



International Journal of Case Reports (ISSN:2572-8776)



Babinski-Nageotte Syndrome: Case Report of a rare variant of Lateral Medullary Infarction

Arunava Saha, MD¹; Kamal Pokhrel, MD¹; Gary Keilson, MD²

¹Department of Internal Medicine, Saint Vincent Hospital, Worcester, MA; ²Chief, Department of Neurology, Saint Vincent Hospital, Worcester, MA

ABSTRACT

Babinski-Nageotte Syndrome (BNS) is a rare variant of lateral medullary infarction, and only a few cases have been reported. In this case, a 52-year-old African-American gentleman presented with acute onset weakness on the left side of the body. On examination, there was a palpable right supraclavicular lymph node. Neurological examination showed right-sided gaze-evoked nystagmus and left hemiparesis with grade 3/5 power. Reflexes were normal except for extensor plantar responses on the left. MRI showed a subacute right-sided lateral brainstem infarct just below the pontomedullary junction with minimal progression in a repeat MRI after 24 hours. Workup for supraclavicular adenopathy with CT chest and CT abdomen and pelvis revealed neither neoplastic nor metastatic disease. Biopsy of supraclavicular lymph node was planned as an outpatient but was deferred as it was presumed to be reactive. He was conservatively managed with daily physiotherapy, aspirin, atorvastatin, and optimal blood pressure control. Babinski-Nageotte syndrome includes all symptoms of the Wallenberg syndrome with contralateral hemiparesis due to a spread of the lateral lesion to the pyramidal tract. Atherosclerotic occlusion and lipohyalinosis of the vertebral artery and posterior inferior cerebellar artery have been identified as the most common causes.

Keywords: Case Report, Lateral Medullary Infarct, Babinski-Nageotte, Wallenberg, Hemi-medullary syndrome.

DECLARATIONS OF INTEREST: The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

*Correspondence to Author:

Arunava Saha, MD
PGY1 Internal Medicine
Saint Vincent Hospital, Worcester,
MA

How to cite this article:

Arunava Saha, Kamal Pokhrel, Gary Keilson. Babinski-Nageotte Syndrome: Case Report of a rare variant of Lateral Medullary Infarction. International Journal of Case Reports, 2022, 6:278.



eSciPub LLC, Houston, TX USA.

Website: <http://escipub.com/>

By using the site/services, you are agreeing to our Policies: <https://escipub.com/terms-privacy-policy-disclaimer/>

Introduction:

Babinski-Nageotte Syndrome (BNS) is a variant of the brainstem syndromes characterized by muscle weakness in the contralateral side in addition to classic Lateral Medullary infarction findings. According to our literature survey, only a few cases have been reported. We report a case of a typical BNS with an ischemic lesion in the lateral medulla evident on MRI.

Case report:

A 52-year-old African-American gentleman presented with complaints of acute onset weakness of the left side of body with buckling of his left lower limb and need to drag the limb while walking, associated with tingling and numbness. Weakness had worsened to a point where he had difficulty in gripping objects in the left hand. This was followed a few hours later by acute onset dizziness with a sensation of the entire room spinning around him, holocranial headache with photophobia and a few episodes of vomiting. He had no difficulty in swallowing food, tinnitus, ear fullness, hearing changes, diplopia or any blurring of vision. He denied history of fever, diarrhea, rashes, trauma, urinary incontinence, seizures or any similar episodes in the past. He had a known history of diabetes, hyperlipidemia and hypertension which was well controlled on Lisinopril, Hydrochlorothiazide, Amlodipine and Metoprolol. He was a non-smoker and had no history of alcohol or recreational drug use.

General examination was significant for acute distress due to vertigo. Vitals were significant for elevated blood pressure of 176/48 mm Hg. There was a palpable right supraclavicular lymph node. Cardiovascular, respiratory and abdominal examination were unremarkable. Detailed neurological examination showed

normal higher mental function, right sided gaze evoked nystagmus with fast component to the left, worsening vertigo on head turning movements, left side hemiparesis with grade 3/5 power in left upper and lower limb, distal more involved than proximal, with extensors weaker than flexors in the arm and stronger than flexors in the legs. Handgrip was weak and pronator drift was present of the left upper arm. Reflexes were normal except for extensor plantar responses on the left.

Routine blood work including CBC, electrolytes, blood sugar, liver function, lipid panel, renal function were unremarkable. A head CT was negative for acute intracranial abnormality but revealed minimal asymmetry of vocal cords being more relaxed on the left side. It also showed right supraclavicular adenopathy measuring 2.0 x 1.7 cm. An MRI showed subacute right sided lateral brainstem infarct just below the ponto-medullary junction with minimal interval progression in a repeat MRI after 24 hours. CT angiography of head and neck did not reveal any atherosclerotic occlusion in the vertebral, basilar and cerebral arteries. Echocardiogram of heart did not reveal any thrombus or vegetation. Workup for supraclavicular adenopathy with CT chest and CT abdomen and pelvis revealed neither neoplastic nor metastatic disease. Biopsy of lymph node was planned as outpatient, but was deferred as it was decreasing in size and presumed to be reactive. He was conservatively managed with daily physiotherapy, aspirin, atorvastatin and optimal blood pressure control. Strength had been improving significantly on left lower extremity while left upper extremity weakness was still significant over the course of 10 days of hospital admission. He was

eventually discharge to a rehab facility and planned for further follow-up.

Discussion:

Babinski-Nageotte syndrome includes all symptoms of the Wallenberg syndrome with contralateral hemiparesis due to a spread of the lateral lesion to the pyramidal tract. It was first

described in 1902 and later named after the neurologists who initially investigated it, Joseph Babinski and Jean Nageotte. There have been a few case reports describing this rare neurological syndrome, which includes Chang et al. (1993)¹, Freitas et al. (2001)² and Irie et al. (2003)³

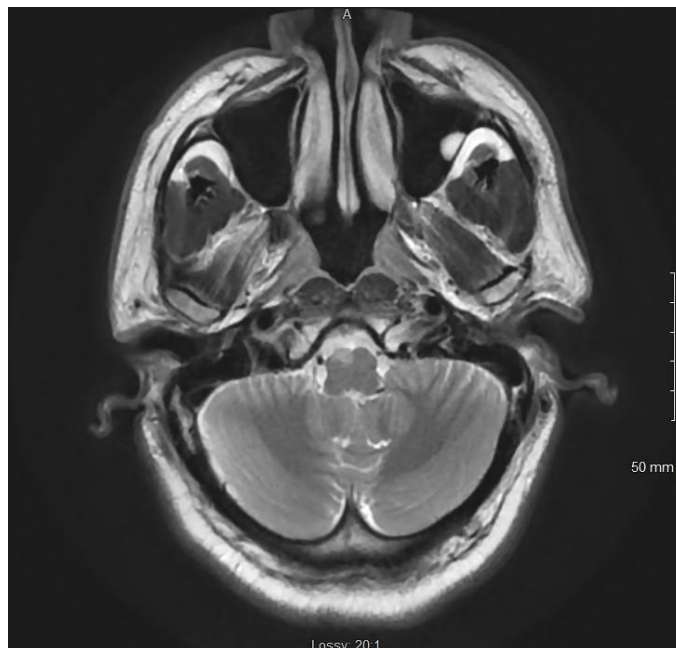


FIGURE 1: DWI MRI Brain, image obtained on admission, showing classic area of infarct involving the right medulla, extending to the corticospinal tract. Replicated with permission from the Department of Radiology, Saint Vincent Hospital, Worcester, MA

Lateral medullary syndrome is traditionally not associated with any limb weakness and it manifests with vestibulocerebellar signs and symptoms (limb ataxia, diplopia, nystagmus, and dizziness), loss of pain and temperature in ipsilateral face and contralateral trunk and limbs, bulbar muscle weakness and Horner's syndrome. Lateral and medial medullary regions are involved in hemimedullary syndrome whereas the involvement is limited to the lateral medullary zone and corticospinal tract in BNS.^{5,6} Hypoglossal palsy, an invariable symptom of hemimedullary lesion is not part of the Babinski-

Nageotte syndrome.⁸ Our patient had vomiting, vertigo and gaze evoked nystagmus which could be explained due to involvement of the vestibular nuclei and olivocerebellar fibers, while the contralateral weakness and positive Babinski can be explained by involvement of the pyramidal tract before decussation.

In the cases presented in literature, atherosclerotic occlusion, lipohyalinosis and dissection of the vertebral artery and posterior inferior cerebellar artery have been identified as the most common causes.²⁻⁵ Other pathologies such as syphilitic endarteritis are rare. In our

case, it was most likely from the underlying lipohyalinosis given the patient's history of hypertension and dyslipidemia.

Patient perspective:

I was doing pretty well until I woke up one fine morning and realized I was not able to move the left side of my body. I was scared, and realized I might have had a stroke. I called the ambulance and was rushed to the hospital. I was evaluated and found to have elevated blood pressures. Along with that, the doctors confirmed my worst suspicion, which was that I have had a stroke. They did an MRI which apparently revealed I had a small blood clot in my brain. I was started on a blood thinner medication. They also did a few more tests including a 2D echo of my heart. They also discovered a swelling on the left side of my chest. I was supposed to have a biopsy done for it after discharge. I was started on Physical and Occupational therapy and I worked with them diligently during the course of my hospital stay. I regained partial strength back in my left lower limb but my left upper limb continued to remain weak. I was discharged to a rehab facility subsequently. At the rehab, I regained my strength back considerably. I was able to walk with the help of a cane. My lymph node biopsy was not done as the surgeon told me it was decreasing in size so they would like to wait and watch.

During the time of hospitalization, I was told I have a rare kind of stroke. The neurologist who evaluated me also confirmed the same. I was explained that such constellation of symptoms with which I presented are usually rare. I was told that it is something which the medical fraternity needs to be aware about and the doctors would like to discuss about my case in a journal. I have happily consented for the same.

• Ethics approval and consent to participate:

Ethics approval was waived.

Consent to participate was obtained by obtaining informed verbal and written consent from the patient.

• Consent for publication:

Consent for publication was obtained both verbally and in written format from the patient, confirmed by signature on a consent form.

• Competing interests: The authors declare that they have no competing interests.

• Funding: Not applicable.

• Authors' contributions:

AS and KP were involved in the data collection, literature search and writing up the manuscript. GK provided expert opinion and also contributed to writing up the manuscript. All authors read and approved the final manuscript.

• Acknowledgements: Department of Radiology, Saint Vincent Hospital, Worcester, MA

References:

- [1]. Chang YY, Wu HS, Chen WH, Liu JS, Chen SS. Babinski-Nageotte syndrome: a case report. Gaoxiong Yi Xue Ke Xue Za Zhi. 1993 Oct;9(10):595-8. PMID: 8133548.
- [2]. de Freitas GR, Moll J, Araújo AQ. The Babinski-Nageotte syndrome. Neurology. 2001 Jun 12;56(11):1604. doi: 10.1212/wnl.56.11.1604. PMID: 11402132.
- [3]. Irie F, Toyoda K, Hagiwara N, Fujimoto S, Okada Y. Babinski-Nageotte syndrome due to vertebral artery dissection. Intern Med. 2003 Sep;42(9):871-4. doi: 10.2169/internalmedicine.42.871. PMID: 12691942.

14518679.

- [4]. Krasnianski M, Müller T, Stock K, Zierz S. Between Wallenberg syndrome and hemimedullary lesion: Cestan-Chenais and Babinski-Nageotte syndromes in medullary infarctions. *J Neurol*. 2006 Nov;253(11):1442-6. doi: 10.1007/s00415-006-0231-3. Epub 2006 Jun 14. PMID: 16775654.
- [5]. Pandey S, Batla A. Opalski's syndrome: A rare variant of lateral medullary syndrome. *J Neurosci Rural Pract*. 2013;4(1):102-104. doi:10.4103/0976-3147.105642
- [6]. Kim JS. Pure lateral medullary infarction: clinical-radiological correlation of 130 acute, consecutive patients. *Brain*. 2003 Aug;126(Pt 8):1864-72. doi: 10.1093/brain/awg169. Epub 2003 May 21. PMID: 12805095.
- [7]. Sacco RL, Freddo L, Bello JA, Odel JG, Onesti ST, Mohr JP. Wallenberg's lateral medullary syndrome. Clinical-magnetic resonance imaging correlations. *Arch Neurol*. 1993 Jun;50(6):609-14. doi: 10.1001/archneur.1993.00540060049016. PMID: 8503798.
- [8]. Mossuto-Agatiello L, Kniahynicki C. The hemimedullary syndrome: case report and review of the literature. *J Neurol*. 1990 Jun;237(3):208-12. doi: 10.1007/BF00314596. PMID: 2196341
- [9]. Krasnianski M, Neudecker S, Schluter A, Zierz S. Babinski-Nageotte's syndrome and Hemimedullary (Reinhold's) syndrome are clinically and morphologically distinct conditions. *J Neurol*. 2003 Aug;250(8):938-42. doi: 10.1007/s00415-003-1118-9.

