A Rare Case of Isolated Cerebellar Nodulus Infarction Without Vertigo

Vertigunun Eşlik Etmemiği Nadir Görülen İzole Serebellar Nodus Infarktı Olgusu

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ABSTRACT Nodulus, along with the flocculus, paraflocculus and ventral uvula constitutes the vestibulocerebellum and is irrigated by the medial branch of the posterior inferior cerebellar artery. Isolated nodular infarction is extremely rare because it is usually associated with infarction of other areas supplied by medial branch of the posterior inferior cerebellar artery. We present a case which severe nausea, vomiting, nystagmus and ataxia were the prominent symptoms of nodulus infarction without prominent vertigo. Patients applying with the same symptoms, even with absence of vertigo, verteobasilar ischemic stroke should come to mind at differential diagnosis and clinical suspicion should lead us to repeat neuroimaging with high resolution for accurate diagnosis.

Key Words: Vertigo; brain infarction; gait ataxia; cerebellar diseases


Anahtar Kelimeler: Vertigo; beyin infarktüsü; yürüme ataksisi; serebellar hastalıklar


The cerebellum can be divided anatomically and functionally into 3 major regions. 1) The cerebellar hemispheres and small part of the posterior vermis form the pontocerebellum, which receives input from the cerebral cortex via the pontine nuclei. 2) The anterior lobe and most of the posterior vermis make up the spinocerebellum, which receives inputs from spinal cord. 3) The nodulus and flocculus are connected with the vestibular nuclei and constitute the vestibulocerebellum. The nodulus lies in the midline cerebellum between the inferior medullary velum and uvula.1 Afferent inputs from the vestibular system is directed to nodulus which are involved in controlling eye movements and in postural ad-
justments to gravity. The nodulus is resiprocally connected with the vestibuler nuclei and functionally these nodulo-vestibuler purkinje fibers have an inhibitory effect on ipsilateral vestibuler nuclei. Nodulus is irrigat ed by the medial branch of the posterior inferior cerebellar artery (mPICA) and infarction in this territory cause symptoms of vertigo, nausea, vomiting, gaze evoked, asymmetrical, direction changing nystagmus, asymmetrical pursuit and/or optokinetic nystagmus, dysmetria, severe occipital headache or dysarthria.

CASE REPORT

A thirty seven years old female patient had headache, nausea and vomiting symptoms which started a month ago before admission to our hospital. Headaches were intermittan, bilateral localized to frontal areas and dramatically subsided with analgesic intake. Nausea and vomiting symptoms occured 2-3 times a day especially after meals. Her relatives also informed that she was slightly confused at the beginning which she couldn’t manage to do her social daily activities yet this symptoms resolved in two weeks. One week later after nausea and vomiting symptoms, fatigue and disequilibrium symptoms were also added. She was consulted to a neurologist and blood tests including complete blood test, BUN, creatinin, blood glucose level, Na, K, SGPT, SGOT, Thyroid function tests, ESR, CRP, Salmonella and Brucella IgM, IgG, AntidsDNA, ANCA were tested which showed no abnormality. Also an open 0.5 Tesla Magnetic Resonance Imaging (MRI) was taken which was reported as normal. As her symptoms persisted she applied to our hospital.

She was a healthy female patient with no previous illnesses or operation. She consumed 40 cigareettes a day for 12 years. At admission she was taking omeprazole and donperidone treatment. Her neurological examination revealed: The patient was conscious, her cooperation and orientation was intact, extraocular movements were full with normal pupils and fundi. She had gaze evoked horizontal nystagmus bilaterally which was prominent at the right side and also torsional nystagmus at upper and lower gazes which continued after fixation. She didnt have head tilt or dysartria. Her cerebellar tests were bilaterally distorted both at the upper and lower limbs and was ataxic to both sides with a negative Rhomberg test. Motor and sensory examination were normal. Deep tendon reflexes were normoactive with no pathological reflexes.

Head impulse test was negative and Dix hallpike test was applied which was negative also. Another 1.5 Tesla MRI was applied and showed restriction at nodulus bilaterally at Diffusion Weighted Image (DWI), no change at Apparent Diffusion Coefficient (ADC) and slight hyperintensity at FLAIR image which was interpreted as subacute infarction of cerebellar nodulus (Figure 1A-C). The patient also underwent MR angiography and didnt reveal any clear vascular abnormality.

She was evaluated by the cardiologist and Transthoracic, Transeusofagial Echocardiography and Holter investigation showed no abnormalities such as PFO, ASD or arrhythmia. Also immunologic markers for vasculitic diseases and levels of protein C, S, antithrombin 3, antiphospholipid antibodies were tested which were in normal ranges. Genetic tests for Factor Five Leiden mutation, methylenetetrahydrofolatereductase (MTHFR) gene mutation were sent and waiting for the results. Acetylsalicylic acid was started and quitting smoking was recommended. After one week her neurological examination improved showing slight bilateral horizontal nystagmus, skilled cerebellar tests both at upper and lower limbs. Her ataxia resolved with a slight distortion at tandem walking. One month later, her symptoms completely resolved showing almost normal neurological examination except for slight bilateral torsional nystagmus at right gaze.

DISCUSSION

Cerebellar nodulus is irrigated by medial branch of posterior inferior cerebellar artery. Nodulus infarction is usually associated with ischemic lesions in other areas supplied by mPICA and isolated infarction is rare and only reported as few cases.
Also infarction in the territory of the mPICA mostly cause severe vertigo which wasn’t prominent in our patient. There are also few cases of cerebellar infarction presenting with isolated lateropulsion without having vertigo or other signs of vertebrobasilar ischemia. Shan et al. described a patient with isolated body lateropulsion with cerebellar infarction but the lesion was in the territory of the lateral branch of the posterior inferior cerebellar artery. Muley and Bushara reported two cases with infarction in the territory of medial branch of superior cerebellar artery who presented purely with gait ataxia. Lee reported a case with isolated lateropulsion caused by a lesion at rostral vermis. These reports highlight the variety of neurological symptoms in cerebellar infarction according to specific lobular infarction or vascular territory.

Other symptoms such as dysmetria, gaze evoked nystagmus, which were the major findings of cerebellar lesions occurred in our patient. Cerebellar infarction in the territory of mPICA mostly imitate vestibular neuritis. Although patients usually have at least one of the central signs, e.g., dysarthria, gaze-evoked, asymmetrical, direction changing nystagmus, asymmetrical smooth pursuit and/or optokinetic nystagmus or dysmetria, there are few cases of nodulus infarction which showed none of the central signs and misdiagnosed as vestibular neuritis. Dysmetria, one of the major finding of the cerebellar infarction in the territory of the medial PICA, may be minimal or absent if the size of the infarct is not large. Also gaze evoked nystagmus commonly occurred in central vestibulopathy of cerebellar origin is sometimes absent in mPICA lesions. Our patient although didn’t have prominent vertigo symptom, after 0.5 Tesla Cranial MRI which revealed no pathology, was also misdiagnosed as vestibular neuritis. Attentive neurological examination by an experienced neurologist would have notify her central signs which were dysmetria, ataxia and gaze evoked nystagmus. Also young age and minimal vascular risk factors which was purely smoking in our patient, shouldn’t avoid us from diagnosing acute vascular events of the brain.
CONCLUSION

Although nodulus infarction is extremely rare, absence of vertigo makes our case more exclusive. Featuring of suspected symptoms even with absence of vertigo, clinicians should be aware of the possibility of infarction of isolated cerebellum lobules. Intense neurological examination, a complete and high resolution brain scan should be done to exclude central nervous system pathologies.

REFERENCES