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Stroke in Young - An Entity that is Under - Recognised

Dr Navneet Kaur[1], Dr Paresh Sukhani[2], Dr Hemant Kumar Mishra[3]

Mahatma Gandhi University of Medical Sciences and Technology

Abstract: ***Cranio-cervical artery dissection is a potentially disabling yet underrecognized entity and often occurs in young and middle-aged adults.Accurate and prompt diagnosis of this condition is crucial because timely and appropriate therapy can significantly reduce the risk of stroke and long term sequelae. Here we present a case of 8-year-old child who presented to us 15 days after the onset of symptoms.***

1. Introduction

Stroke occurs infrequently in young adults as compared to older adults, even the causes of stroke in young adults differ substantially from those in older adults. Imaging plays a crucial role in finding a specific cause of the disease. Risk factorssuch as cigarette smoking, diabetes mellitus and hypertension are important in adult stroke. Atherosclerotic disease and dyslipidaemia are again uncommon in stroke in young (2).

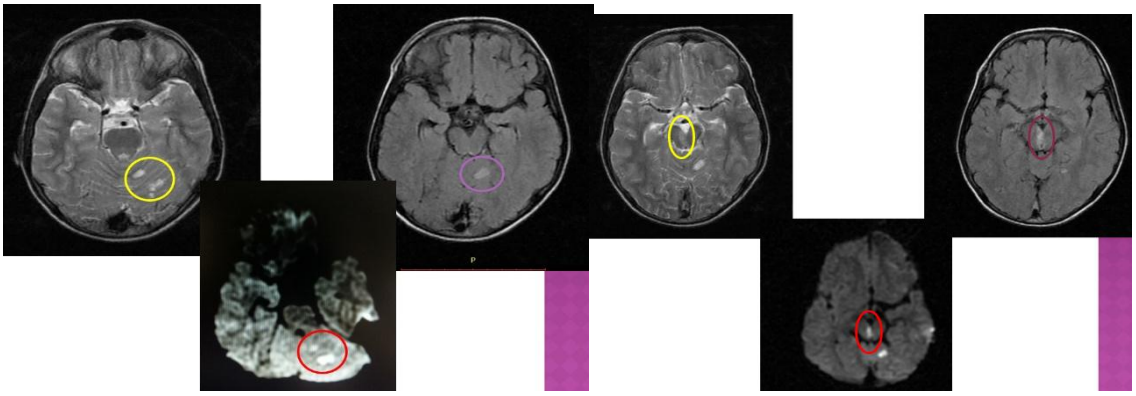
In young and middle-aged patients, spontaneous cranio-cervical artery dissection is the cause of up to one-fourth of strokes, with a peak prevalence in the fifth decade of life(3). However recent data show that cervical artery dissection is a possible cause of ischemia in the elderly and should be considered in diagnostic investigations in this patient group also.(4) Other causes of paediatric stroke incudes vasculopathies, arterial dissections, fibromuscular dysplasia, Moya Moya disease, haematological disorders like sickle cell disease, coagulation disorders, congenital metabolism errors such as Fabrydisease, homocystinuria, ornithine trans-carbamylase deficiency, substance abuse, cerebral sino-venous thrombosis. About half the surviving patient of paediatric stroke develop some neurologic or cognitive impairment and epilepsy. So timely diagnosis is important to prevent lifetime complications.

2. Case Summary

We hereby present a case of 8-year-old child presenting with symptoms related to posterior circulation stroke. 8-year-old male child, right handed presented to us with chief complaints of occipital headache, nuchal pain for 15 days and acute onset weakness of left upper and lower limb for 7 days. He developed imbalance while walking with tendency to fall towards left side and drooping of right eyelid with double vision when looking with both eyes for 7 days. He was taken to private hospital where NCCT head was done which was normal and patient was sent back on conservative treatment. He did not show any improvement following which he had multiple episodes of vomiting with headache and nuchal pain. This time headache was severe in intensity. He was again taken to hospital and as he deteriorated he was referred to higher centre. He was brought to our hospital. There was no history of LOC or any history suggestive ofseizure, rheumatic fever, valvular heart disease, DMT2/HTN, fever, malaise, myalgias, arthritis, weight loss, exposure to STD or TB and any drug addiction. On general examination he was hemodynamically stable. No significant difference was seen in blood pressure in both arms.There was Right eye ptosis. His right eye medial, inferior, superior and bilateral lateral rectus were impaired. Fine nystagmus was present in both eyes on looking towards left side.

Diplopia was present in all gazes. Other cranial nerves were normal. Cerebellar signs were positive. Blood investigations revealed normal serum homocysteine levels. 2D ECHO was normal ruling out cardiac source of thromboembolism. HB electrophoresis was normal ruling out sickle cell anaemia or other hemoglobinopathies. Work up done for connective tissue disorders was negative. MRI brain was ordered and it revealed multiple area of acute infarct in left cerebellar hemisphere, right thalamus, medial aspect of midbrain predominantly on right side. Posterior territory was involved supplied by left superior cerebellar artery, right thalamo-perforating and perforating branches of basilar artery. CT angiography of head and neck vessels was done and was suggestive of intimal flap in V4 segment along with presence of mural thrombus in V3 and V2 segment of left vertebral artery. For confirmation MRI T1 fat sat images were taken at neck level and was suggestive of slow flow in true lumen of involved segment along with intramural thrombus in false lumen of left vertebral artery.

Patient was managed with antiplatelets and anticoagulants.



MRI brain axial images suggesting areas of T2 and FLAIR hyperintensities (yellow and purple circles) showing diffusion restriction(red circles) in left cerebellar hemisphere, superior vermis and medial aspect of mid brain predominantly on right side.

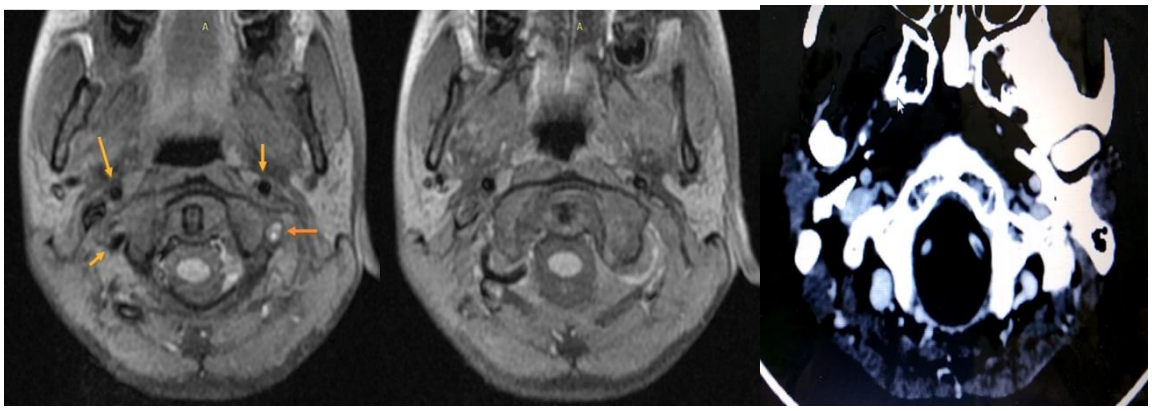


Figure 2: (a and b)MRI neck revealing crescent shaped area of hyperintensity suggestive of mural thrombus around the true lumen. Both carotid arteries and right vertebral artery shows normal flow void. Hyperintensity in the true lumen is suggestive of slow flow. (c) CT angiography image showing us the linear hypointense area in left vertebral artery suggestive of intimal flap.

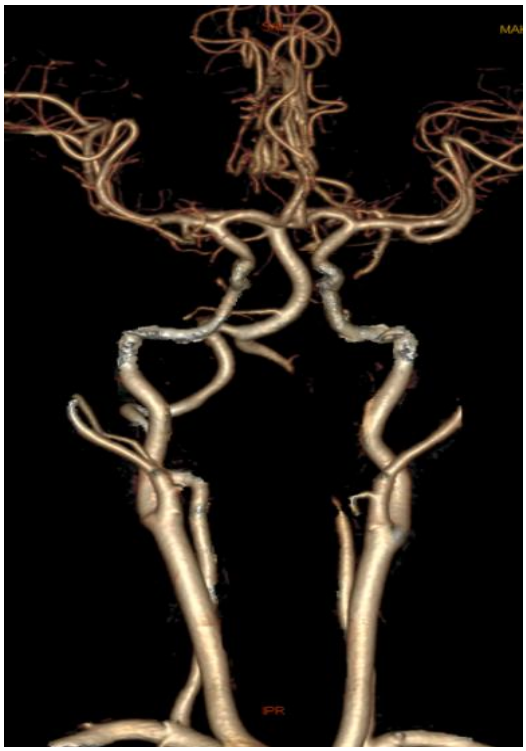


Figure 3: Coronal MIP reconstruction showing abrupt tapering at the V2 segment of left vertebral artery.

3. Discussion

Cranio-cervical artery dissection is rare, with an estimated annual incidence of 1-5 cases per 1,00,000 in the general population(1,5,8). And these causes only 0.4-2.5% of all strokes in the general population but 5-20% of strokes in young patients (2). Vertebral artery dissection can be spontaneous or traumatic in origin.Spontaneous VA artery dissection most commonly occurs in the extra-dural VA although combined intradural and extradural dissections are also seen (2,7). Dissections are mostly located in the pars transversaria segment (V2)- 35% or in the atlas loop segment (V3)- 34% (5). Severe stenosis or occlusion of the artery may result in brainstem or cerebellar ischemia. An intraluminal clot thus formed may can break off and disseminate leading to ischemic stroke(6).It is important to note that, in addition to the identification of the dissections, the next most important feature is to assess whether or not the dissection involves the intradural portion of the vertebral artery(V4) and thus the origin of PICA (9). A treatment plan for the dissection depends entirely on the clinical and imaging features. Presence of subarachnoid haemorrhage alters the treatment plan. Involvement of V4 segment of vertebral artery usually presents with sub-arachnoid haemorrhage(SAH) although was not seen in our case.

Accurate and prompt diagnosis of this condition is crucial because timely and appropriate therapy can significantly reduce the risk of stroke and longterm sequelae. Opposingly in our case patient presented to us after 15 days of onset of neck pain and 7 days of development of stroke like symptoms.Patient was put on anticoagulants as no SAH was seen. He improved symptomatically and was discharged.

4. Conclusion

Spontaneous vertebral arterial dissection though under recognised is one of the important etiology to be considered while dealing the young patients with stroke. Presence of nuchal and occipital pain should be considered an important symptom as dissection usually present along with pain. Accurate and prompt diagnosis of this condition is crucial because timely and appropriate therapy can significantly reduce the risk of stroke and long term sequalae.

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Author Profile

Dr Navneet Kaur – PG 3rd year resident, MGMCH Jaipur Rajasthan

Dr Paresh Sukhani Associate Professor, MGMCH Jaipur Rajasthan

Dr Hemant Kumar Mishra Professor and HOD , MGMCH Jaipur Rajasthan