CASE REPORT

Abulia following an episode of cardiac arrest

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SUMMARY

The word 'abulia' means a lack of will, initiative or drive. The symptoms of abulia include lack of spontaneous action and speech, reduced emotional responsiveness and social interaction, poor attention and easy distractibility. These symptoms are independent of reduced levels of consciousness or cognitive impairment. We describe a case of a socially active 72-year-old female patient who presented with symptoms of abulia which may have occurred due to damage of the frontosubcortical circuits following an episode of cardiac arrest. The patient's symptoms improved dramatically following treatment with bromocriptine.

BACKGROUND

Abulia is defined as a lack of will or motivation. It usually follows injury to the brain in the areas of the basal ganglia, frontal lobes, cingulate gyrus or the frontosubcortical circuits. It is a common but usually underdiagnosed condition. The symptoms of abulia are often confused with aphasia, post-stroke depression and other neuropsychiatric conditions such as dementia. It is essential that abulia be identified as it is potentially treatable and is associated with significant loss of function and increased burden on the carer.¹

We present an interesting and unusual case of abulia which occurred following an episode of cardiac arrest.

CASE PRESENTATION

A 72-year-old socially active female patient with no history of neurological or psychiatric illness presented with lack of spontaneous action, loss of interest and poor concentration in her routine day-to-day activities. There was also lack of speech with loss of appetite and reduced food intake. The patient had difficulty in getting up every morning and would sometimes stay in bed as late as 3pm in spite of going to sleep early the previous night. She did not show any emotional response or interest in recent family events or gatherings.

The patient had a 10-year history of ischaemic heart disease with myocardial infarction, having undergone angioplasty twice. One year previously she had two episodes of ventricular tachycardia, one of which was complicated by cardiac arrest that required treatment with cardiopulmonary resuscitation and defibrillation with multiple shocks. An implantable cardioverter defibrillator was placed after this event. Her cardiac function had since then remained stable. According to her family members, it had become apparent soon after this episode that she had started displaying her present symptoms. The patient was diagnosed as

having depression and was started on various antidepressants but with no benefit. She also received cognitive-behavioural therapy with similar poor results. Antidementia drugs were also tried later with little benefit noted.

On examination, the patient appeared withdrawn and inattentive. She did not maintain eye contact with the examiner and usually looked the other way. Her vitals were stable. She was conscious and well oriented to time, place and person. Her speech was limited to single words or simple phrases, those too on repeated prompting. Her voice lacked the normal volume and animation. Her reading and writing skills in her first language were also intact though slow to elicit. Mini mental state examination (MMSE) was not possible due to poor cooperation. Her cranial nerve examination, power, tone, reflexes and plantars revealed no significant abnormalities. There were no classical signs of Parkinson's disease including rigidity or tremors.

INVESTIGATIONS

Routine investigations including full blood count, serum electrolytes and B12 levels were normal. Urine microscopy was normal. Two-dimensional echocardiography showed a hypokinetic posteroinferior and apical wall of the heart with mild mitral regurgitation. Left ventricular ejection fraction was 30–35%. CT scan of the brain showed bilateral periventricular hypodensity suggesting ischaemic changes. MRI of the brain was contraindicated as the patient had an automated implantable cardioverter defibrillator (AICD).

DIFFERENTIAL DIAGNOSIS

- Post-stroke depression: mood disorder. Patients have a persistent sad mood and negative thought content. Such patients may have a history of depression.
- Aphasia: language disorder. Patients appear to be well with normal mood and behaviour. They attempt to communicate but with difficulty. They socialise appropriately.
- Parkinson's disease: movement disorder. Patients exhibit rigidity, tremors, slowness of movement and difficulty in walking. Cognitive and behavioural problems occur at a later stage of the disease.

TREATMENT

On the basis of symptoms and examination findings and in view of a possible neurological insult during cardiac arrest, the patient was diagnosed as abulic. She was treated with bromocriptine 2.5 mg once a day and was monitored for possible side effects. Within a week, she started to take interest in her

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surroundings with improved motivation levels. The bromocriptine dose was increased gradually and the patient showed maximal response at a dose of 5 mg two times per day. No side effects were reported. Her antidepressants were gradually discontinued.

OUTCOME AND FOLLOW-UP

After 3 months of treatment, the patient had returned to a near prearrest state level of function, getting up early, socialising, taking interest in daily activities as well as having a normal appetite with good verbal output.

DISCUSSION

Abulia is the lack of motivation and goal-directed behaviour that is seen predominantly with lesions of the basal ganglia, frontal lobes and cingulate gyrus. The severe form of this disease, akinetic mutism, occurs following bilateral lesions at or rostral to the mesodiencephalic junction or following bilateral damage to the frontal lobes.

There are a series of separate, parallel frontal-subcortical circuits that are believed to link-specific areas of the frontal lobe to areas within the basal ganglia and thalamus. Alexander *et al*² first described the parallel organisation of circuits linking the basal ganglia and frontal cortex. Each circuit has a specific behavioural disorder where executive function deficits occur with lesions of the dorsolateral prefrontal circuit, disinhibition with lesions of the orbitofrontal circuit, and apathy with injury to the anterior cingulate circuit.³ In our case, we believe that the symptoms of abulia were mostly due to damage of the fronto-subcortical circuits which occurred following an episode of cardiac arrest. This was supported by the CT scan findings of the brain which showed periventricular hypodensity.

Namekawa *et al* have discussed a case of abulia in a 68-year-old man who presented with acute-onset somnolence without hemiparesis, dysarthria or sensory disturbance. Brain MRI showed a bilateral capsular genu infarct without involvement of the inferior thalamic peduncles and single-photon emission CT (SPECT) showed hypoperfusion in the bilateral frontal cortex. The cause of somnolence and abulia was considered to be disconnection of the thalamofrontal projection at the genu of the internal capsules.⁴ Samson *et al*⁵ discussed a case of a 62-year woman with bilateral caudate nuclei infarcts who was treated for depression for 2 years but without any result and was later diagnosed with abulia and successfully treated with bromocriptine. Cairns *et al*⁶ studied a case of a third ventricle cyst causing lateral pressure on the medial thalami and thus producing the disorder.

Abulia is associated with damage to the frontal-subcortical circuits linking the anteromedial frontal lobe to the thalamus and basal ganglia.^{7 8} This results in damage to the dopaminergic system, and thus drugs that increase the dopamine levels in the system are used as a possible treatment for abulia.⁹ Drubach *et al*¹⁰ have discussed four cases of abulia which improved with treatment of carbidopa/levodopa. Amantadine has also been successfully used for treatment of abulia.¹¹ Corcoran *et al*¹² described a significant clinical response in three cases of apathy following treatment with bupropion, a dopamine reuptake inhibitor, even when dopamine agonists had failed. Use of bromocriptine, a dopamine agonist, in the treatment of abulia has been reported in a number of published cases.⁹ Echiverri *et al*¹³ reported four akinetic and mute patients who were successfully treated with bromocriptine. Crismon *et al*¹⁴ reported specific improvement in spontaneity, articulation of speech and intelligibility in three patients following treatment with bromocriptine. Powell *et al* treated six men and five women with a

history of traumatic brain injury or a subarachnoid haemorrhage. Bromocriptine improved motivation and cognitive function but not mood.¹⁵

Reynolds *et al*¹⁶ conducted a retrospective cohort study to compare the outcome of comatose post-arrest patients treated with neurostimulants (methylphenidate and/or amantadine) with a matched control group and found that patients receiving neurostimulants had improved rates of following commands, survival to hospital discharge and improved distribution of cerebral performance category and modified Rankin scale scores. Their conclusion was to consider neurostimulants to stimulate wakefulness in selected post-cardiac arrest patients. Our patient, in line with the above results, showed excellent response to neurostimulants post-cardiac arrest.

Abulia is often difficult to diagnose in the elderly where symptoms are confused with conditions such as post-stroke depression, in which patients have a persistently sad mood with negative thought content. Such patients may have a history of depression. Hence, in the elderly, it becomes necessary to perform a thorough neurological and psychosocial assessment to rule out any underlying cause.¹⁷

In conclusion, clinicians should keep in mind abulia as a differential diagnosis in brain-injured patients with lesions in the basal ganglia, thalamus, frontal lobe or cingulate gyrus. Treatment could include trials of bromocriptine, commencing with a low dose and gradually increasing to the full dose depending on the response.

Learning points

- ▶ Abulia means a lack of will, initiative or drive.
- ▶ To consider abulia as a differential diagnosis in brain-injured patients with lesions in the basal ganglia, thalamus, frontal lobe or cingulate gyrus.
- ▶ It is important to differentiate abulia from depression as antidepressants do not seem to be effective.
- ▶ Abulia should be considered as a possible diagnosis in patients with cognitive and behavioural dysfunction following cardiac arrest, and a therapeutic trial of dopaminergic agents considered.

Competing interests None declared.

Patient consent Obtained.

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