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CASE REPORT

An isolated infarction of cerebellar nodulus (lobule X)

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ABSTRACT

Cerebellar nodulus (lobule X) is an anterior and inferior structure of the vermis and is the epicenter of flocculonodular lobe (vestibulocerebellum). Nodulus coordinates with the vestibular nuclei through afferent Mossy and efferent Purkinje fibers and plays a pivotal role in maintaining balance and is responsible for vestibulo-ocular reflex. Any lesion disrupting these fiber tracts impairs the person's ability to control ocular movements during head tilting and posture while standing and walking. Isolated infarction of nodulus is extremely rare with only few cases been reported earlier. In this article, the authors present a case of an elderly female with severe giddiness and cerebellar signs and were diagnosed to have an isolated cerebellar nodulus infarction on imaging studies. Conservative management to which our patient responded well is also discussed.

Key words: Cerebellar vermis, medial posterior inferior cerebellar artery infarction, nodulus, nystagmus, vestibulocerebellum

Introduction

Nodule (lobule X) is a median anteroinferiorly located, phylogenetically most primitive structure in the cerebellum. Along with the flocculus, the paraflocculus and the pedicles, it forms the flocculonodular lobe (archicerebellum or vestibulocerebellum) [1-5]. Cerebellar nodulus and ventral uvula (lobules X and IXc, d of the vermis) has direct reciprocal connections with the vestibular nuclei and direct vestibular afferent inputs as Mossy fibers and thus play a functional role for vestibular processing [6]. Neurons located in the flocculus and the paraflocculus execute vestibulo-ocular reflex and discharge in relation to different stimuli (head movements or visual target movements), including but not limited to, the position of the eyes in the orbit during fixation and the eye movements during smooth pursuit, vergence and vestibular responses (thus maintaining the balance) [5,7-10]. We herein report a rare case of an isolated cerebellar nodulus infarction in an elderly female who presented with severe giddiness, unsteadiness of gait and bidirectional gaze evoked nystagmus.

Case Report

The present case report is about a 61-year-old right handed female, a known hypertensive, who presented with sudden onset of giddiness associated with vomiting. She also complained of postural imbalance on standing and was experiencing difficulty during walking. Her symptoms were continuous throughout the day. There was no history of fall, loss of consciousness, sensory disturbances, ear discharge, hearing impairment or tinnitus. There was no history of dysphagia, dysarthria or diplopia. Patient was referred to the neurology department with above complaints. On presentation, patient was conscious, coherent and afebrile with stable vitals. Higher mental functions, cranial nerve examination, motor power, tone, deep tendon reflexes were normal. All cerebellar function tests except for tandem walking test were normal. We noticed broad based gait and sway to the right when she was performing tandem walk. Ocular examination showed bidirectional horizontal gaze evoked nystagmus. Head impulse test showed no delay in ocular saccade, Dix-Hallpike test showed continuous nystagmus with no latency, adaptability, fatigability and there was no suppression of nystagmus with visual fixation. Routine otological examination lateralized the cause to the vestibular system. Based on the clinical history and examination, possibility of central cause vertigo was thought and magnetic resonance imaging (MRI) was advised. MRI brain was performed on GE® HDXT 750W 3 Tesla MRI with spin echo, gradient and diffusion weighted sequences. MRI showed a well-defined T1 hypointense, T2 and fluid-attenuated inversion recovery hyperintense lesion showing diffusion restriction involving nodulus of the cerebellum [Figures 1 and 2]. Contrast MRI showed no lesions [Figure 3]. MR angiogram [Figure 4] showed normal medial posterior inferior cerebellar artery (mPICA) arteries bilaterally. Diagnosis of an isolated infarct of cerebellar nodulus was confirmed and investigations to identify the embolic cause for any ischemic events were negative. While the carotid artery showed a mild atherosclerotic disease (intimo-medial thickening), the bilateral vertebral arteries were normal. Following treatment with oral anti-platelet agent (tablet 100 mg acetylsalicylic acid once daily for 1 week), the patient's condition improved and she was discharged on the 7th day

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CASE REPORT

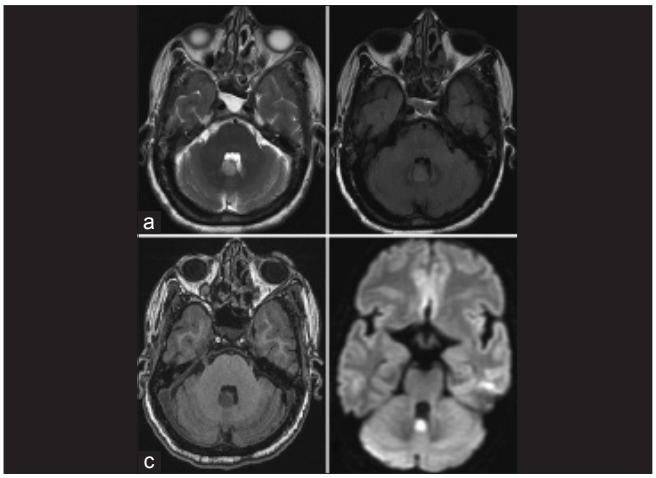

Figure 1 Axial T2 weighted (a), axial fluid-attenuated inversion recovery images (b) showing hyperintense lesion involving nodule of cerebellum, on T1 spoiled gradient sequence lesion is hypointense (c), on diffusion sequence lesion is showing restriction (d)

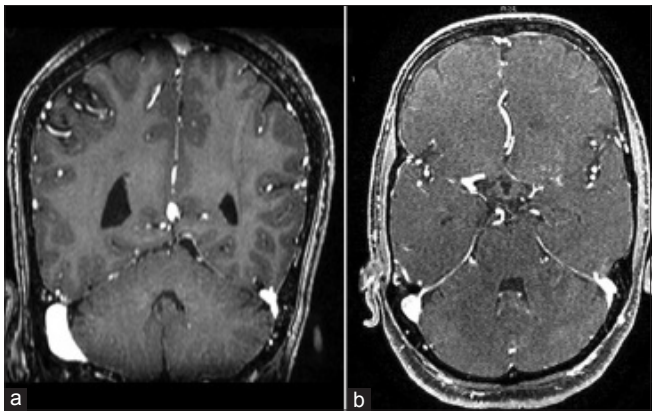

Figure 3 Magnetic resonance angiogram (a) and contrast enhanced T1 spoiled gradient recalled echo images (b) showing non-enhancing hypointense lesion in cerebellar nodule

following admission, on the same oral anti-coagulant for a period of 6 weeks. At the time discharge, patient improved significantly, with her vertigo decreased by 75% and she was able to walk with support. At 3 months of follow-up, her vertigo was relieved completely and she was able to walk without support.

Discussion

An isolated cerebellar nodulus infarction is a rare but potentially treatable variant of cerebrovascular disease. Nodulus is an anterior inferior structure of the vermis and is the epicenter of the flocculonodular lobe. It is sandwiched between inferior medullary velum (anteriorly) and uvula (posteriorly) [1,3,4,8]. Nodulus co-ordinates with the vestibular nuclei through afferent Mossy and efferent Purkinje fibers. Any lesion disrupting these fiber tracts impairs the person's ability to control ocular movements during head

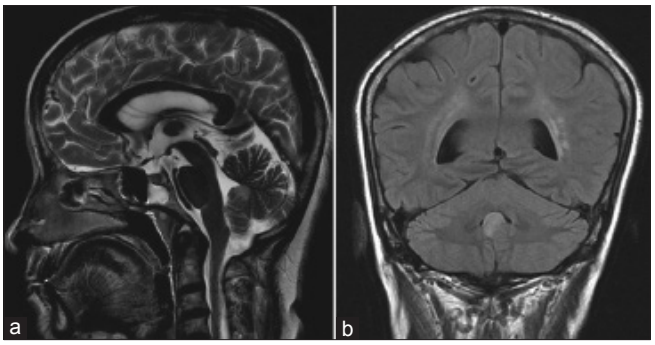

Figure 2 Sagittal T2 weighted (a) and coronal fluid-attenuated inversion recovery images (b) showing hyperintense lesion in nodule

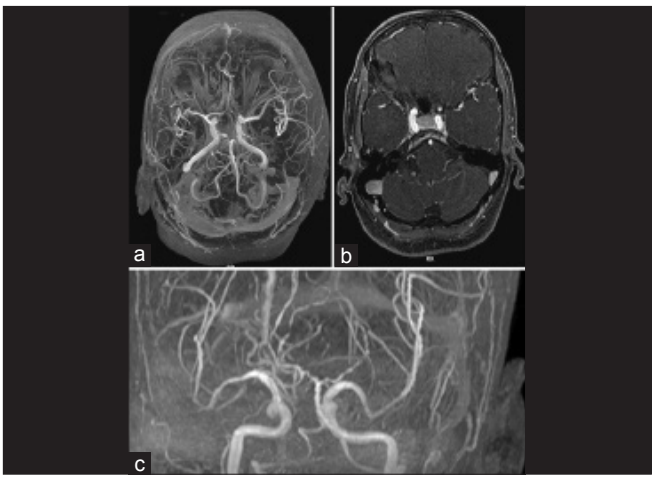

Figure 4 Magnetic resonance contrast angiography 3D maximum intensity projection image showing normal vertebral arteries and circle of Willis (a and c), source image (b) showing normal medial posterior inferior cerebellar artery

tilting and posture and also while standing and walking. In an attempt to attain steadiness, such patients adapt to a wide based stance and gait [4,7-9,11,12]. The medial branch of the PICA supplies the nodulus along with the uvula and the medial inferior vermis [3,4,10,13,14]. The mPICA infarcts involving vestibulocerebellum are often clinically misdiagnosed as peripheral vertigo syndromes [2-4,14]. Dix-Hallpike test can differentiate between central nystagmus versus peripheral nystagmus as earlier lacks latency, fatigability, adaptability, reproducibility. Whenever, there is no decrease in the amplitude of the nystagmus on visual fixation, it is said to be of central origin. Isolated nodular lesion or ischemic lesion in the mPICA involving nodulus should be suspected in all elderly patients with isolated prolonged sudden onset severe vertigo in whom head impulse test is negative. It should also be suspected in all cases of vertigo having vascular risk factors and when severe vertigo is associated with bidirectional gaze evoked nystagmus [10,15]. Our patient, though did not have any vascular risk factors, she had severe prolonged vertigo without paroxysmal changes and head impulse test was negative. Acute central vertigos are commonly secondary to vascular etiology,

with MRI diffusion weighted imaging only being helpful in such cases [13,16]. Management of the mPICA infarcts is similar to any other stroke; however based on the severity of vertigo, labyrinthine sedatives can be added. Small isolated nodular infarcts have a benign course with complete recovery; however larger mPICA infarcts may cause obstruction to the fourth ventricle by mass effect resulting in obstructive hydrocephalus [10,14,16].

Conclusion

In summary, isolated infarction of inferior vermian nodulus occurs following an atypical occlusion of the medial PICA and can present with pseudo-labrythine symptoms. A high index of clinical suspicion is essential to identify these cases as it can often be mistaken for other common labyrinthine disorders. Perhaps the management of this condition requires a different management approach with only the use of an anti-platelet agent rendering relief of the symptoms with subsequent improvement in the disease condition in most cases. Basic clinical examination, i.e. head impulse test and Dix-Hallpikes test can help differentiate cerebellar cause of vertigo from other inner ear causes, however the MRI with magnetic resonance angiography and diffusion sequences are highly sensitive in diagnosing these small cerebellar infarctions.

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Authors' Contributions

All authors participated in clinical diagnosis, drafting the manuscript, sequence alignment, writing discussion, and revision of the paper. All authors read and approved the final version before publication.

Consent

NSSK certifies that a written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the editor-in-chief of this journal.

Competing Interests

The authors declare that they have no competing interests.

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