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Athletics, minor trauma, and pediatric arterial ischemic stroke

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Abstract

Pediatric arterial ischemic stroke may occur as the result of trivial head or neck trauma sustained during a sports activity. We describe three cases of sports-related stroke in previously healthy school-age children and discuss acute and long-term stroke care. Possible mechanisms of sports-related stroke are addressed, as is evaluation for cause of stroke in children. In one of the reported cases, the child was found to have a vertebral artery dissection as the cause of his stroke, but no definitive cause of stroke was identified in the other two cases despite extensive evaluation. The advisability and timing of returning to athletic activities after stroke is also discussed. Many children with sports-related stroke are initially seen by a sports trainer, a pediatrician, or an ER physician. Thus, it is particularly important that these professionals are aware of the possibility of ischemic stroke occurring after even mild athletic injury. Childhood stroke may result from injuries sustained during athletic activities and should be considered when a child has acute focal neurologic signs.

Keywords

Pediatric stroke; Sports-related stroke; Athletic injury; Arterial dissection

Introduction

Children frequently sustain serious injuries such as broken bones, torn ligaments, and concussions as a result of athletic activity. Though much less common, pediatric arterial ischemic stroke (AIS) can also occur as the result of the trivial head or neck trauma that occurs during a sports activity.

AIS occurs in approximately two to three per 100,000 children per year [10]. Conditions such as congenital heart disease, sickle cell disease, infection, arterial dissection, and prothrombotic disorders have been associated with AIS in children, and mild head and neck trauma has also been identified as a risk factor. Often the cause and mechanism of pediatric AIS remain unknown even after thorough evaluation.

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We report three cases in which neck trauma during athletic activity preceded AIS and discuss the significance of these cases in terms of acute and long-term care of child athletes with AIS.

Case descriptions

Case 1—A 10-year-old boy developed a left hemiparesis after colliding with another player during a soccer game. The force of the impact was focused on his right head and neck. Within a few minutes, he developed drooping of the left side of the face, dysarthria, and significant left arm and leg weakness. By the time he reached the hospital, his symptoms were improving but not gone. Left-sided weakness and an extensor plantar response of the left foot were noted. His past medical and family histories were unremarkable.

Magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) revealed a right basal ganglia stroke (Fig. 1). Non-contrast MRA of the neck, 1.5 tesla (T), performed 2 h after presentation to the ER was unremarkable. Axial MRI with fat saturation was not done. A CT angiogram that was done 4 h later did not show a large vessel dissection, but traumatic arterial dissection was still felt to remain a probable cause of his stroke, and the patient was started on unfractionated heparin (UFH). Four-vessel digital subtraction angiography (DSA) performed 76 h after admission also showed no evidence of extra- or intracranial arterial dissection, and the patient was transitioned from UFH to aspirin. He was found to be heterozygous for the factor V Leiden mutation and methylenetetrahydrofolate reductase (MTHFR) C677T polymorphism. He had a normal echocardiogram.

He recovered well and less than 2 months after the stroke had only subtle left-sided weakness. At a 2-year follow-up visit, he had no residual weakness and had returned to playing competitive soccer.

Case 2—A 12-year-old boy sustained right neck and shoulder trauma during a lacrosse game. He reported that his right arm and leg felt “funny” after the collision, but he did not leave the game. Later that night, he developed tingling of his right face, a severe headache, and right-sided neck pain. The next morning, he awoke with vomiting, right-sided weakness, and disturbed gait (falling to the right). He presented to the emergency room approximately 24 h after the neck trauma occurred with nystagmus, severe truncal ataxia, and dysmetria of the right upper and lower extremities.

MRI confirmed acute stroke in the right posterior pontomedullary junction. Contrast enhanced MRA (1.5 T) of the head and neck was normal. These studies were done within an hour of presentation for medical care while the patient was still in the ER, but this was more than 24 h after neck trauma occurred. Axial MRI of the neck with fat saturation was not performed. As traumatic vertebral artery dissection was suspected, he was started on UFH. Four-vessel DSA performed 12 h following hospital admission showed no signs of vessel abnormities or dissection, so UFH was replaced with aspirin. An echocardiogram revealed a tiny patent foramen ovale. Thrombophilia evaluation identified an elevated lipoprotein (a) level and heterozygosity for the MTHFR C677T mutation with a normal homocysteine.

One month after the stroke, the patient reported no deficits. Upon examination, he showed only very mild motor slowing of the right side and slight difficultly with tandem gait.

Case 3—A 7-year-old boy had acute onset of right-sided numbness and tingling approximately 3 h after karate practice. He had been sparring with another child and had received a karate chop to his neck. Ataxia and right hemisensory loss were noted at the time of presentation to a regional ER. An hour later in the ER, he developed flaccid right-sided weakness of the face, arm, and leg.

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His brain MRI showed acute infarcts in the left posterior parietal lobe, right cerebellum, and left medial medulla (Fig. 2a). UFH was started. When MRA of the neck (1.5 T) with contrast, performed immediately upon arrival at our hospital and 12 h after symptom onset, did not show an arterial dissection, he underwent four-vessel DSA of the neck, which showed a right vertebral artery dissection with intraluminal clot (Fig. 2b). He was transitioned from UFH to warfarin. Thrombophilia evaluation identified only heterozygosity for the MTHFR C677T mutation with a homocystine level of 5.4. He had a normal echocardiogram. Axial MRI of the neck with fat saturation (1.5 T) was performed 2 days after admission with plans to visualize and follow the vertebral artery dissection non-invasively. However, the dissection was not well seen on this specialized MRI, perhaps due to mild motion artifact.

He required inpatient rehabilitation, but at the 2-month follow-up visit, he had only mild gait ataxia and right arm weakness. Six-month follow-up DSA revealed occlusion of the vertebral artery at the dissected segment, but good collateral flow and no impairment in cerebral perfusion. Aspirin was started and warfarin was discontinued. One year after his stroke, he had no detectable weakness or unsteadiness.

Discussion

Many children with sports-related stroke are initially seen by a sports trainer, a pediatrician, or an ER physician. Thus, is it particularly important that these professionals are aware of the possibility of ischemic stroke occurring after even mild athletic injury. Any child with recent head or neck trauma who presents with acute stroke-like symptoms (dysarthria, unilateral weakness, or altered sensation that is unilateral) should be immediately evaluated for possible AIS. Stroke may occur days to weeks after a sports-related arterial dissection [9]. Therefore, in a child with stroke-like symptoms, primary care providers should consider AIS related to arterial dissection even if there is no history of head or neck trauma in the immediate past.

Diagnostic evaluation for suspected cervical artery dissection

A child presenting with stroke-like symptoms should be urgently evaluated with brain MRI and MRA or CT angiography of the head and neck. Some centers also perform axial MRI of the neck with fat saturation to assess for hematoma within the wall of the injured artery. As demonstrated by these cases, even when a dissection is thought to be present, it may not always be visualized, particularly with noninvasive imaging or when imaging is delayed [14]. A key point is that, at times, children must be treated presumptively with anticoagulation when there is a strong history and clinical picture suggestive of arterial dissection before a definitive diagnosis is made.

As is illustrated by our cases, diagnosis of an arterial dissection may require multiple imaging modalities. While catheter angiography is still considered the gold standard, recent reviews suggest that MRI and/or CTA provide highly sensitive and specific diagnostic information [6], and catheter angiography may only be needed in cases where noninvasive imaging is negative or inconclusive [20]. It should be noted that vertebral artery dissections represent a greater diagnostic challenge than carotid dissections for both MRI/MRA (e.g., flow artifacts and peri-arterial venous enhancement simulating a mural hematomas) and CTA (bone artifact, particularly at the skull base) [8]. All three of our patients underwent non-invasive vascular imaging, which was inconclusive, prior to having DSA. These children were all imaged prior to the availability of 3 T MRI at our center.

If an arterial dissection is deemed likely, conventional four-vessel DSA should be considered as it remains the gold standard imaging study for cerebrovascular diseases. An important consideration in decision-making regarding vascular imaging is the fact that catheter angiography is more operator dependent than noninvasive imaging and that invasive vascular

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imaging itself carries a small risk (0.4–1%) [5,15,21] of vascular trauma and stroke, which is dependent on the experience of the examiner. A recent study has shown, however, that classically feared complications such as arterial spasm, arterial dissections, and thrombo-embolic events leading to transient or permanent neurological deficits are nowadays extremely rare (no instance in a series of 241 consecutive pediatric cerebral angiograms) [5]. Nonetheless, it remains that many centers may have limited availability of neuroradiologists with experience in pediatric cerebral angiography. Thus, the small yet non-negligible risk of angiography must be balanced with the risk of 3 to 6 months of anticoagulation for presumptive arterial dissection on an institutional basis. Decisions regarding additional vascular imaging when MRI and MRA do not visualize a suspected arterial dissection must be individualized. In some cases, due to center- or patient-related factors, a decision must be made to treat a patient as though he or she has a cervical dissection in the absence of results from the “gold standard” angiogram.

Failure to find the cause of a pediatric stroke is not uncommon. In one study, 23% of patients with pediatric AIS had no clear stroke etiology [4]. All three of our patients developed symptoms of a stroke within minutes to hours of a sports injury with associated trauma to the neck, but despite thorough diagnostic investigations, including evaluation for embolic sources, no clear cause of stroke was identified in two of three children. The pattern of multiple foci of infarction in the posterior circulation seen in case 3 is rarely seen except in vertebral artery dissection and should be viewed as an important clue to the presence of a dissection.

Delayed diagnosis and pediatric stroke

These cases highlight the issue of delay in diagnosis of pediatric stroke. Delays may occur before the child reaches the hospital if the family does not recognize stroke-like symptoms and bring the child in for medical care, and additional delay may occur if the stroke is not recognized by healthcare providers. One study found that the median time to presentation for medical care in 24 children was 9 h for ischemic stroke and 10.5 h for hemorrhagic stroke [11]. A more recent study of 209 children with arterial ischemic stroke admitted to an academic medical center with pediatric stroke expertise found a delay of 1.7 h from symptom onset to hospital arrival but a 12.7-h delay from hospital presentation to diagnosis [16]. Even in this setting, a diagnosis of stroke was suspected at initial medical evaluation in only 38% of cases. Delay in diagnosis in pediatric stroke may have serious consequences as earlier diagnosis and treatment may prevent progression or recurrence.

Treatment of cervical artery dissection

Since there have been no controlled trials comparing antiplatelet therapy with anticoagulants, treatment for cervical artery dissection is based on expert opinion. Treatment with anticoagulation is typically recommended [6] because the mechanism of stroke after arterial dissection is thought to be embolization and propagation of thrombus after a tear in the wall of a blood vessel. Anticoagulation is usually for a period of 3 to 6 months to allow for healing of the vessel wall followed by aspirin [18]. These treatment recommendations are consistent with the recent American Heart Association guidelines for the management of stroke in infants and children [17]. If a dissection is not visualized on DSA, we recommend discontinuing administration of anticoagulants prior to hospital discharge and placing the patient on aspirin therapy for secondary stroke prevention.

Early diagnosis and treatment of dissection with anticoagulation may prevent recurrent stroke. The progression of symptoms in case 3 is representative of the known risk for early recurrence of stroke from cervical artery dissection; however, late recurrence is rare [1].

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Stroke mechanism after minor trauma

Due to the nature of the injuries sustained, one possibility is that our patients 1 and 2 had "occult dissections." For example, they may have had minor intimal injuries, which were either too small to be documented radiologically or had already healed at the time of angiography, acting as embolic sources. In the case of patient 2, it is also possible that the stroke resulted from cardioembolism due to the presence of a small patent foramen ovale. Patient 3 had a classic dissection of his vertebral artery, which led to multiple embolic strokes.

Stroke occurring in children after mild head trauma, unrelated to arterial dissection, has been reported, and several possible mechanisms have been hypothesized. Shaffer et al. described six cases of basal ganglia infarction in children and postulated that these strokes may have been caused by mechanical disruption of flow in perforating branches of the middle cerebral artery, intimal trauma followed by thrombosis, or arterial spasm following trauma [19]. Kieslich et al. described stroke in eight children and discussed post-traumatic endothelial lesions along with minor blood clotting abnormalities as a possible etiology for stroke [12]. Cases of stroke after physical activity and mild mechanical neck trauma have also been reported [10]. Mechanical neck trauma can contribute to arterial dissection [7], which can lead to stroke anywhere from seconds to weeks after the initial trauma. Approximately 7% to 20% of pediatric AIS cases are associated with arterial dissection [3].

Return to play after sports-related stroke

As most children with sports-related stroke are serious athletes, families frequently ask whether the child can continue their participation in athletics. Pediatric neurologists are divided on this issue, and at this time, there are no clear recommendations. Neurologists generally agree that patients should be encouraged to participate in non-contact and low-contact sports such as tennis or swimming instead of high-contact sports. In a survey of pediatric neurologists with stroke expertise, most suggested waiting at least 6 months before resuming contact sports. Some reported that they would never recommend participation in high-contact sports after AIS [2]. Those surveyed also showed more hesitation in letting a patient with an arterial dissection return to athletics in comparison to a patient with idiopathic AIS [2]. Though most studies find that the recurrence risk for arterial dissection is approximately 1% per year [6], the recurrence risk in children with sports-related AIS may be higher; rates of up to 30% have been cited [2, 13].

Conclusion

These three cases are representative of sports-related AIS, which is often associated with arterial dissection after neck trauma. Stroke mechanism is not always clear. When arterial dissection is strongly suspected based on clinical history, thorough vascular imaging with multiple modalities may be required to definitively exclude dissection. Screening for hypercoaguable state is also mandatory. Acute treatment for arterial dissection may include anticoagulation therapy; long-term treatment usually includes aspirin therapy and some restrictions on high-contact activities. These cases illustrate the critical importance of making a timely and specific diagnosis of stroke following minor head and neck trauma in child athletes. Childhood stroke may result from injuries sustained during athletic activities and should be considered when a child has acute focal neurologic signs.

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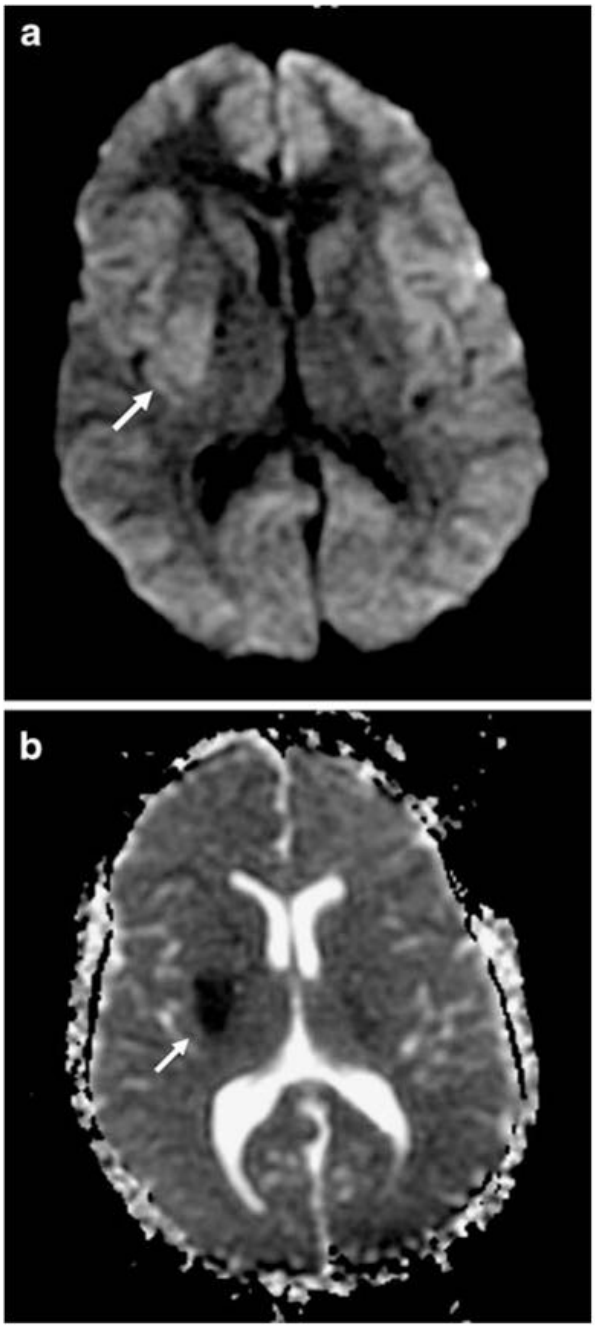

Fig. 1.
For case 1, diffusion-weighted MRI (**a**) shows faint bright signal, restricted diffusion, in the right basal ganglia. Apparent-diffusion coefficient image (**b**) is correspondingly *dark*, confirming acute ischemic infarction. *White arrows* indicate lesion site

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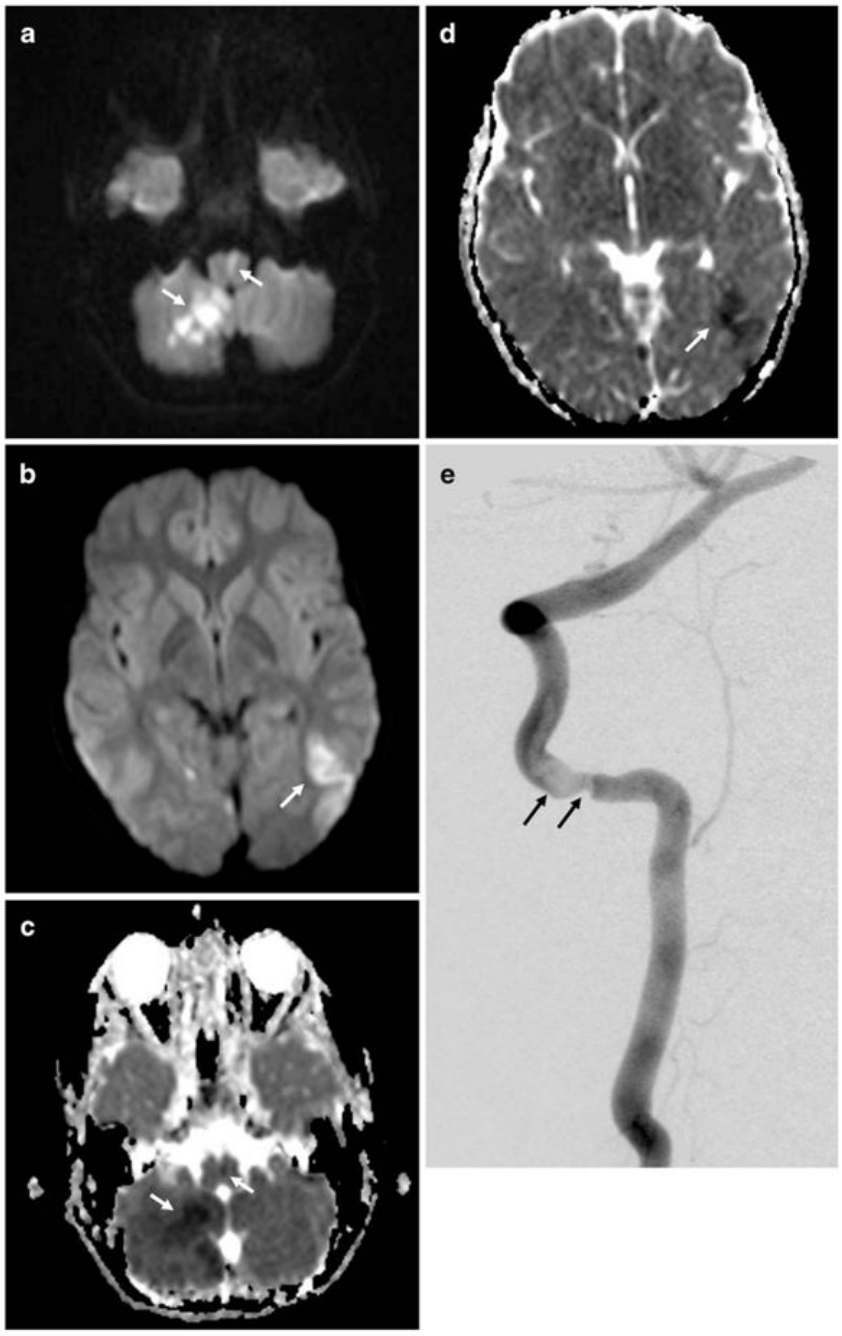

Fig. 2.

For case 3, diffusion-weighted MRI shows restricted diffusion in the right cerebellum, left medial medulla (**a**) and left posterior parietal lobe (**b**) with corresponding dark signal on apparent-diffusion coefficient images (**c** and **d**). *White arrows* indicate lesion sites. In **e**, digital subtraction angiography shows a right vertebral artery dissection with intraluminal clot (*black arrow*)