Research Article

Post Stroke Abulia: A Case Report of Fronto-Subcortical Circuit Dysfunction

Dr. Aishwarya[1], Dr. Ramachandran[2]

[1]Post graduate, Department of General Medicine, Sree Balaji Medical College and hospital, Chennai
[2]Professor,Department of General Medicine, Sree Balaji Medical college and Hospital, Chennai.
Corresponding Author:
Dr. Aishwarya
Junior Resident, Department of General medicine, Sree Balaji Medical college and Hospital, Chennai Bharath institute of Health and Education Research. Email: reddyaishwarya5@gmail.com.

Abstract

Background: Abulia is a disorder of diminished motivation characterized by reduced speech, reduced spontaneous activity, and emotional unresponsiveness.

It is commonly associated with lesions involving frontal-subcortical circuits. We report a 79-year-old female with type 2 diabetes mellitus who presented with progressive reduction in speech, responsiveness, and spontaneous activity associated with a fall. MRI brain showed a left gangliocapsular infarct. Initially treated as post-stroke metabolic encephalopathy, the patient showed no improvement in clinical status with deficit disproportionate to the infarct. Persistent apathy, reduced initiation, and diminished speech led to the diagnosis of post-stroke abulia. Treatment with amantadine and levodopa-carbidopa resulted in clinical improvement. This case highlights the importance of early recognition of abulia as a treatable complication of stroke and the role of dopaminergic therapy in improving outcomes.

Introduction

Abulia is syndrome of hypo function characterized by impaired motivation, reduced initiative, and diminished speech. It occurs due to Lesions involving the frontal cortex, basal ganglia, thalamus, and cingulate gyrus disrupting the fronto-subcortical circuits, especially involving dopaminergic pathways. It is an under-recognized complication of stroke and is frequently misdiagnosed as depression, aphasia, encephalopathy, or hypoactive delirium.

Case Presentation

A 79-year-old female with type 2 diabetes on oral hypoglycemic since 5 years with no other co-morbidity presented with reduced speech and responsiveness for 4 days following a fall 2 weeks earlier. The fall was not associated with loss of consciousness or seizures. However, the family members noticed that the patient had poor interaction and was slow to respond to questions. However she progressively became drowsy with no spontaneous motor activity and hence was brought to hospital for further care. An MRI brain with MRA, MRV done 13 days after the fall revealed a subacute infarct in left gangliocapsular region. On physical examination patient was drowsy, arousable only with painful stimuli, with Blood pressure 180/100mm Hg, Pulse:98/min, Respiratory rate:16/min. Oxygen SaO2:98% at room air,
GCS 5/15, with increased tone in all four limbs, brisk reflexes in Right upper limb and lower limb, Plantar bilaterally extensor.

Routine labs showed Hemoglobin: 11.1 g/dL, Platelets: 108,000/mm³Renal and liver function: within normal limits, Electrolytes were normal, CSF analysis was normal, Procalcitonin was negative, EEG showed generalized slowing without evidence of seizure.

The patient was initially managed as a case of post-stroke metabolic encephalopathy with Anti platelets, Statins, Antiepileptics, Antibiotics and Supportive care. However, since there was no improvement in responsiveness despite treatment with persistent decreased speech, initiation, and no spontaneous activity, a review of the diagnosis was done.

Patient was suspected to have post stroke abulia with persistent decreased initiation and speech, A review of the Imaging revealed the lesion potentially along the frontal / subcortical circuit. Patient was therefore started on amantadine and levodopa-carbidopa after which the patient improved symptomatically with better responsiveness.

Discussion

Poor responsiveness in a post stroke patient is a common clinical challenge and may result from multiple etiologies including metabolic encephalopathy, non-convulsive seizures, post-stroke depression, aphasia, hypoactive delirium, catatonia, medication effects, infections, or structural brain lesions affecting consciousness and motivation. In the present case, the patient initially presented with progressive reduction in speech, responsiveness, and spontaneous

activity following a fall and was subsequently found to have a left gangliocapsular infarction on magnetic resonance imaging. The degree of clinical impairment appeared disproportionate to the size and location of the infarct, raising concerns regarding alternative causes of altered sensorium. Extensive evaluation including metabolic parameters, cerebrospinal fluid examination, and electroencephalography failed to identify infectious, metabolic, or epileptic etiologies. Despite standard post-stroke management and supportive care, the patient's responsiveness remained significantly impaired, necessitating reconsideration of the diagnosis. The persistence of profound apathy, lack of initiation, delayed responses, and absence of spontaneous motor and verbal activity suggested a motivational disorder..

Abulia results from disruption of dopamine-mediated fronto-subcortical circuits particularly involving the basal ganglia, anterior cingulate cortex, thalamus, and dopaminergic pathways. Damage leads to lack of will, drive or initiative for action, speech and thought.

This case highlights an uncommon presentation of left gangliocapsular infarction manifesting predominantly as abulia. The gangliocapsular region plays an important role in frontal-subcortical networks that regulate motivation, behavior, and goal-directed activity. Damage to these circuits disrupts dopaminergic transmission and may result in severe apathy and reduced voluntary activity despite preserved motor pathways. Previous studies have shown that lesions involving the basal ganglia and internal capsule are strongly associated with motivational disorders including abulia and apathy after stroke. The following structural lesions have been attributed as factors in the etiology of abulia:

1. Anterior cingulate cortex.
2. Unilateral anterior cerebral artery.
3. Bilateral lesions in the medial frontal lobes, basal ganglia, supplementary motor areas, caudate nuclei, and cingulate gyri.
4. Frontal convexity damage.
5. Focal subcortical lesions of the caudate nucleus, anterior thalamus, globus pallidus, internal capsule, and midbrain.
6. Disconnections of limbic tracts projecting from the anterior thalamus to the cingulate.
7. Embolism occurring in the subthalamic-thalamic penetrating artery of Percheron leading to bilateral meso-diencephalon infarct may damage the medial thalamus and upper mesencephalon.
8. Bilateral lesions at or rostral to the meso-diencephalic junction or bilateral damage to the frontal lobes.

Diagnosis of abulia is primarily clinical. Important features include decreased spontaneity of speech and movement, prolonged response latency, reduced initiative, and lack of persistence with tasks. Recognition of this syndrome is clinically important because delayed diagnosis may lead to complications such as malnutrition, dehydration, pressure ulcers, deep vein thrombosis, and prolonged hospitalization. Dopaminergic agents have shown benefit in improving motivation and responsiveness. In the present case, initiation of amantadine and levodopa-carbidopa resulted in marked symptomatic improvement, supporting the role of dopamine dysfunction in the pathophysiology of post-stroke abulia. Early recognition and prompt initiation of targeted therapy can significantly improve neurological recovery, functional outcomes, and quality of life in affected patients.

Conclusion

Abulia is an under-recognized but treatable complication of stroke. This case of left gangliocapsular infarction presenting predominantly with reduced speech, apathy, and diminished spontaneous activity highlights the diagnostic challenge associated with the condition. Early recognition of abulia is essential, especially in stroke patients with persistent decreased initiation despite stable metabolic and neurological parameters. Prompt diagnosis and initiation of dopaminergic therapy can lead to significant clinical improvement and better functional recovery. Early diagnosis is essential for better outcomes.

References

1. Vijayaraghavan L, Krishnamoorthy ES, Brown RG, Trimble MR. Abulia: a delphi survey of British neurologists and psychiatrists. Mov Disord. 2002 Sep;17(5):1052-7. [PubMed]
2. Hastak SM, Gorawara PS, Mishra NK. Abulia: no will, no way. J Assoc Physicians India. 2005 Sep;53:814-8. [PubMed]

3. Siegel JS, Snyder AZ, Metcalf NV, Fucetola RP, Hacker CD, Shimony JS, Shulman GL, Corbetta M. The circuitry of abulia: insights from functional connectivity MRI. Neuroimage Clin. 2014;6:320-6. [PMC free article] [PubMed]
4. Jorge RE, Starkstein SE, Robinson RG. Apathy following stroke. Can J Psychiatry. 2010 Jun;55(6):350-4. [PubMed]
5. Spiegel DR, Chatterjee A. A case of abulia, status/post right middle cerebral artery territory infarct, treated successfully with olanzapine. Clin Neuropharmacol. 2014 Nov-Dec;37(6):186-9. [PubMed]
6. Thome J, Foley P. Agomelatine: an agent against anhedonia and abulia? J Neural Transm (Vienna). 2015 Aug;122 Suppl 1:S3-7. [PubMed]
7. Naik VD. Abulia following an episode of cardiac arrest. BMJ Case Rep. 2015 Jul 01;2015[PMC free article] [PubMed]
8. Muqit MM, Rakshi JS, Shakir RA, Larner AJ. Catatonia or abulia? A difficult differential diagnosis. Mov Disord. 2001 Mar;16(2):360-2. [PubMed]