Anterior Spinal Artery Infarct Secondary to "Rotatory Atlantoaxial Subluxation" in a Pediatric Patient: A Case Report and Review of the Literature

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Abstract

Spinal cord infarction in children is a rare condition that is becoming more widely recognized. There are few reports in the pediatric literature characterizing etiology, diagnosis, treatment options, and prognosis of anterior spinal artery infarction following rotatory atlantoaxial subluxation. In many children, the cause of spinal cord ischemia in the absence of vertebral fracture is unknown and is often wrongly diagnosed. Imaging diagnosis of spinal cord ischemia is often difficult due to the small transverse area of the cord, cerebrospinal fluid artifact, and inadequate resolution. The prognosis depends on the level of spinal cord damage, early identification and reversal of ischemia, and follow-up with intensive physical therapy and medical support. In addition to summarizing the literature on spinal cord infarction in children following cervical trauma, this article adds to the literature another case of a child with anterior spinal artery territory infarction due to rotatory atlantoaxial subluxation, leading to kinking and occlusion of vertebral artery, which highlights the difficulties and controversies in the management of this condition.

Keywords

- anterior spinal artery
- infarction
- quadriparesis
- rotatory atlantoaxial subluxation
- vertebral artery occlusion

Introduction

Spinal cord infarctions (SCIs), especially in children, can have distressing long-term consequences, but we found very few descriptions of the etiologies, presentation, treatments, and outcomes of pediatric cord infarction in the literature. A review of the literature showed that dissection of the extracranial vertebral artery (VAD) usually leads to brainstem or posterior fossa infarctions rather than spinal cord involvement.[1] Compared with cerebral infarction, SCI is very uncommon in adults and children and can be difficult to distinguish from other myelopathies.[2] Cord infarction may be ischemic or hemorrhagic, and etiology ranges from hypoperfusion, thromboembolic events, trauma, iatrogenic to other rarer causes.[3] Among the various causes of anterior spinal artery (ASA) infarction, trauma is one of the uncommon etiological factors, and infarction can occur in the absence of any evident radiologic abnormality or vertebral injury that is often missed by the initial scans. One-third of patients can have poor outcome, and old age and ASA watershed infarction represented important risk factors.[4]

In this report, we describe the case of a young boy who presented with quadriparesis after an episode of trivial injury when he jerked his neck while breaking a stick with his knee. After thorough evaluation, he was found to have ASA territory infarction of the cervical spinal cord in the presence of rotatory atlantoaxial subluxation (RAAS), leading to kinking and dissection of VA with sudden cutoff at C2 foramen transversarium. The boy made dramatic spontaneous recovery with antiplatelet drugs and supportive treatment.

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Review of the literature highlights some unique features in this case report, such as reversible infarct secondary to RAAS, condition being common in adults not children, no predisposing factors such as atherosclerosis, smoking, and age, and rostral involvement of brainstem and posterior fossa being more common[4] than spinal cord involvement, as in our case.

Case Report

A 10-year-old boy presented to our emergency department with a history of progressive weakness in all four limbs. On eliciting detailed history, he revealed that 1 day before the onset of symptoms, while playing he was holding a stick in his hands and while forcefully trying to break it with his knee, he jerked his neck and had a sharp stabbing pain in his neck for few seconds and then resumed his game normally afterward. Next morning when he woke up, he felt weakness and heaviness in limbs, particularly upper limbs. His respiration was unaffected and he had grade-II power in upper limbs and grade-III power in lower limbs. Pain and touch sensations decreased below C4 dermatome, but posterior column sensations were intact. There were no signs or symptoms of brain ischemia. Bladder and bowel functions were normal. Dynamic imaging showed that there was atlantoaxial dislocation (AAD) with atlantodens interval of 5 mm in flexion (►**Fig. 1**). Noncontrast computed tomography (CT) of the cervical spine (NCCT-spine) showed atlantoaxial rotatory subluxation CT focusing right facet showing posterior rotation of C1 on C2 and focus on left facet showing anterior rotation of C2 on C1 (►**Fig. 2a–c**). Magnetic resonance imaging (MRI) C-spine showed infarction in the territory of ASA in the region extending from C1 to C4–5, depicted by hyperintense signal in the anterior half of the cord on sagittal and axial images, respectively (►**Fig. 3a** and **b**). Although right VA was dominant, CT angiography revealed left VA occlusion probably due to occlusion/spasm with cutoff at C2 foramen transversarium (►**Fig. 4a** and **b**). He was started on antiplatelet drugs (aspirin), and cervical traction was applied immediately for a period of 72 hours, after which he was put on a Philadelphia collar. Traction resulted in reduction of AAD on X-ray. In the meantime, he began to have marked clinical improvement in motor power and sensory deficits. Power improved to grade IV in all four limbs over a period of 1 week, and sensations also improved. He was advised to wear the Philadelphia collar for 3 months. At 3-month follow-up, the patient walked independently with grade-V power in all four limbs. Deep reflexes were brisk in lower limbs, and spinothalamic sensations were normal. He had normal range of motions of neck without any symptoms. Repeat MRI could not be done due to financial constraints of parents. But dynamic X-ray of craniovertebral junction (CVJ) showed no AAD (►**Fig. 5**).

Discussion

Infarction in the territory supplied by the cervical ASA occurs infrequently, especially in young people. SCI is considered a rare complication of VA dissection (VAD), and its clinical features and outcomes have not yet been well documented.[4] The ASA syndrome was first described by

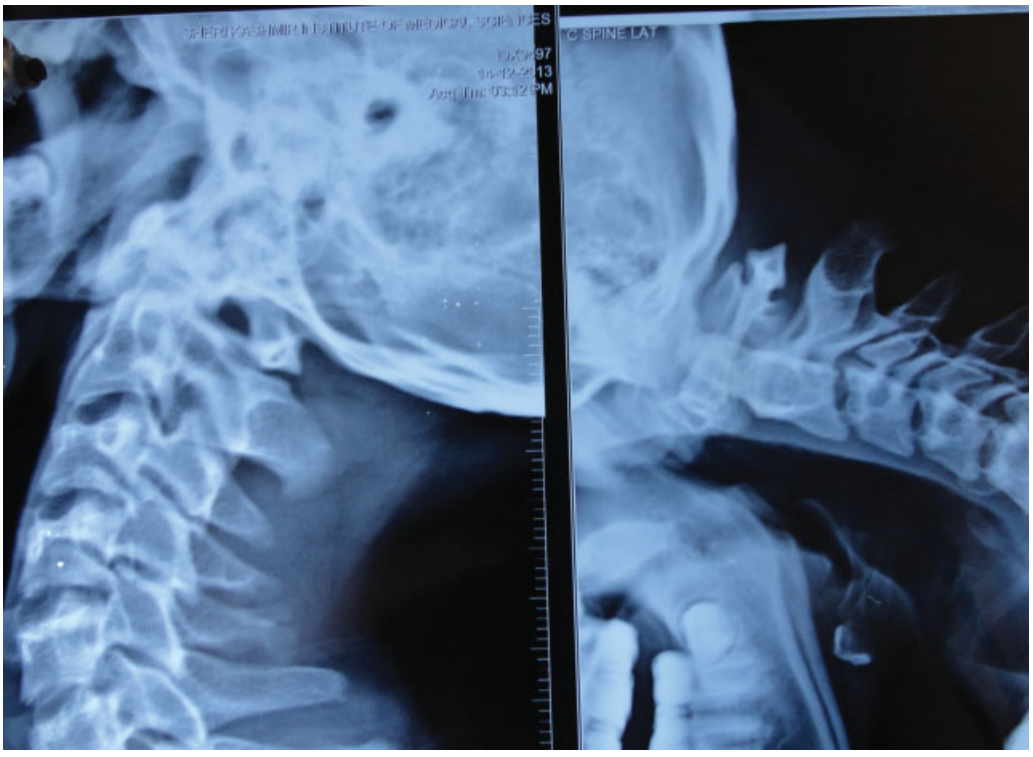

Fig. 1 Dynamic X-ray of craniovertebral junction showing mobile AAD reducing in extension.

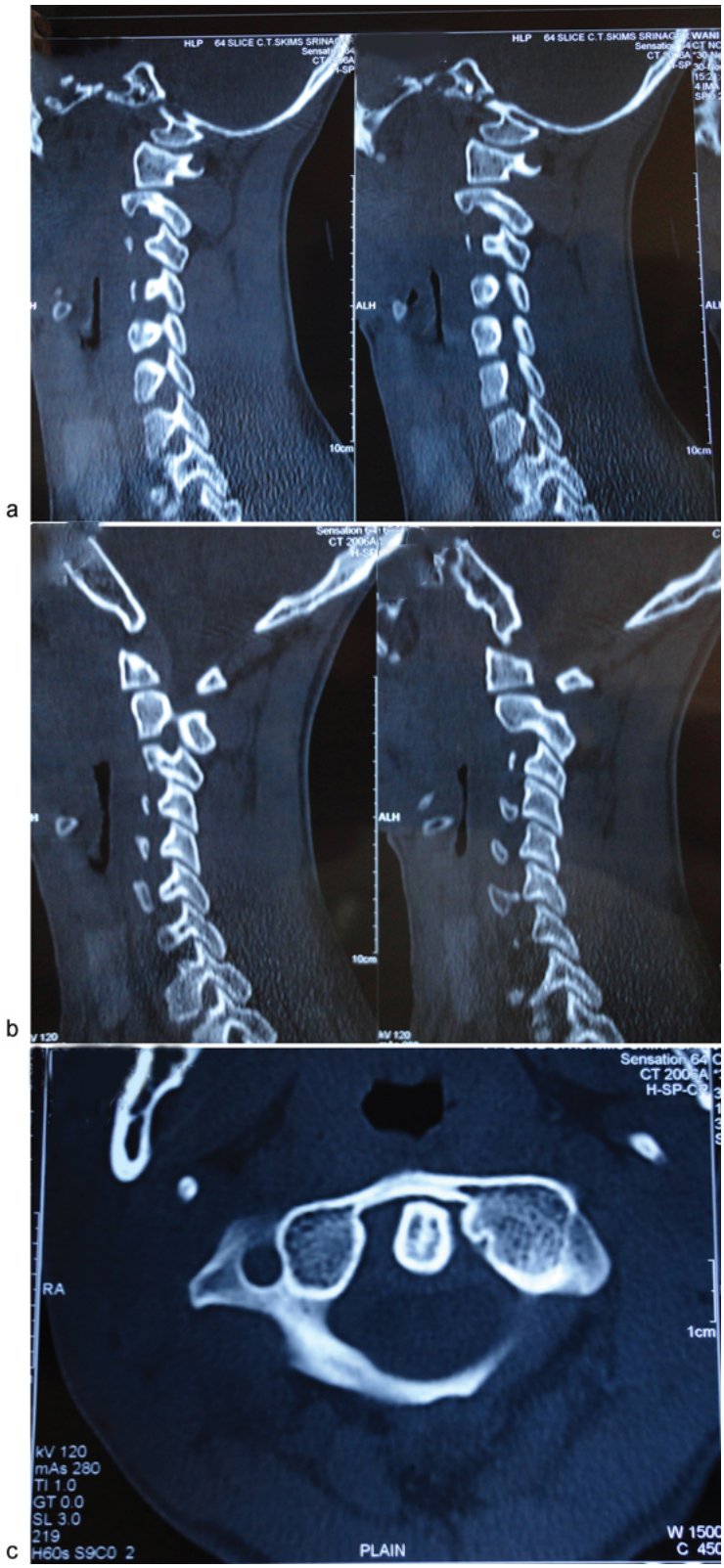

Fig. 2 (**a**) Computed tomography (CT) of right facet showing posterior rotation of C1 on C2. (**b**) CT of left facet showing anterior rotation of C2 on C1. (**c**) Axial CT showing rotation of odontoid toward left lateral mass of C1.

Spiller[5] in a patient with ASA thrombosis, and infarct was noted at autopsy in the anterior part of the spinal cord, extending from C4 to T3. In 1966, Garland et al[6] found that this syndrome could also be due to a fall in perfusion pressure or local interference with the spinal cord blood supply.[7]

The cervical cord blood supply is mainly from the VA, but it also receives branches from the occipital, deep cervical,

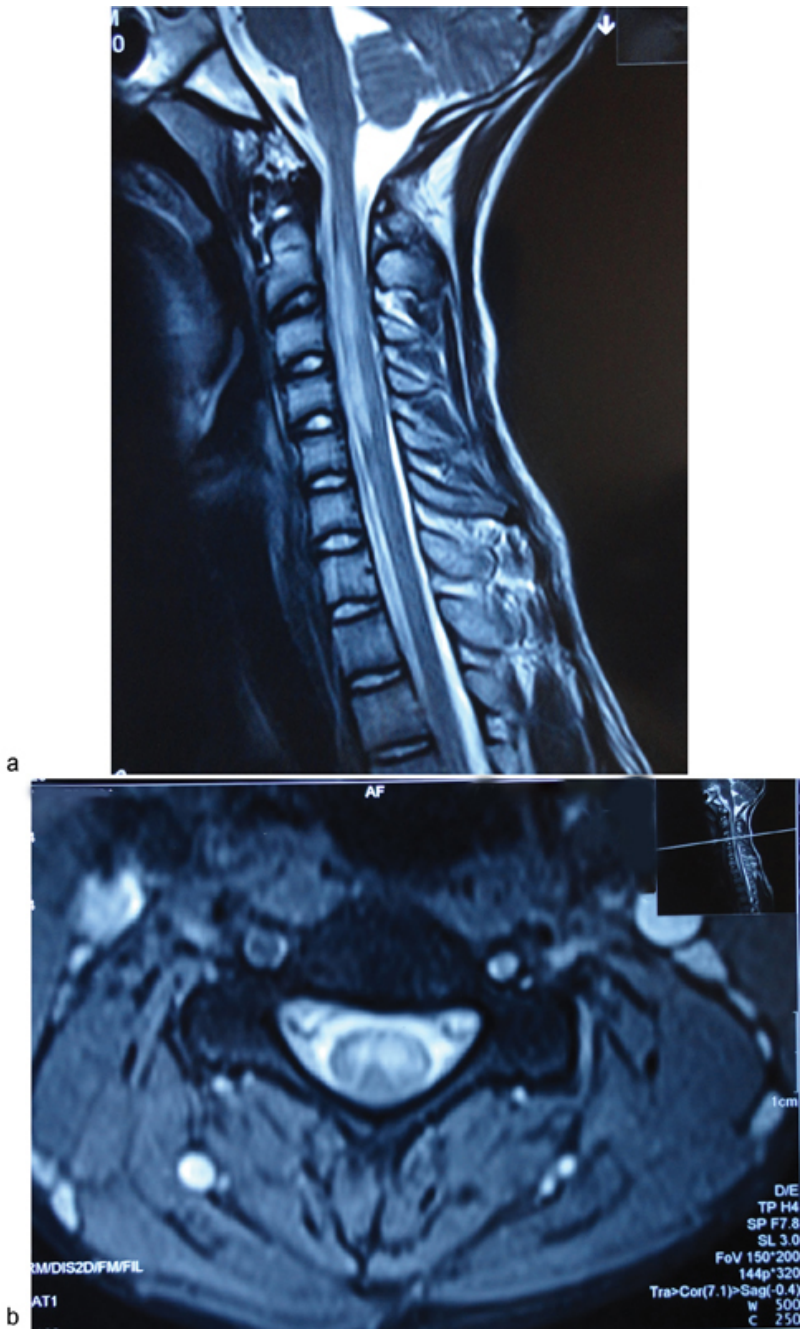

Fig. 3 (**a**) T2 sagittal magnetic resonance imaging (MRI) showing hyperintense signal in the anterior half of the cord extending from C1 to C5 with cord swelling. (**b**) T2 axial MRI showing hyperintense signal in the anterior half of the cord.

and ascending cervical arteries.[8] Lazorthes et al[8] described that the blood supply of the cervical cord can be divided into three regions: the superior cervical segments that are supplied from the intracranial VA through its anterior spinal branches, the middle cervical segments that are supplied by radicular arteries arising from the extracranial VA, and the inferior segments that are supplied by a solitary constant artery arising from one of the costocervical trunks, a branch of the subclavian artery.[8] The ASA supplies the ventral two-thirds of the spinal cord and is the major source of blood supply. ASA arises bilaterally from the vertebral arteries that unite in front of the cerebromedullary junction to form a common trunk that descends in the anterior spinal fissure. One of these vessels is larger than the other and is the dominant artery. In the cervical cord, the ASA gets collaterals from anterior radicular arteries arising from the cervical branches of the vertebral arteries and the ascending cervical arteries.[9] ASA was predominantly arising from either nondominant vertebral artery at the level of CVJ or a radicular artery from left vertebral artery at C1-C2 level, which might have been important collateral to ASA in our patient explaining the infarction.

As in adults, ischemic SCI in children is caused by decreased blood flow due to hypotension; vascular injury, compression, or

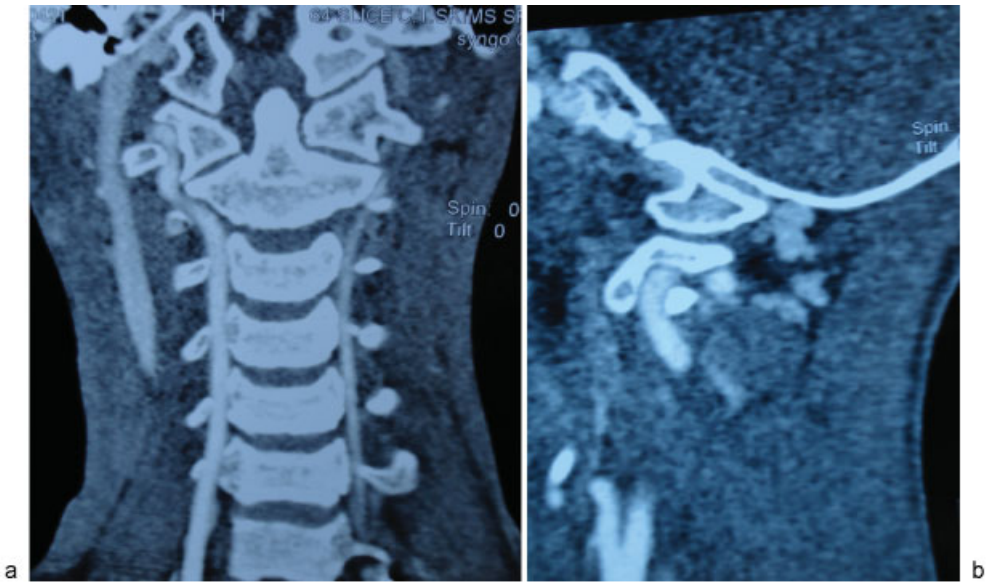

Fig. 4 (**a**) CT angiography showing cutoff of left vertebral artery at the level of C1–C2 facet joint. Also, note is made of right dominant vertebral artery. (**b**) Close-up view shows cutoff of left vertebral artery just under the facet of C2.

other impairment; or thrombosis or embolization to spinal arteries. Risk factors and underlying etiologies may differ with age in pediatric age group, young adults, and elderly population. The risk factors for pediatric ischemic SCI include obstruction of blood flow associated with cardiovascular compromise or malformation, iatrogenic or traumatic vascular injury, cerebellar herniation, thrombotic or embolic disease, infection, and vasculitis. ASA infarction has also been described in adults in association with atherosclerosis and diabetic arteriopathy,[10] vasculitides, aortic disease, anemia, sickle cell disease, polycythemia, paradoxical embolism through a patent foramen ovale,[11] atlanto-occipital dislocation,[12] cervical spondylosis,[13] cervical spinal trauma or sprain,[14] recreational drugs such as cocaine,[15] and infections such as tuberculosis, schistosomiasis, and Neisseria meningitidis.[16]

Spinal cord ischemia occurs because of one of the four proposed mechanisms for spinal cord injury without radiological abnormality.[17] Hyperextension may provoke a temporary occlusion or spasm of the vertebral arteries.[18] The pediatric spine is more elastic and therefore more deformable.[17] These findings are most prominent in the

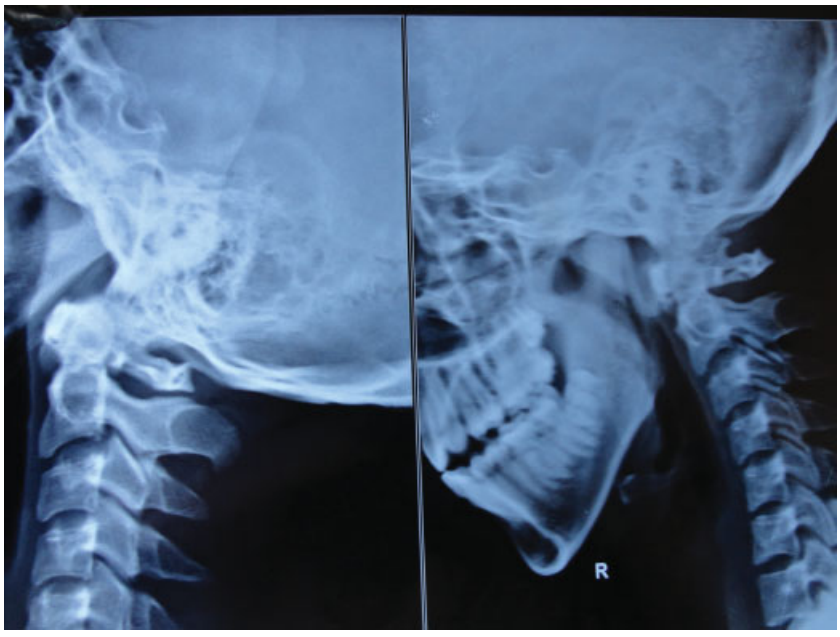

Fig. 5 Dynamic X-ray of craniovertebral junction at 3 week showing reduced AAD.

upper three to four segments of the C-spine facilitating anterior slipping between adjacent bodies with spinal injuries resulting in this region in children. Pang and Wilberger proposed that in children, the relatively less elastic spinal cord is strained inside of the more flexible spinal column during hyperflexion injuries and that this may result in reactive vasospasm of spinal cord arteries leading to ischemia.[17]

Brainstem infarctions have been reported in association with chiropractic manipulation, trivial trauma, and hyperextension of the neck.[19] Trivial trauma such as violent coughing, trampoline exercises, neck turning during a parade, driving, basketball game, dancing, swimming, and minor falls have preceded the initial symptoms of dissection.[20] Names such as "bottoms up" stroke and "beauty parlor" stroke have also been applied to this syndrome.[21] Cases are reported in the literature where trauma to CVJ causing atlantoaxial dislocation has resulted in VAD with neurologic deficits due to vertebrobasilar insufficiency.[1,19] In VAD, mechanical stretching and compression of the artery may cause intimal tearing and consecutive intramural hematoma. The VA is most susceptible to mechanical injury at its mobile distal part after leaving the transverse foramen of the axis and before entering the intracranial cavity. The anatomical relationship between the VA and its neighboring bony structures and ligaments, and the special mechanisms of head and neck movement may account for the unique vulnerability to mechanical injury at the atlantoaxial level.[22] A reduction of vertebral blood flow during rotational and hyperextensive head movements has been demonstrated angiographically.[23]

The pathophysiology of VA occlusion has been well studied. The vertebral arteries penetrate the atlanto-occipital membrane and the cervical dura, having emerged from the transverse foramina of the first cervical vertebra; it is at this level that at least 50% of rotation occurs during head turning. Angiography during head rotation has shown such VA compression at Cl associated with the development of symptoms of brainstem ischemia,[24] and even in normal patients, vertebral arterial flow may be reduced or even halted when the head is maximally turned to the opposite side. Faris et al[23] described unilateral VA occlusion during head turning in 11 of 43 asymptomatic male volunteers; however, the angiograms were not illustrated and the levels of the occlusion were not specified. With rotational obstruction of a VA, symptoms of vertebrobasilar insufficiency are unlikely to occur unless the contralateral VA is either hypoplastic or occluded.[25] Compromise of the VA at the atlantoaxial joint after neck manipulation[26] in patients with rheumatoid arthritis and atlantoaxial subluxation[26] has been described. Okawara and Nibbelink[27] and Grossmann and Davis[28] reported single cases in which unilateral occlusion of the VA occurred with rotation of the head to the opposite side. In both cases, it was believed that positional occlusion led to formation of thrombi, which became the source of intracranial emboli to the posterior circulation. Barton and Margolis[29] described two patients in whom head rotation resulted in a narrowing of the contralateral VA at the C1–C2 level. The ipsilateral atlantoaxial articulation is fixed during rotation of the head, whereas the atlas moves both downward and forward in relation to the axis on the opposite side. It has been hypothesized that stretching of the VA associated with this movement at the atlantoaxial joint may produce narrowing or occlusion of the artery.[29]

Except for few cases,[19] all the cases of VAD following trivial trauma presented with features of brainstem or posterior fossa ischemia. Our patient is among very few cases reported in the literature who presented with reversible ischemia of upper spinal cord following trivial trauma to CVJ resulting in RAAS, which responded dramatically to the reduction of dislocation with traction. In our patient, hyperextension as a result of sudden jerk as the stick suddenly broke with resulting occlusion/spasm of VA due to RAAS, as described earlier, might have caused spasm or blockage of blood flow through ASA at CVJ. Correction of RAAS with traction resulted in clinical improvement of patient. We assume that either correction of RAAS led to relief of block to VA or spontaneous resolution of spasm resulted in clinical improvement.

The clinical picture of ASA syndrome varies with the level of ischemic insult. Incidence of SCI is more in adults than in children. This might be due to age-related processes, such as atherosclerosis, affecting perfusion of the cord, with children being less vulnerable as cord has extensive collateral blood flow.[30] Due to the relatively smaller volume and cross-sectional area of the pediatric spinal cord, the ability of radiographic imaging to detect infarctions in a small cord is limited. Patients usually experience sudden local pain with subsequent onset of rapid neurologic deterioration following a "free interval" of few minutes to 2 days.[2] In our case as well, symptoms and signs appeared next morning when he woke up after a lucid interval of more than 12 hours. There is different degree of muscle weakness and dissociated sensory loss: pain sensation is decreased or absent while proprioception is relatively or completely spared. Similarly, in our patient also, dorsal column sensations were typically preserved. The loss of motor power usually corresponds to that of pain because of the anatomical proximity of the pyramidal and spinothalamic tracts in the cord. Flaccid motor paralysis and absent deep tendon reflexes may later progress to spasticity and hyperactive tendon reflexes. Few reports have linked lower motor neuron injury in upper extremities to spinal cord ischemia, supporting the notion that the anterior horn is a site vulnerable to hypoperfusion injury. Bilateral weakness of the small hand muscles and wrists lasting 24 hours has been noted as an acute complication during vertebral angiography.[31] Bladder and bowel dysfunctions are often seen with retention or incontinence of urine and feces. Our patient had significant motor weakness, grade-II power in upper limbs and grade-III power in lower limbs, which improved to grade V on follow-up. However, preserved bowel and bladder functions suggest that severity of infarction was to a lesser degree because of rapidly reversible spasm of the vessels unlike VAD seen in most of the cases in which patients had incomplete motor and sensory recovery along with bowel and bladder dysfunction due to irreversible ischemic changes in the cord.

Going through the literature, we saw that vertebral/spinal artery occlusions had variable presentations. The usual presentation of ASA infarction is an acute, painful myelopathy. Pain is a frequent, initial symptom in traumatic and spontaneous VAD.[32] In traumatic dissection, it occurs either immediately after the trauma or delayed for hours to days. Pain is characterized as sudden onset, localized in the upper part of the neck and occiput, ipsilateral to the side of dissection, dull and nonthrobbing. Pain can be severe, diffuse, radicular, or girdle-like—one reason being that extracranial cerebral arteries are known to be pain sensitive, and the pain in VAD can be caused by stimulation of nociceptors in the vessel wall. Clinically, visceral pain in arterial dissection is difficult to differentiate from pain of musculoskeletal origin, especially in patients with traumatic VAD in whom tenderness of neck muscles may also result from trauma. Hence, nuchal pain associated with VAD is difficult to diagnose as long as symptoms of ischemia are absent. It exemplifies the significance of a careful search for recent trauma in patients with VAD. Some cases (50%) may present with clinical features of Wallenberg syndrome implicating the dorsolateral medullary infarction associated with VAD.[33] Along with brainstem ischemia, involvement of thalamus and cerebral/cerebellar hemisphere is seen. Although uncommon, ischemic myelopathy is being increasingly encountered as a complication of VAD.[34] Cases presenting with features of radiculopathy and focal motor deficits are also recognized.[34] Sometimes, a partial syndrome is seen with only gray matter of the cord being affected, but sphincter control and sensory function are preserved. This entity has been increasingly recognized since MRI scanning of the spinal cord has become available. According to Hsu et al, neck pain or headache are warning symptoms of VAD, and the presence of a concomitant sensory level or Brown-Séquard syndrome is useful for the timely diagnosis of cord infarction following VAD.[4] Michael[35] concluded that bilateral neurodeficits are more common but unilateral ASA deficits are also frequently reported. This happens either because of occlusion of a unilateral sulcal artery or because incomplete collateralization with the posterior spinal artery maintains perfusion on one side of the cord.[35]

Technically inadequate radiographic series of the C-spine may miss the diagnosis of a C-spine injury. Views required include lateral dynamic, posteroanterior, lateral, and odontoid. MRI is a sensitive modality in the evaluation of the spinal cord for infarction. Before MRI, the diagnosis of SCI relied on autopsy or documented vascular occlusion by angiography. MRI rules out extradural compression, vascular malformations, and space-occupying lesions. The differential diagnosis includes infectious or parainfectious myelitis, multiple sclerosis, and vasculitis. In inflammatory or demyelinating lesions, it is well known that T2-weighted images show lesions some time before the clinical onset, whereas with ischemic lesions a normal or almost normal MRI is usually seen in the first few hours. The sudden onset of clinical symptoms after a rapid movement of the neck is very suggestive of infarction, but not of myelitis. In the acute phase after infarction, the diameter of the spinal cord remains normal and diagnosis is based on signal abnormalities on the long time-to-relaxation sequences, which reflect the presence of cytotoxic edema. However, in the subacute phase, with the appearance of extensive vasogenic edema, the high signal abnormalities are more evident and associated with cord swelling. In our case, the anterior location and the MRI changes, together with the sudden onset and rapid development of typical clinical symptoms, helped us in differentiating spine infarction from other spinal cord lesions such as myelitis, multiple sclerosis, and neoplastic conditions. Diffusion-weighted imaging, although not commonly used on the spinal cord, now allows identification of cord ischemia within hours of the insult.[36] The limitations of spinal cord MRI include increased artifact due to cord movement and cerebrospinal fluid flow.[37]

The clinical contribution of angiography in cases of anterior spinal infarction with an obvious traumatic event and in which a high-quality C-spine MRI has ruled out spinal vascular malformations and vascular dissection is thought to be limited and is often not considered mandatory. MR angiography (MRA), a noninvasive technique, is rapidly replacing conventional angiography as the gold standard in the diagnosis of dissections, with the advantage of showing the intramural flap and hematoma.[38] Usual abnormalities observed are poor signals across the vessel, irregularity of vessel wall, aneurismal dilatation, intimal flap, intramural hematoma, or complete drop of signal beyond a level suggesting occlusion. Standard catheter angiography still remains the gold standard for diagnosis, as it allows excellent characterization of the dissection.[30,31] The usual features observed are irregularity and/or stenosis of the vessel, “string sign” (arising as a result of a dissection that extends circumferentially around the lumen over a long segment), “double lumen,” pseudoaneurysm formation, or complete occlusion.[39] Stenosis is by far the commonest finding, being caused by a subintimal hematoma.[39]

Given the rarity of the diagnosis, there is no consensus for any particular treatment approaches. Management of ASA syndrome in children is supportive, ideally with intensive multidisciplinary rehabilitation in a spinal unit.[18] There is limited evidence for the acute use of steroids (methylprednisolone) in traumatic spinal cord injury[39]; however, there is no evidence for their use in ischemic SCI. Anticoagulation with heparin, coumadins, and aspirin to restrict thrombus progression and thromboembolism appeared to improve outcome in patients with SCI and thrombotic disorders.[40] Duration ranged from 5 to 35 days. In the case of embolism, initial anticoagulant therapy seems very reasonable. If dissection includes the intracranial VA segment, subarachnoid hemorrhage should be first excluded before anticoagulants are administered. Repeat angiography 3 months after initiation of anticoagulant therapy to decide on further therapy is recommended. Anticoagulants are discontinued if recanalization occurs and the arterial lumen appears smooth. If the vessel remains occluded, continue anticoagulants for as long as 6 months followed by antiplatelet drugs for 2 years, but further study on the natural history of these lesions is urgently needed.[33] Endovascular treatment is tried in centers equipped with such facilities. For

dissections, Continuance of anticoagulation therapy may be guided by MRA or ultrasound. Persisting irregularity or stenosis at third month needs continuation of therapy for another 3 months. Beyond 6 months, shift the patient to antiplatelet agents.[41]

Supportive care and rehabilitative physical therapy are the key to long-term treatment of children with SCI. Our patient was managed conservatively with cervical traction and antiplatelet drugs followed by rigid Philadelphia collar application till patient had achieved complete recovery.

Dreadful complications of SCI include ventilator dependence and decreased bowel/bladder function, according to areas of SCI. Patients are more susceptible to respiratory infections and bladder infections. Psychological problems, chronic pain, and spastic deformities are reported complications of cord infarction.[42]

Studies showed that patients with some preservation of motor function or pain sensation below the level of lesion progress better than those without these two functions.[43] ASA recovery is better if the spinal cord damage is incomplete. The sparing of pain sensation is associated with a higher incidence of useful motor recovery. This may be explained by the anatomical proximity of the spinothalamic and corticospinal tracts in the lateral columns and it ensures that parts of the corticospinal tracts that lie posterior to the spinothalamic tracts in the lateral funiculi may have been spared.

Overall prognosis of this clinical entity is encouraging and factors such as severity of ischemic insult and collateral flow affect the outcome.[44] In the literature, 75% of patients make excellent recovery and the overall death rate is less than 5%. Almost 90% of the stenosis resolve and two-thirds of the occlusions are recanalized.[44] Maximal improvement occurs in the first 3 months following a dissection.[44] Outcomes after SCI have been extensively reported in adults than in children. A review of outcomes in 199 patients by Salvador de la Barrera et al showed 22% deaths, 24% were unimproved, 35% showed some improvement, and 19% cases markedly improved.[45] This review included few children, but results with respect to age, cause, or spinal cord region involved were lacking. Two series on adults with cord infarction established that outcome was poorer in patients who had significant disability at onset.[45] Robertson et al in a study of 117 spinal infarction patients concluded that slow but sure improvement is not uncommon after SCI and it may continue long after discharge. While severe neurodeficits at nadir is the strongest predictor of poor final outcome, significant recovery is also possible in a minority of these patients.[32]

We believe that early detection, correction of RAAS with traction, and vigorous pharmaco- and physiotherapy was the reason for reversal of ischemic insult to cord. Eventually, our patient had an excellent recovery following precise management.

Conclusion

Although this condition is not very uncommon in adults, there are few reports in the literature describing etiology, diagnosis, treatment options, and prognosis of ASA infarction following RAAS in pediatric age group. Diagnosis of this condition is difficult and treatment options are limited. However, if patients are timely diagnosed with appropriate investigations and properly treated with immobilization, anticoagulants, and physiotherapy, they can be the key to an excellent outcome.

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