Key Words: akinetic mutism, frontal–subcortical networks, neurorehabilitation, stimulant therapy

Akinetic Mutism Following Bilateral Infarcts Associated with a Mitral Valve Papillary Fibroelastoma

By Stanley Lyndon

ABSTRACT *~ Akinetic mutism is a rare but important clinical syndrome characterised by a profound decrease in goal-directed behaviour and speech output, yet with preservation of consciousness. This report describes the case of a 58-year-old male with a background of hypertension, alcohol use disorder, smoking, and MTHFR C677T homozygosity who experienced two ischaemic strokes within weeks of each other. The initial infarct involved the right thalamus, posterior putamen, external capsule, and subcortical anterior frontal lobe. Approximately five weeks later, he developed a second stroke in the left hemisphere, eventually leading to the identification of a mitral valve mass consistent with a papillary fibroelastoma. Following surgical resection of this mass, the patient demonstrated severe reductions in spontaneous movement and speech in a pattern consistent with akinetic mutism. He ultimately improved with rehabilitative measures and the initiation of a stimulant medication. This case highlights the intricate relationship between bilateral frontal–subcortical network injuries and the development of akinetic mutism.*
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Introduction

Akinetic mutism (AM) is a fascinating clinical condition first characterised by a profound lack of motor initiation (akinesia) and diminished speech (mutism). Despite the marked reduction in externally driven behaviour, patients often retain a notable level of consciousness, as manifested by appropriate eye movements, some degree of visual tracking, and apparent awareness of the environment. This apparent paradox between alertness and an inability to initiate movement or speech has captured interest for decades. Although AM can result from hydrocephalus, bilateral anterior cerebral artery territory infarcts, or lesions to the mesial frontal cortex, several other anatomic structures have also been implicated, including the basal ganglia, thalami, supplementary motor area, and cingulate cortex. This emphasises the diversity of possible lesion locations, all converging on frontal–subcortical circuits crucial for motivated and goal-directed behaviour.

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In the context of cerebrovascular disease, AM may develop due to extensive bilateral involvement of frontal–subcortical pathways. While smaller lacunar infarcts may typically produce focal motor or sensory deficits, the sum of repeated or multifocal lacunar insults to strategic locations can yield more global and complex syndromes, including AM. When observed clinically, differentiating AM from aphasia, catatonia, and severe depression can be challenging, but is essential in guiding treatment.

This report details the clinical course of a 58-year-old man who initially presented with a right-sided subcortical stroke and was later found to have a left-sided stroke, leading to the identification of a papillary fibroelastoma on his mitral valve. After surgical resection of this mass, the patient exhibited features consistent with akinetic mutism. This was successfully treated with a stimulant and rehabilitation.

Case Report

Patient Background

The patient is a 58-year-old male with MTHFR C677T homozygosity, a history of hypertension, alcohol use disorder, and long-term smoking. Prior to day 0, his only significant health issues were hypertension, active smoking, and alcohol use. He had no known valvular disease or arrhythmias such as atrial fibrillation. An echocardiogram in the past was reportedly normal, with an ejection fraction of 55%.

Presentation and First Stroke

The patient was found collapsed at home by family members. A non-contrast CT scan performed at an outside hospital revealed no large haemorrhage, though there was suggestion of a possible remote lacunar infarct. A CTA demonstrated moderate focal stenosis of the right internal carotid artery (ICA). Subsequent MRI revealed a subacute infarct in the right thalamus, posterior putamen, external capsule, and subcortical anterior right frontal lobe. Clinicians attributed these findings to small vessel disease, compounded by his vascular risk factors.

The patient was not deemed a candidate for intravenous thrombolysis (tPA). A transthoracic echocardiogram (TTE) showed no valvular disease and preserved systolic function (ejection fraction 55%). Telemetry did not reveal atrial fibrillation. He was started on dual antiplatelet therapy (aspirin and clopidogrel) but was later switched to aspirin 325 mg daily. His neurological examination at that stage showed drowsiness, delayed responses, attentional deficits, but intact naming. There was a mild neglect phenomenon, a rightward gaze preference, and mild left

nasolabial fold flattening. Motor examination disclosed pronounced left upper extremity weakness and marked left lower extremity weakness (almost complete lack of movement except for trace toe movement), accompanied by pathological reflexes (extensor toe response, clonus). These deficits were congruent with the right-sided infarct.

By day 16, he was transferred to a rehabilitation hospital. He remained somewhat drowsy, with delayed processing and mild neglect, but was engaged in therapies to regain left-sided strength and improve cognitive function.

Second Stroke (Around Day 35)

On approximately day 35 of his rehabilitation stay, the patient's speech became noticeably worse, and he developed new deficits on the right side. These new symptoms included right facial weakness and right upper extremity dysmetria, as well as reports of confusion and difficulty eating. He denied additional left-sided weakness or new sensory complaints. MRI completed soon thereafter (around day 39) revealed an acute stroke in the left periventricular white matter and corona radiata region. A transoesophageal echocardiogram (TEE) uncovered a mitral valve mass measuring approximately 6 mm on the posterior leaflet, suspicious for a papillary fibroelastoma, myxoma, vegetation, or a metastatic lesion. Given this finding, he was transferred to a tertiary centre for further cardiac surgical evaluation.

Preoperative Status

Prior to surgery, the patient was awake but had evolved deficits in speech and motivation. On neurological examination, he had a right gaze preference, a right lower facial droop, and significant right-sided weakness with only minimal withdrawal to noxious stimuli. He continued to follow commands better with his left upper extremity and displayed relatively preserved left-sided strength. However, his overall language output declined; he could produce only grunting sounds, though he did demonstrate comprehension by squeezing hands to answer questions. This pattern began to raise concern for a superimposed global aphasia or possible akinetic mutism, especially in the context of bilateral infarcts involving key frontal–subcortical circuits.

Surgical Intervention (Around Day 60)

On day 60, the patient underwent resection of the anterior mitral valve papillary fibroelastoma, alongside coronary artery bypass grafting

(CABG x1) and left atrial appendage (LAA) ligation. The procedure was uneventful from a surgical standpoint, and postoperative imaging did not show any new large infarcts or haemorrhages. However, his neurological exam in the days following surgery showed pronounced immobility and mutism. He was awake and alert, as evidenced by spontaneous eye opening and visual tracking, but persisted with very limited speech and almost no volitional movement, especially on the right side.

Postoperative Neurological Course

Around one week after surgery (around day 67), he continued to show global weakness that was more severe on the right side than the left, with persistent dysarthria and minimal speech output. During physiotherapy sessions, he showed inconsistent but definite capacity for movement on the left, enough to scratch his face or wipe away tears. On repeated examinations, he had a spastic catch in the right arm, and occasionally withdrew that arm to painful stimuli. His reflexes were brisk on the right side, with an upgoing toe, whereas the left side demonstrated more normal tone and less hyperreflexia. Sensory responses to light touch were present bilaterally.

From days 70 to 75, there was growing concern for akinetic mutism. The patient's level of consciousness was clearly above that of a comatose or severely encephalopathic individual, but he rarely initiated speech or movement. He would sometimes repeat single words or short phrases with extensive encouragement, and occasionally answer "yes" or "no" out loud. Upon being offered a handshake, he would extend his left hand, indicating some capacity for reactive movements. There was no evidence of catatonia per se, as he was reactive to certain stimuli and was not displaying classic catalepsy or echophenomena. Further, he did not exhibit emotional flattening; rather, he occasionally became tearful, suggesting that the emotional sphere remained intact. The possibility of a depressive disorder was also considered, but his neuroanatomical lesions and partial though inconsistent responses were more suggestive of a frontal–subcortical motivational deficit.

Clinical Diagnosis of Akinetic Mutism and Treatment

The pattern of alertness combined with minimal motor behaviour and speech raised the clinical suspicion of akinetic mutism. He was started on a trial of a stimulant (methylphenidate) with the goal of enhancing dopaminergic–noradrenergic tone and improving his drive to move and speak. Within a day, he demonstrated notable improvements in his ability to communicate. Initially limited to nodding and shaking his

head and verbalising "yes" and "no," he quickly progressed to forming more words. By the second day, he began producing short, whispered verbal responses, and within a few days, he was forming full sentences during conversations. This rapid development in speech was highly encouraging, and by day 85, his speech was significantly more coherent, with improved fluency and clarity.

During this period, physiotherapists observed that he actively participated in up to 75% of one-step commands, demonstrating the ability to initiate movement in the left upper and lower extremities and even wiggle his right toes or ankle. While his progress was steady, it was a clear indication that enhancing dopaminergic pathways was aiding in overcoming motivational deficits.

By day 85, he continued to display spasticity in the right arm and modest improvement in right-sided strength. He occasionally moved his right arm or leg on command and lifted his left arm to scratch his face. Muscle tone on the right side remained elevated, with an extensor plantar response, whereas the left side exhibited relatively normal tone.

Intensive neurorehabilitation, including speech therapy, occupational therapy, and physiotherapy, was ongoing, with a focus on enhancing language output and initiating movement. These efforts supported his continued recovery and provided hope for further functional improvements.

Discussion

Akinetic mutism is characterised by an absence of voluntary movement (akinesia) and a marked reduction in speech (mutism), despite preservation of wakefulness and awareness.[1] Patients frequently exhibit intact visual tracking and exhibit some responses to strong external stimuli. This dissociation between alertness and lack of activity can be unsettling and often leads to diagnostic confusion with conditions such as aphasia, catatonia, locked-in syndrome, or severe depression.[2] However, in akinetic mutism, patients typically retain the capacity to move or speak under certain conditions yet fail to initiate those actions spontaneously.

Neuroanatomical substrates of akinetic mutism include damage to the supplementary motor area, anterior cingulate cortex (ACC), basal ganglia, thalamus, and associated dopaminergic pathways.[3] The ACC and basal ganglia, for instance, are integral in translating motivation into purposeful action. When these pathways are compromised, the individual may possess the raw ability to move or speak, but the motivational drive to execute such actions is diminished. In the case presented, bilateral involvement of subcortical structures (including the right thalamus

and putamen, and subsequently left corona radiata) likely disrupted parallel cortical–subcortical loops in both hemispheres.[4]

Clinically, it can be challenging to diagnose akinetic mutism, especially when the patient has underlying aphasia, confusion, or a mood disorder. In this case, mild neglect and dysarthria also complicated the presentation. Nevertheless, the noticeable improvement with stimulant therapy—alongside occasional spontaneous or reactive movements—strongly supports a motivational deficit. The presence of tearfulness and partial cooperation further suggested that his emotional capacity was not entirely blunted, distancing the picture from catatonia or severe depression.

Another pivotal feature of this case was the detection of a mitral valve papillary fibroelastoma. These benign cardiac tumours are often asymptomatic and are most commonly identified incidentally on imaging.[5] However, they can occasionally lead to cardioembolic events. Although small vessel disease was initially suspected, the subsequent discovery of a mitral valve mass and a new left hemispheric stroke raised the suspicion of embolic phenomenon. Surgical resection of fibroelastomas is often considered, given their potential to cause recurrent strokes.[6] In this instance, the patient underwent successful removal of the mass, which likely eliminated a source of emboli but did not immediately resolve the significant deficits accrued from bilateral infarcts.

Long-term management of akinetic mutism relies on aggressive rehabilitation, addressing underlying causes, and in some circumstances, pharmacological interventions.[7] Dopaminergic or dopaminergic–noradrenergic agents such as amantadine and methylphenidate can promote improvement in motivation, speech, and motor initiation.[8] Ultimately, the degree of recovery can vary widely based on the extent of the brain lesions and the patient's comorbidities.

Conclusion

This case underscores several key considerations in stroke management and outcomes. First, despite an initial suggestion of small vessel disease as the culprit for a right subcortical stroke, a second event on the contralateral side prompted further investigation, revealing a mitral valve papillary fibroelastoma. Cardioembolic sources should always be thoroughly evaluated, especially when stroke patterns are unexplained or recurrent in different vascular territories. Second, akinetic mutism can arise from bilateral frontal–subcortical network damage, leading to striking reductions in movement and speech despite relatively intact consciousness. Recognising and differentiating akinetic mutism from aphasia, depression, or catatonia is vital to ensure optimal rehabilitation

and, where relevant, to trial specific agents that may enhance motivational drive. Finally, the patient's improvement with stimulant therapy and dedicated physiotherapy illustrates the potential for meaningful recovery in such complex post-stroke states, albeit over a prolonged course.

In summary, patients with bilateral subcortical or frontal lesions may present with akinetic mutism, highlighting the need for clinicians to identify this condition early and tailor management accordingly. The discovery of an embolic source such as a papillary fibroelastoma further emphasises the importance of comprehensive stroke aetiology workup to prevent recurrent events. Through close neurological monitoring, intensive rehabilitation, and targeted medical therapy, these patients can achieve incremental but significant improvements in both motor function and communicative abilities. ✤

Patient Consent Statement

The patient provided verbal consent for this case report publication.

Conflict of Interest Statement

The author has no conflict of interest to disclose.

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