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Case Report

Acute Spinal Cord Infarction After Neck Exercise in a Young Child: The Need for DWI and ADC Maps in Spinal Cord Imaging

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Abstract

Spinal cord infarction is rarely seen in children. They could present clinically with either anterior or posterior cord syndrome. Apart from clinical features, MRI is an important modality to investigate the same. However, usually, only T1, T2 and FLAIR imaging is done for the cord. In this case report, we tend to prove that DWI and ADC maps are important modalities of spinal cord imaging. Using these sequences in routine could help in accurate diagnoses of the cause of myelopathy.

Keywords: Acute Spinal Cord; Neck; Child; Spinal Cord

Introduction

Spinal cord infarction (SCI) is a well known entity however, it is still a rare phenomenon in the paediatric age group. This disease is debilitating and can cause major morbidity affecting the quality of life. The signs and symptoms varies in accordance with the spinal artery territory involved. In case of anterior spinal artery involvement, there is spinothalamic tract and the corticospinal tract involvement with sparing of the posterior column and vice versa in the case of posterior spinal artery involvement. However, several atypical SCI presentations do not fit the anatomically defined spinal blood distribution [1,2]. Magnetic resonance imaging (MRI) is an essential imaging modality to rule out misdiagnoses of SCI such as compressive myelopathy. However, in usual scenario, only T1 and T2 images are performed for spinal cord examination. Our case describes the importance of DWI and ADC in spinal cord imaging and thus in preventing misdiagnosis.

Case Report

Our patient is a 13-year-old girl child, born out of a non-consangious marriage and had no co-morbidities. She went for her normal play in the park one evening and did arm rotation and overhead exercises at the local park gym. After coming home, she started to have burning sensation in the left half of the body and the neck. After a few minutes, she started to have weakness in the right half of the body which was sudden in onset and progressed rapidly and she became quadriparetic. When she arrived in the ER, she was hemodynamically stable. Her general physical examination was normal. Since she had predominantly right hemiparesis with only subtle weakness of the left side, she was initially thought to be a case of left MCA stroke in the ER. However, a neurology consult was done. On detailed neurological examination, she had flaccid qudreparesis (weakness of right half was more than the left). Power in right upper limb and lower limb was grade 0 and in left upper limb

and lower limb was grade 3. She had loss of pain and temperature below T1. Her joint position and vibration was normal. The cranial nerve examination was normal. On the basis of clinical history and examination, a lesion in the cervical cord was predicted sparing the posterior column. The chid was send for immediate MRI. The routine imaging demonstrated subtle hyper intensity in the C3-C5 region which was non enhancing. On discussion with the radiologist and on the basis of clinical history, it was decided to do the DWI and ADC maps for this patient. The imaging had diffusion restriction along with reduced values in the ADC maps in the said regions. This clinched the diagnosis that the child's symptoms were due to an acute spinal cord infarction. Her MRI angiography of neck vessels along with arch of aorta was also done which was normal. She was planned for DSA of the spinal artery but was refused by the relatives due to affordability issues. She was then managed conservatively and symptomatically. Unfortunately, she did not improved and was discharged in a stable condition in a quadreparetic state. 3 weeks after the onset repeat imaging was done which was suggestive of a sub-acute infarct in the form of subtle contrast enhancement. This case strengthens the need for spinal cord DWI imaging in cases where spinal cord infarction is suspected and also routinely which will help in clinching the diagnosis and would avoid any unnecessary investigations.

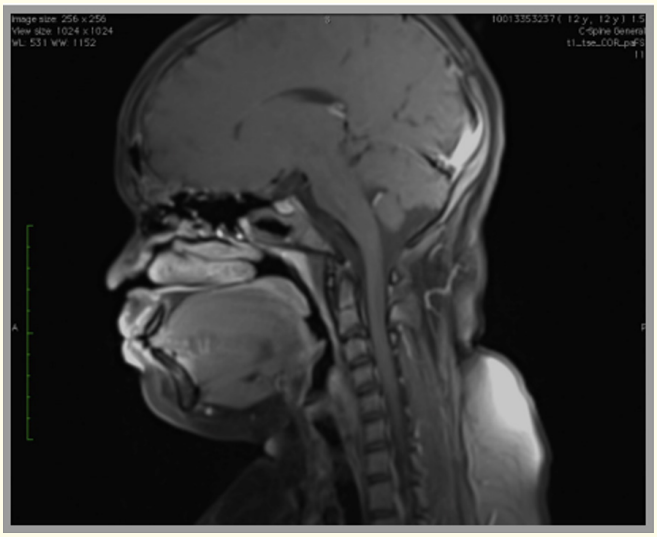

Figure 1: T1 images (normal without enhancement).

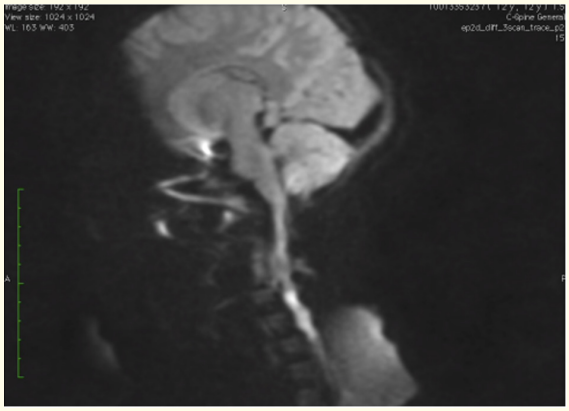

Figure 2: DWI images showing restriction in C3-C5.

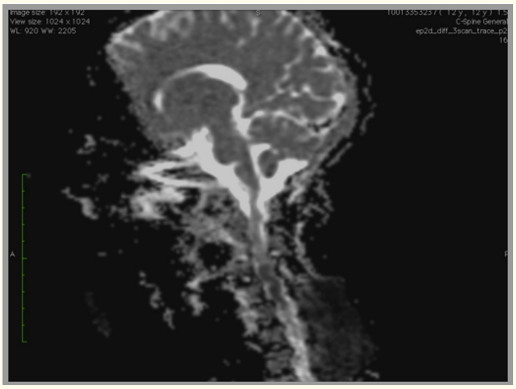

Figure 3: ADC maps showing reduced value in C3-C5.

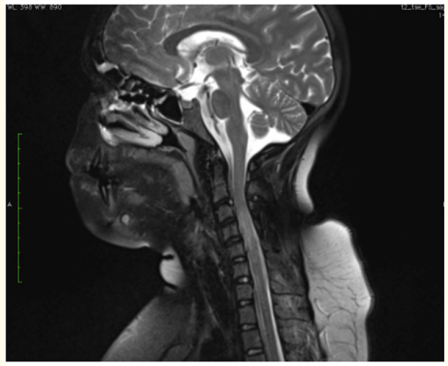

Figure 4: T2 images showing hyper intensities in C3-C5.

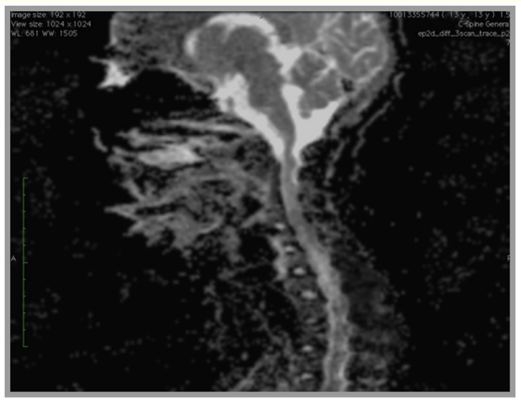

Figure 5: Interval T1 showing patchy enhancement.

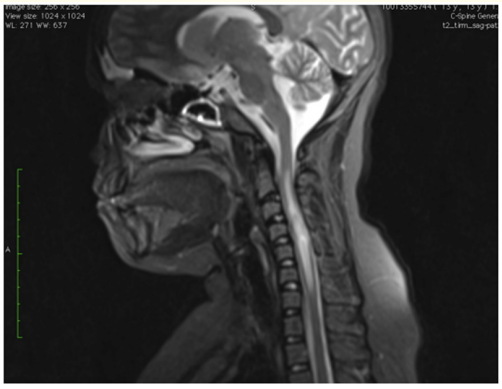

Figure 6: Interval DWI showing mild DWI resolution.

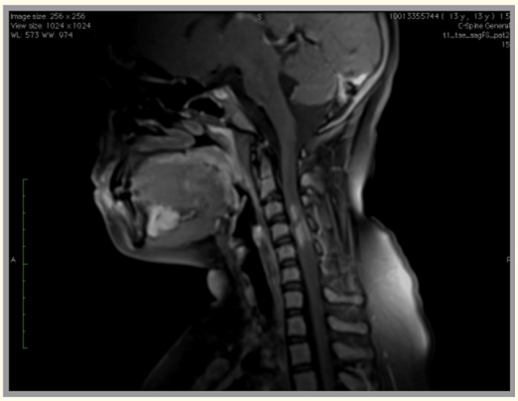

Figure 7: Interval ADC showing normal values.

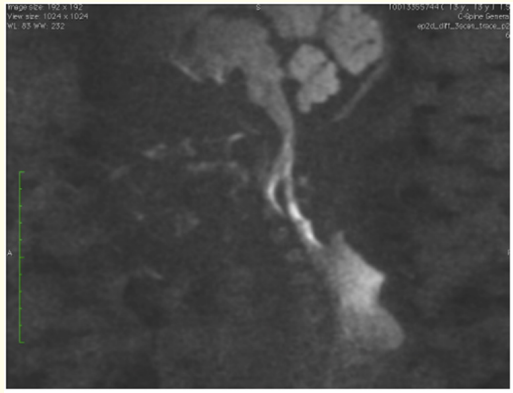

Figure 8: Interval T2 images showing extensive hyper intensity.

Discussion

SCI is a rare entity with varied presentations including some atypical presentations like in our case where however, the patient was quadreparetic, she had right sided weakness more than the left side. It is important to quickly recognise this syndrome and notify the radiology team so that all the sequences of MRI can be done and the delay in diagnosis is avoided. Common etiologies of SCI include prolonged arterial hypotension, cardioembolic disease, tumor embolism, dissection, and systemic arteriopathy. An increasingly recognized etiology is fibrocartilaginous embolization (FCE) [3,4] In adults, the commonest cause remains post aortic surgery hypotension however in the paediatric age group, FCE is being increasingly recognised as a cause. Sudden quadriplegia is the most important feature of SCI. The differential diagnosis of this syndrome can include transverse myelitis, demyelination and also GBS as during the phase of spinal shock, there may be areflexia. In our case, there was a sensory level and dermatomal distribution of sensory loss which lead us away from this diagnosis. A combination of DWI with ADC maps must be done to distinguish SCI from myelitis and demyelination [5,6]. SCI usually appears hyperintense on T2-weighted MRI, predominantly in the grey matter, and DWI of our patient also identified a hyperintense signal in the areas of T2 signal abnormalities with a decreased ADC value. On conventional MRI, inflammatory and ischemic spinal cord lesions may both show T2 hyperintensity; however, with gadolinium, acute infarction will not enhance which was the case in our patient. Therefore, when clinical suspicion for

infarct is high neurologists should specifically ask for a spinal MRI with DWI to confirm the diagnosis.

In conclusion, the initial clinical presentation of SCI can be variable and nonspecific and may not pertain to a single vascular territory, the abrupt onset of weakness and sensory symptoms should be considered as SCI until proven otherwise. An early diagnosis of SCI is important, and MRI with DWI of the spine should be done as soon as possible to ensure appropriate diagnosis and management.

Conclusion

The initial clinical presentation of SCI could pertain to any vascular territory. High index of suspicion must be kept in any patient presenting with acute onset motor or sensory symptoms and SCI must be kept as a differential. We recommend to proceed with DWI and ADC maps of spinal cord in MRI for appropriate and fast diagnosis.

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