strangulation as the possible etiology for the unilateral anoxic pattern. Forensic investigation revealed strangulation to indeed have occurred. After a prolonged stay in the hospital, patient 1 was discharged in the care of nonkinship foster parents.

Patient 1 experienced long-term left sided neglect and weakness upon discharge, which improved with rehabilitation. He remains in foster care and is engaged in weekly behavioral therapy for childhood post-traumatic stress disorder.

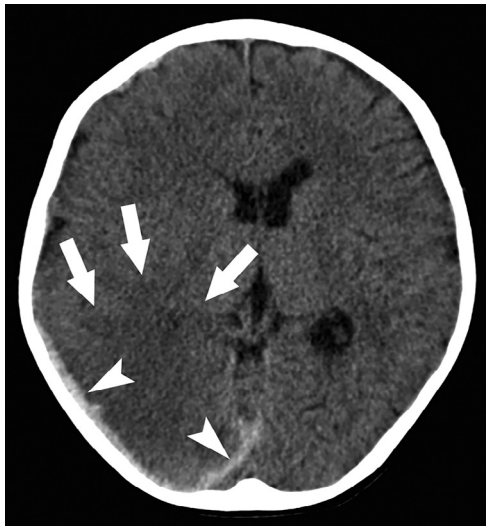

Fig. 1 – Case 1. Nonenhanced CT head performed at an outside facility showing hyperdense subdural blood products on the right tentorium cerebelli (arrowheads). Also shown is hypoattenuation and loss of gray-white differentiation in the posterior right MCA distribution (arrows) and 4 mm leftward midline shift.

Case 2

Patient 2 is a 12-month-old male who was transferred from an outside hospital for further Peds NS and Trauma Surgery evaluations of suspected NAT. He presented to the outside hospital emergency department with altered mental status, facial bruising, and report of a 4-foot fall from bed onto a carpeted floor. Noncontrast CT imaging revealed left subdural hematoma, left scalp hematoma, and left 8th- 11th posterior rib fractures with associated extrapleural hematoma. Upon arrival to tertiary care center, Child Abuse Team was consulted. Bruises to left and right mandible and linear distribution of petechiae extending across anterior neck were observed, in addition to bruises of face, bilateral external ears, and scrotum.

At the time of injury, the patient was living with his kinship foster mother (maternal aunt) and her boyfriend for 2 weeks after child welfare agency took protective action due to biological father's recurrent substance abuse. The patient had been living with his biological father since 1 month of age after being removed from the biological mother's home for failure to thrive and medical neglect. The Pediatric Intensive Care Unit course was complicated by focal seizures and ischemic stroke with right hemiparesis. Seizures were managed with levetiracetam and gabapentin was initiated to control neuropathic pain. Ophthalmology dilated examination documented severe multi-layer retinal hemorrhages OS > OD and large dense subhyaloid hemorrhage extending through the central macula, OS.

The initial CT performed at the outside facility demonstrated anterolateral and vertex left subdural hematoma without findings to suggest ischemia (Fig. 3).

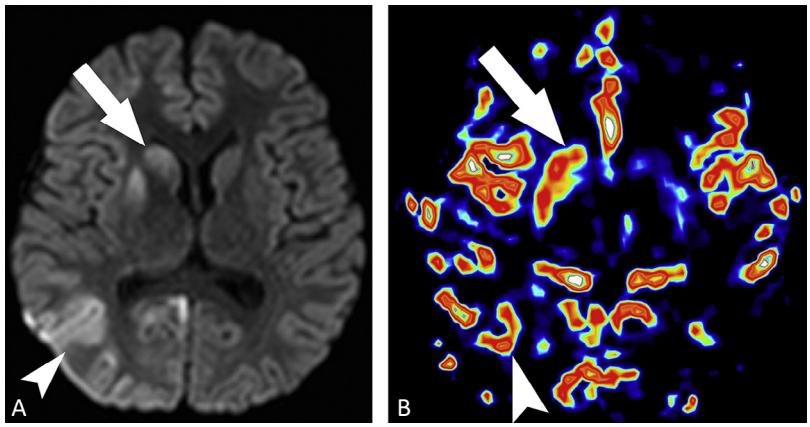

Fig. 2 – Case 1. (A) Axial B1000 image shows diffusion restriction in the basal ganglia (arrow) and cortex of the right cerebral hemisphere (arrowhead). (B) Nonquantitative single phase arterial spin labeling perfusion image demonstrates hyperperfusion in a unilateral hemispheric regional pattern with the areas of hyperperfusion matching the areas of diffusion restriction in the right basal ganglia (arrow) and cortex (arrowhead).

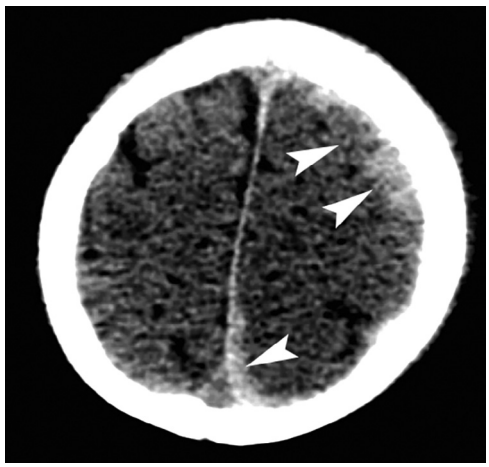

Fig. 3 – Case 2. Nonenhanced computed tomography head performed on patient 2 at an outside facility showing hyperdense left frontal and posterior falx subdural blood products (arrowheads).

MRI and magnetic resonance angiography with ASL perfusion imaging was subsequently performed on the day of transfer and demonstrated diffuse left hemispheric cortical and left thalamic restricted diffusion (Fig. 4A). Hyperperfusion was identified by ASL perfusion imaging, corresponding to the regions of restricted diffusion (Fig. 4B). Evaluation of the cervical vasculature revealed patent vessels without dissection or thrombus.

Perfusion and diffusion imaging findings led the neuroradiologist to posit temporary occlusion of the carotid artery ipsilateral to the affected brain via strangulation as the possible etiology for the unilateral anoxic pattern. Forensic investigation revealed strangulation to indeed have occurred. After a prolonged stay in the hospital, patient 2 was discharged in the care of nonkinship foster parents.

Patient 2 is followed by neurology for right hemiparetic cerebral palsy; seizures have resolved. Expressive language delay and articulation disorder are most significant; he receives rehabilitative physical, occupational, and language therapies weekly and is transitioning from foster family to biological mother.

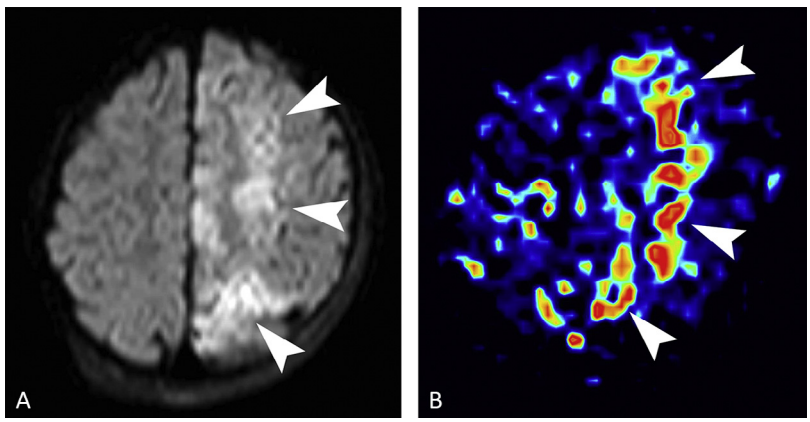

Fig. 4 – Case 2. (A) Axial B1000 image demonstrates diffusion restriction in the left frontal and parietal cortex (arrowheads). (B) Nonquantitative single phase arterial spin labeling perfusion image shows hyperperfusion in a unilateral hemispheric regional pattern with the areas of hyperperfusion matching the areas of diffusion restriction (arrowheads).

Discussion

Although diagnosing NAT is largely clinical, the radiologist plays a vital role as many of the diagnostic findings are image-based with particular emphasis on patterns of injury. Abusive head trauma can manifest in many ways, including hypoxic-ischemic injury, intracranial hemorrhage, retinal hemorrhages, and skull fractures [2,3]. The suspicion for NAT is amplified when multiple such findings are present simultaneously or separated in time [2]. Bruises to ears, neck, or genitalia are rarely associated with accidental mechanisms of injury in infants and children. Neck and facial petechiae and bruises involving mandible, submental space, and neck warrant further evaluation for strangulation or other asphyxiation [4,5].

Anoxic brain injury is the result of prolonged cessation of oxygen supply to the brain, typically symmetrically affecting the bilateral cerebral hemispheres due to systemic processes resulting in global hypoperfusion such as cardiac or respiratory arrest [1–3,6–10]. Unilateral anoxic brain injury is a unique pattern suggestive of unilateral occlusion or stenosis of a proximal large vessel supplying the entirety of the anterior circulation; namely, the cervical carotid artery [11]. It has been proposed that the pediatric brain is particularly susceptible to anoxic injury due to generalized lower mean arterial pressure when compared to adults [3]. It is believed that during an anoxic event, underlying metabolic changes result in loss of autoregulation and decreased vascular resistance, which, in turn, lead to marked hyperperfusion on ASL [6,7]. It has also been shown in animal models that ischemia leads to an increase in ASL perfusion signal secondary to a change in the blood brain barrier permeability [12].

The classically described anoxic brain injury can be detected at its earliest stages as restricted diffusion on MRI which involves the grey matter of the cortex and basal ganglia in a symmetric and bilateral pattern [1–3]. Measuring perfusion has classically employed contrast enhanced CT and MR techniques [13,14]. ASL is an alternative to these techniques that is capable of quantitatively or nonquantitatively measuring cerebral blood flow without requiring intravenous contrast. ASL is classically performed by applying a pulsed RF signal to the inflowing cerebral blood in the neck, thus labeling blood water protons that will cross the blood brain barrier and travel to the cerebral parenchyma in proportion to the degree of blood flow [8,15]. Anoxic brain injuries have been described with ASL imaging. With quantitative ASL imaging there is global hyperperfusion of all territories after an anoxic injury [7]. With nonquantitative ASL imaging, there is a regional pattern of hyperperfusion that matches the areas of diffusion restriction. Anoxic injury is believed to cause a disruption in autoregulatory mechanisms and possibly change the blood brain barrier permeability leading to the finding of hyperperfusion on ASL [12].

Sequelae of anoxic brain injury can be substantial with profound long-term consequences including motor and sensory deficits, speech deficits, and behavioral issues [1–3]. Such is the case with the 2 described patients.

In the literature, there are 2 additional reported pediatric cases of unilateral hypoxic-ischemic brain injury occurring in patients aged 7 months and 14 months. Diffusion-weighted imaging in these cases showed restriction in a unilateral hemispheric distribution. In each case, the findings were attributed to unilateral cervical vascular compression in NAT. Perfusion imaging was not performed in either case [2]. In our cases, unilateral diffusion restriction was also demonstrated without cervical carotid artery occlusion or dissection. Additionally, use of ASL perfusion confirmed hyperperfusion in the regions of restricted diffusion, thus assisting in the diagnosis of anoxic injury. Radiologists should be aware of the unilateral anoxic injury pattern and in cases of NAT, strangulation should be proposed as a possible etiology. Preexisting stenosis of 1 carotid might predispose an individual to develop a unilateral anoxic pattern with a global insult, therefore it is prudent to perform vascular imaging in such anoxic cases.

Conclusion

Unilateral anoxic injury is an uncommon radiographic finding identified on diffusion and perfusion imaging which when present should prompt suggestion of ipsilateral temporary vessel occlusion secondary to strangulation as a possible etiology especially in suspected cases of nonaccidental trauma.

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