Acute mutism: a useful lesson

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Broca’s aphasia may manifest as mutism in some patients. Mutism may be misinterpreted as part of a depressive illness in patients with a psychiatric history. We report on a patient presenting with acute mutism who had a history ofamphetamine and cannabis abuse and was later found to have sustained an infarct of the dorsolateral frontal cortex. Recognition of this mode of presentation will aid early diagnosis and treatment.

A 40-year-old right-handed man presented to casualty with a 3-day history of mutism. He had had an altercation with his partner and had left the house to stay with a friend. Later that evening he was found mute, sitting on the sofa having been incontinent of urine. During the next two days he was cared for by his partner but remained mute, with minimal interactions with others.

He had a history of depression since April 2007, following the death of his mother. He had been low in mood and consuming large quantities of amphetamine (1 ounce per week) and diazepam (100 ‘street’ tablets per week), smoking cannabis and drinking eight cans of lager a day. A few weeks before admission he had tried to overdose with diazepam. He had a history of self-harm and drug overdoses. He had been diagnosed with anti-social personality disorder and had a forensic history of domestic violence. There was no relevant past medical history. There was a family history of depression. He smoked 20 cigarettes per day.

On admission to casualty he could not communicate verbally but could respond to questioning through gesture. His temperature was 37.2°C, blood pressure 128/78 mm Hg and pulse 78 beats per minute and regular. His score on the Glasgow Coma Scale was 15/15, and on initial assessment no evidence of head injury or abnormal neurological findings had been reported. Toxicology screening of urine was positive for cannabis and benzodiazepines. Blood investigations revealed neutrophilia (11.5×10⁹/l) and leucocytosis (14×10⁹/l) with normal inflammatory markers and no other abnormalities.

He was referred to psychiatric services with mutism thought to be secondary to a depressive illness. He was communicating with the staff through gestures and writing. Objectively his mood was low but reactive. Thoughts, perception, cognition, judgment and insight were difficult to assess due to his mutism. Formal neuropsychological assessment was not permitted, because of difficulties in communication. During his admission he started to produce a few words with perseverance of speech. This prompted an urgent CT brain scan and referral to neurology services.

Further neurological examination revealed normal phonation, partial receptive dysphasia and complete expressive dysphasia. He had right upper motor neuron facial weakness, right upper limb dyspraxia, right hemi-neglect and right-sided pathologically brisk reflexes. Both plantar responses were flexor. Sensory examination was not possible and no cerebellar or extrapyramidal signs were detected. The general examination was normal, with no carotid bruits observed.

The CT brain scan showed a 3.7 cm×3.9 cm low attenuation area in the left dorsolateral frontal cortex (figure 1A). Subsequent MR imaging confirmed a regional infarct in the supply area of the left middle cerebral artery, with a trace of haemorrhagic component (figure 1B). MR angiograms of both the extracranial and intracranial major vessels did not reveal any abnormalities. An autoimmune screen was negative.

This patient presented with acute mutism secondary to a dorsolateral frontal cortex infarct, likely related to cerebral vasculopathy as a result of chronic amphetamine and cannabis abuse. The patient was given aspirin and simvastatin and was advised to stop smoking and using illicit drugs. He was referred to speech and language services and has since made a good recovery of his speech. He is continuing psychotherapy and support from the substance abuse services.

Acute mutism may occur in both organic and non-organic disease and may cause diagnostic difficulties. Important organic causes include head injury, encephalitis and lesions affecting the dorsolateral frontal cortex, causing Broca’s aphasia. In this case, Broca’s aphasia was caused by infarct attributable to misuse of amphetamine and cannabis, both of which have been implicated in stroke independently,1 2 causing severe hypertension and vasospasm.3 Arterial dissection can occur in amphetamine abuse.4 Expression, naming and production of spontaneous speech are affected, resulting in non-fluent aphasia, with telegraphic speech and agrammatism. Comprehension appears relatively spared. Hypophonia, limb apraxia and hemiparesis may be present.

Figure 1 CT brain scan image (A) showing a large hypodense area in the left dorsolateral frontal cortex (Broca’s area due to cerebral infarction, with hyperdensities representing haemorrhagic changes in the centre (white arrow). The same is confirmed on a T2 axial MRI (B), which revealed slight posterior extension of the infarct.
Mutism in psychiatric disease may co-present with catatonic signs of negativism, stereotypy, posturing or stupor. A published review and case series of 22 patients with mutism ascertained that nine had an affective disorder, seven schizophrenia, two personality disorder and four an organic cerebral cause. Stroke was the commonest organic cause in the series. In that case series, features suggesting an organic aetiology included irregular respiration, abnormal pupillary responses, roving eye movements, facial weakness and exaggerated jaw jerk. Resistance to eye opening suggested non-organic aetiology. Catatonic features did not distinguish between organic and non-organic mutism. The presence of urinary incontinence in this case suggested an organic aetiology, although incontinence may occur in non-organic catatonic states.

Patients presenting to the emergency department with acute mutism must be assumed to have an organic pathology irrespective of their psychiatric history. Thorough neurological assessment and urgent brain imaging will prevent delay in ascertaining the cause of mutism and aid early treatment.

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Accepted 21 July 2009

Competing interests None.

Patient consent Obtained.
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Emerg Med J published online July 20, 2010

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