ECRONICON OPEN ACCESS

EC NEUROLOGY
Case Report

Subcortical Aphasia due to Tuberculoma: A Rare Case and Review of Literature

Faheem Arshad[1]*, Reshma Sultana Naik[1], Shumyla Jabeen[2], Raghavendra Kenchaiah[1], Abhinith Shashidhar[3] and Chandrajit Prasad[2]

[1]Department of Neurology, National Institute of Mental Health and Neurosciences (NIMHANS), Bangalore, India

[2]Department of Neuroimaging and Interventional Radiology, NIMHANS, Bangalore, India

[3]Department of Neurosurgery, NIMHANS, Bangalore, India

***Corresponding Author:** Faheem Arshad, Department of Neurology, National Institute of Mental Health and Neurosciences (NIMHANS), Bangalore, India.

Received: December 25, 2020; **Published:** Janauray 30, 2021

DOI: 10.31080/ecne.2021.13.00852

Abstract

Subcortical aphasia refers to impairment in language due to pathology in regions other than cerebral hemispheres. It is an under-recognized entity and usually involves the thalamus and basal ganglia. Thalamic lesions have multiple causes, and a single etiology may have variable presentations. Language disturbances in the form of aphasia can occur with thalamic involvement and have not been reported frequently. We report a probable case of thalamic tuberculosis with subcortical aphasia and review its mechanism.

Keywords: *Aphasia; Thalamus; Language; Tuberculosis*

Introduction

Thalamus is a multifunctional structure between the midbrain and forebrain which acts as a relay of sensory information between the cerebral cortex and subcortical structures. It also plays an important role in memory, emotions, regulating sleep wake cycle and motor control via its connections to basal ganglia, cerebellum and hippocampus. Though the role of thalamus in language remains a matter of debate, multiple thalamic nuclei have been implicated as indicated by their connectivity with the cerebral cortex. Thus, language impairment (also known as aphasia) may occur after involvement of deep cortical structures due to disruption of the relay pathways to and from cortical regions that mediate language processing. Fluent speech output, impaired comprehension and impaired naming with verbal paraphasia are characteristic findings observed in language impairments involving the thalamus (also known as thalamic aphasia). Thus, specific features of language impairment suggest that subcortical structures contribute to language organization which has been proved by lesion studies. In addition, the resulting patterns of impairment are typically milder in form and recover better than aphasias that follow left cortical damage. Aphasia as a manifestation of thalamic involvement has been infrequently reported. We describe a patient who presented with subcortical aphasia secondary to a bithalamic lesion.

Case Presentation

32 year lady presented to us with two weeks history of gradual onset, global, non-throbbing headache associated with vomiting. After 1 week, family members noticed that she remained drowsy for several hours but was still obeying verbal commands and has difficulty in forming sentences. Husband reported that she had word finding difficulty with difficulty in understanding and naming objects, was lethargic and used to sleep for long periods. They denied any history of fever, seizures, weakness of any side or past history of any chronic illness. Her systemic examination was normal. She was drowsy though oriented. Memory was grossly intact. Language assessment showed a fluent speech with occasional naming difficulty, word finding difficulty, normal repetition, mildly affected comprehension and

verbal paraphasia. Fundus examination showed bilateral mild papilledema. Her vertical saccades were slow and amplitude was reduced. Pupils were normal. She did not have weakness, sensory loss, pyramidal, cerebellar or meningeal signs.

Routine blood investigations and chest X-ray were normal. Computed tomography (CT) of the brain revealed evidence of a well-defined predominantly hypodense lesion involving bilateral thalami. It showed a thick, irregular hyperdense rim on plain CT with peripheral enhancement on post-contrast imaging (Figure 1a and 1b). Extensive surrounding edema was noted with effacement of the third ventricle. Magnetic resonance imaging (MRI) of brain showed a well-defined bithalamic lesion with extensive surrounding edema isointense to gray matter on T2 weighted images with few hypointense areas. It was hypointense on T1 weighted images with a hyperintense rim. Thick peripheral enhancement was seen on post-contrast images. Well-defined lesion with surrounding edema, T2 hypointense areas and T1 hyperintense rim with peripheral enhancement weighted the diagnosis in favour of tuberculoma. Patient was started on antitubercular treatment and asked to monitor her liver and renal function tests regularly. Language assessment on follow up after 2 months showed mild improvement in her word finding and comprehension. Currently patient is on regular follow up.

Figure 1a

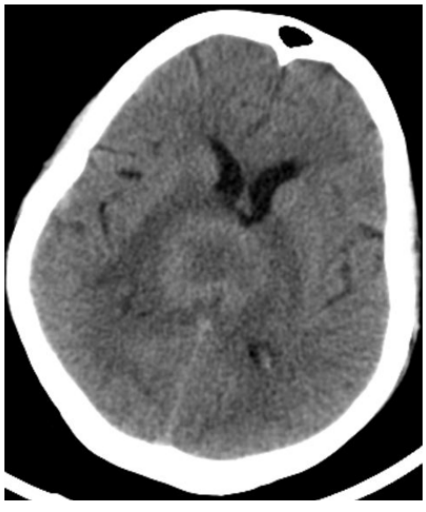

Figure 1b

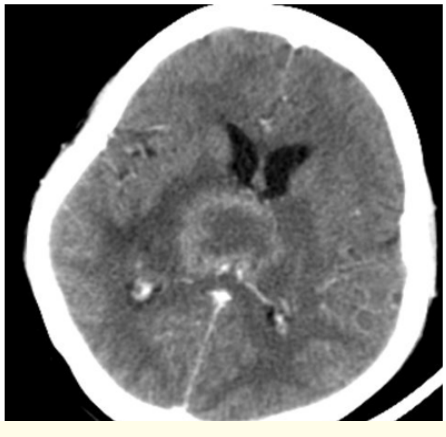

Figure 1: *CT brain (a)- Non contrast study shows well-defined hypodense lesion involving both thalami with a thick hyperdense rim (b) Contrast study shows peripheral enhancement of the lesion. Note the surrounding edema.*

Figure 2a

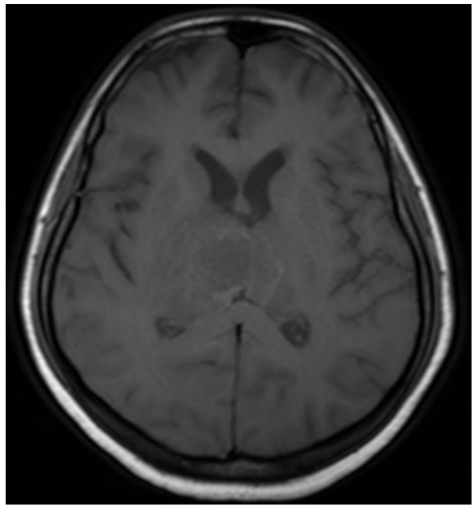

Figure 2b

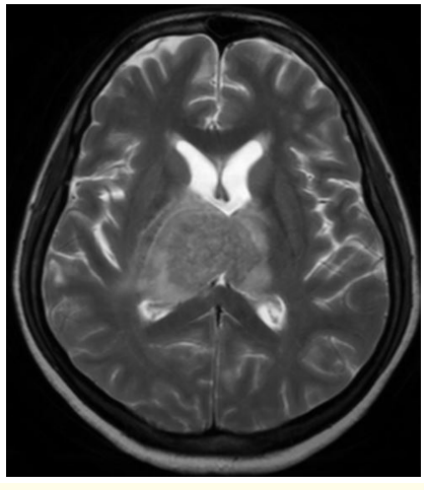

Figure 2c

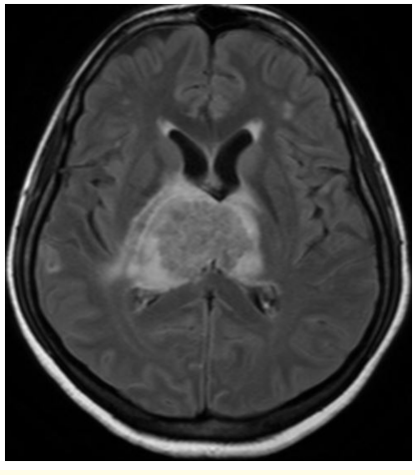

Figure 2d

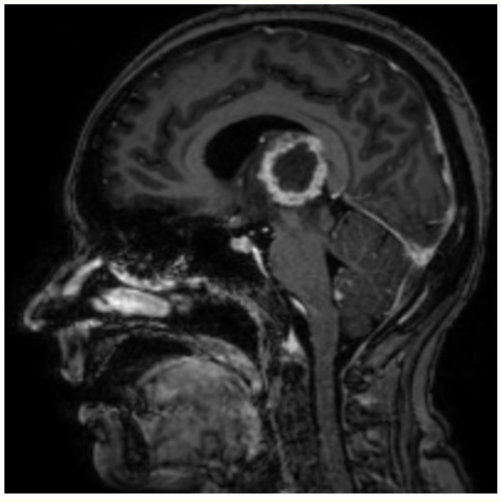

Figure 2: *MRI brain (a) T1 weighted MRI shows a well-defined bithalamic hypointense lesion with a hyperintense rim; (b, c) T2/FLAIR weighted image shows bithalamic lesion isointense to gray matter with few hypointense areas, (d) Post contrast T1 weighted image shows thick peripheral enhancement. Areas of T2 hypointensity with T1 hyperintense rim and peripheral enhancement are features suggestive of a tuberculoma.*

Discussion

We report an uncommon case of thalamic aphasia that occurred due to a giant tuberculoma. Though impairment of language abilities have been described after thalamic damage but the anatomical precision of the lesion location and language assessment in those cases has been variable [1,2]. This could be due to varied aetiology of the lesions in the thalamus. In most of the cases, etiology for bilateral thalamic lesion is vascular in the form of arterial (artery of percheron infarct) or venous infarcts (deep venous thrombosis) or space occupying lesions.

Language disorder secondary to subcortical damage was spurred when Marie in 1906 challenged the traditional discussion of aphasia. Subsequently Penfield and Roberts model of thalamic involvement in language was proposed and they postulated that multiple thalamic regions were in reciprocal relationship with language related cortical structures thus suggesting a subcortical integration site. The phenomenon of thalamic lesion induced cortical hypoperfusion and hypometabolism has been demonstrated in patients with subcortical aphasia. Thus, language disorder results from secondary functional cortical alterations which are potentially reversible, as opposed to the structural damage caused by the lesion itself. This reversible nature of the deficits might explain the strong tendency for improvement of subcortical aphasia [3]. Cortical diaschisis theory may explain the language deficits in subcortical damage. Transient depression of the function of intact brain regions, remote from, but connected to a focal lesion is defined as diaschisis. According to this theory due to the loss of essential input from thalamocortical projections cortical neuronal activity is critically decreased [4]. Thalamus has about 13 distinct groups of nuclei. In order to localise the lesions four principal thalamic functional regions have been defined- the anterior, lateral, medial and posterior. Their main functions mirror those of the corresponding regions of cortex to which they project. The anterior nuclei are involved in language and memory function. They are supplied by the polar artery, which if absent, may result in language disturbances in the form of dysphasia and amnesia, as a part of paramedian thalamic infarction [5]. Schmahman JD [6] described four thalamic stroke syndromes which closely correspond to the functional deficits. One of the vascular syndromes is the polar artery territory stroke which results in fluctuating arousal, expressive dysphasia and impaired naming. Thus, many studies [7] have described deficits in language in patients with thalamic infarcts. However, patterns of language deficits in infective or neoplastic lesions of the thalamus remain poorly understood.

Reports suggest that there is decreased regional cerebral blood flow in several peri-Sylvian cortical regions, including the left inferior frontal gyrus, supramarginal gyrus, and superior temporal gyrus after infarction of left anterior thalamic structures manifested as dysnomia and poor semantic and phonemic fluency [8]. These data suggest an apparent lack of a 1:1 structural-functional relationship of thalamic nuclei and their cortical targets with respect to language. Crosson B [9] suggested that the common deficits produced by disparate lesions point to the possibility of damage to different portions of a common rather than separate neural systems. Thus, role of networks may play a role in explaining the deficits in language which may be different in different lesions of thalami. Mass lesions produce intermittent difficulties with language rather than persistent deficits as seen in vascular lesions. Thus, it is the disconnection between cortical language centers and thalamic nuclei which results in thalamic aphasia.

Gaye M., *et al*. [10] have reported a case of thalamic tuberculoma in a pregnant lady who presented with systemic symptoms and hemiplegia but language disturbances were not reported. Mridula., *et al*. [11] reported a case of thalamic tuberculoma presenting as corticobasal syndrome and focal cortical atrophy. Agarwal., *et al*. [12] reported a case of thalamic abscess with encephalopathy for whom language assessment was not possible. There is paucity of literature in terms of primary thalamic tuberculosis with subcortical aphasia which was seen in our patient and is rare. Further research to delineate the exact pattern of language deficits in patients with thalamic lesions is warranted.

Conclusion

To conclude, thalamus may be involved in many neurological diseases with varied presentations including aphasias which are not always cortical. Thus, in a patient with reduction in verbal output, word finding difficulty, verbal paraphasias, preserved repetition and defective comprehension, a diagnosis of thalamic aphasia should be considered. Though a single lesion may not be able to explain the deficit, further network studies are required in this regard to substantiate the literature.

Bibliography

1. De Witte Lieve., *et al*. "Cognitive, affective and behavioural disturbances following vascular thalamic lesions: a review". *Cortex* 47.3 (2011): 273-319.

2. Hoffmann Michael. "Thalamic semantic paralexia". *Neurology International* 4.1 (2012): e6.

3. Benson DF. "Aphasia and related disorders: a clinical approach". *Mesulam MM. Principles of Behavioral Neurology* (1985): 193-238.

4. Astrup Jens., *et al*. "Thresholds in cerebral ischemia-the ischemic penumbra". *Stroke* 12.6 (1981): 723-725.

5. Perren Fabienne., *et al*. "The syndrome of combined polar and paramedian thalamic infarction". *Archives of Neurology* 62.8 (2005): 1212-1216.

6. Schmahmann Jeremy D. "Vascular syndromes of the thalamus". *Stroke* 34.9 (2003): 2264-2278.

7. Van Der Werf., *et al*. "Deficits of memory, executive functioning and attention following infarction in the thalamus; a study of 22 cases with localised lesions". *Neuropsychologia* 41.10 (2003): 1330-1344.

8. Shim Yong S., *et al*. "A serial study of regional cerebral blood flow deficits in patients with left anterior thalamic infarction: anatomical and neuropsychological correlates". *Journal of the neurological sciences* 266.1-2 (2008): 84-91.

9. Crosson Bruce. "Thalamic mechanisms in language: a reconsideration based on recent findings and concepts". *Brain and Language* 126.1 (2013): 73-88.

10. M Gaye., *et al*. "Thalamic Tuberculoma: A Case Report". *The Internet Journal of Neurosurgery* 14.1 (2018).

11. Mridula Kandadai Rukmini., *et al*. "Corticobasal syndrome due to a thalamic tuberculoma and focal cortical atrophy". *Journal of Neurology, Neurosurgery and Psychiatry* 79.1 (2008): 107-108.

12. Agrawal Amit and Rafael Cincu. "Spontaneous thalamic abscess in a nonimmunocompromised patient". *Infectious Diseases in Clinical Practice* 18.1 (2010): 83-84.

Volume 13 Issue 2 February 2021