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A Case of Akinetic Mutism in Patient with Unilateral Anterior Communicating Artery Stroke

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ABSTRACT

Akinetic mutism, a rare neurological condition involving sensory, motor, language, behavior, and emotional changes, mimicking locked-in syndrome, should be considered in acute stroke patients presenting with speech deficits. This condition, although debilitating initially, does have prospects for recovery. In this case report, we present the case of a patient presenting to the ER for altered mental status, found to be in DKA, NSTEMI, suspected narcotic overdose, and unilateral acute ischemic stroke of the left anterior communicating artery (ACA), who was found to have Akinetic Mutism. In stroke patients presenting with aphasia and dysarthria, it is worth considering akinetic mutism in addition to Broca's and Wernicke's aphasia.

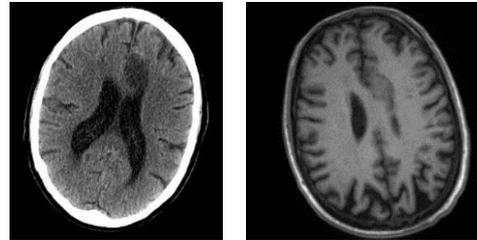
INTRODUCTION

Akinetic Mutism (AM), is a term designated by Cairns et al that was used to describe the syndrome of a patient with a third ventricle tumor who was conscious and alert, but had markedly reduced speech, gesture, and motor function. The patient's symptoms slowly improved after the cyst was decompressed. Akinetic Mutism includes two criteria – **lack of voluntary movement (akinetic) and lack of speech (mutism)**¹. Patients are awake and alert, but they do not possess motivation to perform tasks such as eating, drinking, or following commands. As a result, patients are often misdiagnosed with depression or locked-in syndrome.

AM is an extreme form of abulia¹ where patients lack willpower. This syndrome is not localized to any one specific region of the brain but can manifest from damage to various functional parts. In a paper by Arnts et al., the authors describe insults to parts of the brains causing AM including anterior cingulate gyrus, striatum, pallidial complex, and thalamus. Furthermore, Nagaratham et al describes AM presenting in strokes of the caudate nucleus, putamen, and thalamus. All of the aforementioned parts of the brain are involved in the neurocircuitry for behavior and motivation. Disruption in the functions and projection pathways to these structures cause causes the spectrum of symptoms found in akinetic mutism.

CASE REPORT

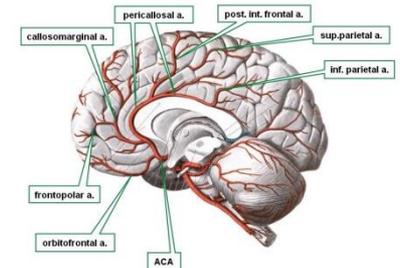
Patient was a 57 year old male with no prior medical history. EMS was called to hotel after patient's mother found him wedged between the bed and the wall. Patient had right sided weakness and complete aphasia; last known normal the night before. On presentation, patient was unable to move any extremities and had absent reflexes on the right side. Initial NIHSS was 27. CT found **acute left front lobe infarct** in the anterior cerebral artery (ACA) distribution. CTA showed **occlusion of the distal left ACA in the pericallosal segment**. Stat MRI showed acute infarction of the parasagittal posterior left frontal lobe in the ACA territory.



Patient's ER course was complicated by acute stroke, DKA, AKI, Rhabdomyolysis, NSTEMI, Septic shock from UTI. He was admitted to the ICU where his DKA was corrected and shock and rhabdomyolysis were treated. Hyponatremia was slowly corrected. Neurological exam by neurologist showed pupils equal and reactive to light, inability to move right upper and lower extremities, fasciculation and hypertonicity of the left arm. The neurologist noted that on speech exam, **patient had expressive aphasia, but did not exhibit the typical signs including hesitancy and frustration due to inability to speak**. It appeared that patient was not making any attempts to speak. Additionally, on daily exam, patient was able to demonstrate grip strength on left, however, at times, refused to grip. Patient was **exhibiting abulia, or a lack of will or motivation for speech, action, or thought**. This can be seen in ACA stroke distribution (as described by Nicolai et al), making this a case of **akinetic mutism**. Patient was provided speech, physical, and occupational therapy after his ICU stay. He was eventually discharged to a rehab facility. Due to loss in follow up, there was no reports on patient's condition until several months later when he appeared to his GI physician for feeding tube removal. In GI notes, they noted that he was able to speak.

DISCUSSION

Our patient demonstrated a stroke in the region of the left ACA at the level of the **pericallosal artery**, as found in the CTA. This artery supplies the **anterior cingulate cortex (ACC)** as well as the medial portion of the **primary motor cortex (PMC)**. The PMC infarction was unilateral, therefore, our patient had right-sided lower extremity weakness (and to some degree, upper extremity weakness).



Source: https://operativeneurosurgery.com/doku.php?id=anterior_cerebral_artery

Furthermore, because of the ACC insult, our patient also demonstrated lack of affect-regulation. The **anterior cingulate gyrus** serves as a bridge to the prefrontal cortex and the limbic system. The prefrontal cortex is responsible for cognitive functions while the limbic system is responsible for emotional functions. The ACC is responsible for translating an emotion from the limbic system and executing it to an actual behavior. In our case, the patient's ACC is unable to select the motivation for movement and speech and pass it to the motor centers due to the disruption of the connections. This lack of motivation in speech and movement is the crux of akinetic mutism. Unilateral dysfunctions to the anterior cingulate gyrus, which is the presumed location of our patient's stroke given the blood supply disruptions, only produces transient motivations dysfunctions and gradually recovers over time¹. Although various treatments for AM have been proposed including dopamine and GABA agonists, our patient did not require any medical treatment since his AM course slowly resolved with speech and physical therapy.

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