Sir,
Akinetic Mutism, apathy, Aboulia describe diminished motivation relating to decreased expression of a behaviour\(^1\) which can be in the form of motor or speech behaviour. Aboulia is a syndrome of hypofunction which is characterized by lack of initiative, spontaneity and drive, apathy, slowness of thought (bradyphrenia), and blunting of emotional responses and response to external stimuli.\(^2\) Distinguishing aboulia from inability to perform an activity due to cognitive, linguistic or physical disabilities\(^3\) is important as it interferes with the diagnosis and management of the neurological population. Aetiology may be vascular, trauma, tumour, degeneration, drug-induced, Dementia, Fronto-temporal dementia, Anterior Cerebral Artery Infarctions, and disorders of the right hemisphere such as stroke and white matter diseases.\(^3\)

GR, a 43-year-old female, reported to the Neuroemergency care in March 2020 with complaints of continuous throbbing intense headache for 3 days. She became withdrawn, drowsy most of the day with minimal monosyllabic appropriate utterances. No known comorbidities. On examination, she was apathetic, dull, no aphasia or dysarthria, subtle pyramidal signs on the right with no evident signs of meningeal irritation. MRI of the brain [Fig 1a and 1b] with MRA and MRV showed acute thrombosis of deep cerebral venous sinuses indicating Venous Infarct. Medical management was initiated with anticoagulants and anti-oedema measures followed by the endovascular intervention.

Subsequently, a detailed cognitive, speech, language and swallowing evaluation by Speech Language Pathologist (SLP) noted adequate consciousness, alertness, orientation to space and person and the right upper and lower limb weakness with reduced faciomesial expressions. Oral mechanism examination with gag and velar elevation was normal. Cranial nerves for speech, hearing and swallowing were normal clinically. Language assessment using Modified Western Aphasia Battery (WAB) in the first language Tamil indicated her responses were slow, elicited with repeated questioning, and constant reminders to focus, paucity of spontaneous speech, slowness of thought, limited verbal fluency to single-word or two-word phrases with increased response time and preserved reading skills. Impaired writing due to right-sided weakness. Visual Neglect and Agnosia was ruled out. Speech was slow, monotonous with normal articulation, hypophonic with a breathy voice, and normal nasalance. Grade 1 (G-1, R-0, B-3, A-0, S-0) indicated Mild Dysphonia. Maximum phonation time could not be recorded, while cough efforts were strong. Bedside swallow evaluation was normal and was gradually started on oral feeds. VLS by ENT for persistent dysphonia with odynophagia indicated normal larynx and laryngopharynx. No prior history of anger burst out, depression or anxiety was reported. On follow-up, she was passive, poorly participating in conversations. She was unable to complete Mini Mental State Examination. A score of 4 on modified Rankin Scale (mRS) was given. Prior to admission, GR was a happy homemaker. Speech and Language evaluation was not conclusive of an apparent significant linguistic, cognitive or speech deficit. Although Dysarthria was ruled out, her voicing efforts were inadequate for verbal communication. Bradyphrenia and reduced spontaneous motor movements were conclusive of an extrapyramidal disorder. The finding of apathy was eliminated with a detailed evaluation which denoted aboulia.

The lack of spontaneity and volitional behaviours is linked to the neural network that consists of the Dorsolateral prefrontal cortex (DLPFC), Anterior Cingulate Cortex (ACC), Supplementary Motor Area (SMA), Thalamus and Basal Ganglia with Dopamine as an important neurotransmitter.\(^4\) However, aboulia may also be caused by subcortical lesions of the anterior thalamus,\(^5,6\) caudate nucleus,\(^5,6\) globus pallidus, internal capsule, frontal lobes,\(^4\) Progressive Supra Nuclear Palsy (PSP).\(^4\) Functional localization of aboulia involve disruptions in the network.

Reciprocal connections with cingulate gyrus, other limbic structures and frontal cortex provide an interface between the decision-making process and the emotions. The connection between motivation and action is governed by the limbic striatopallidal connections. These are derived from the concepts of observation that the amygdala and hippocampal formation has afferent connections with limbic striatopallidal and projects to the rest of the basal ganglia in turn involved in eliciting and forming motor performances.\(^1\) [Figure 1c]

The diagnosis is done clinically. In Fisher’s “Telephone test” the patient thought to respond better over a telephonic conversation than face to face.\(^2\) [Table 1]

| Clinical Features of Aboulia considered based on the factor analysis\(^4\) |
|---------------------------------------------------------------|
| (1) Poor initiation and sustenance of purposeful movements |
| (2) Lack of spontaneous movement |
| (3) Poverty of spontaneous speech |
| (4) Increased response time to queries |
| (5) Passivity |
| (6) Reduced emotional responsiveness and spontaneity |
| (7) Diminished social interaction |
| (8) Poor interest in usual pastimes |

\(\text{Table 1: Common Clinical Features of Aboulia considered based on the factor analysis}\(^4\)\)
The clinical presentation of abulia as a communication disorder is under reported and often ignored by clinicians skilled in diagnosing and rehabilitating linguistic, cognitive and communication disorders. Cerebral dysfunction such as delirium, language disorder such as Aphasia, mood disorder such as depression may co-exist in several conditions. Aphasia in subcortical strokes are presumably less. Since abulia is commonly seen in subcortical involvement, clinicians should look for distinguishing abulia from other disorders especially Aphasia. Primary care providers (PCPs) such as neurologists may be focused on motor recovery, which could be hindered due to an underlying unattended cause such as Abulia presenting as a barrier to recovery in a patient, otherwise misdiagnosed as post stroke depression (25%-50%).[1] Dysfunction in a region out of the area of a lesion can occur in regions that are connected functionally (Diaschisis). A comprehensive Neurological, Neurocognitive and Language examination is of great clinical value in identifying, quantifying and treating Abulia. A more commonly seen Parkinson’s disease can cause abulia.[2,3] Due to poor initiation and varied presentation, people with abulia may even suffer from malnutrition and dehydration.[2,7] This state of diminished motivation can be partially or completely reversed with appropriate medical management and rehabilitation. Dopamine agonists have been stated effective in Abulia.[8,9]

Direct or indirect damage to the circuit that controls voluntary behaviours can present in the form of a communication disorder which is non-classical of aphasia, dystarhria or cognitive-communication disorder. Also, the disparity in the severity of a disorder and their speed of motor, language recovery may be additionally dependent on this factor. PCPs should involve Rehabilitation specialists such as SLPs playing a critical role in the evaluation and management of such complex patients and in turn help improve their quality of life. Since the management is complex and long term, involvement of multidisciplinary team of experts helped GR gradually progress to an mRS score of 1 with appropriate medical management and cognitive rehabilitation.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

Declaration of patient consent
The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Srimathy Narasimhan, Shankar Balakrishnan
Centre for Hearing, Communication and Swallow Sciences, Department of Neurology, MIOT International Hospitals, Chennai, Tamil Nadu, India

Address for correspondence: Ms. Srimathy Narasimhan, Centre for Hearing, Communication and Swallow Sciences, MIOT International Hospitals, Chennai - 600 089, Tamil Nadu, India. E-mail: srimathy.narasimhan@gmail.com

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Submitted: 24-May-2020 Revised: 07-Jun-2020 Accepted: 26-May-2020 Published: 25-Sep-2020

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DOI: 10.4103/aian.AIAN_467_20