Mutism with abulia for contralateral function: a case of acute left anterior cerebral artery territory stroke

Introduction

This article describes a case of an acute left anterior cerebral artery territory stroke presenting as mutism with abulia for contralateral function. The brain imaging demonstrated an acute left parasagittal infarct associated with focal narrowing of the A3 segment of the left anterior cerebral artery. The images highlight the location of the acute ischaemic lesion and associated anterior cerebral arterial disease, and their role in the aetiology of the presenting symptoms and signs is discussed.

Discussion

Anterior cerebral artery territory infarction is known to cause mutism, akinesia and abulia in addition to contralateral weakness (Kang and Kim, 2008). Lesions of the cingulate gyrus or supplementary motor area within the anterior cerebral artery territory give rise to these symptoms and signs directly or by interrupting frontal-subcortical neural circuits.

Many structures within these circuits receive dopaminergic innervation and it has been suggested that ischaemic perturbation of the subcortical dopaminergic system may play an important role in producing extrapyramidal features which may then improve with dopamine agonists (Yang et al, 2007).

Nagaratnam et al (2004) suggested that unilateral anterior cerebral artery territory lesions give rise to transient akinetic mutism, independent of the lesion being right- or left-sided. Other studies have also shown urinary incontinence to be unrelated to lesion localization (Kang and Kim, 2008). However, Kumral et al (2002) demonstrated different clinical patterns depending on lesion side with left-sided infarction producing mutism, dysphasia and hemiparesis; right-sided infarction being associated with hemineglect in addition to hemiparesis; and bilateral infarction presenting with aki-

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Figure 1. a. Axial fluid-attenuated inversion recovery and (b) diffusion-weighted magnetic resonance imaging demonstrating subcortical white matter hyperintensities, previous lacunar infarcts (arrow 1) and an acute infarct in the parasagittal left frontal lobe (arrow 2). c and d. Magnetic resonance angiography showing a severe short segment narrowing of the A3 segment of the left anterior cerebral artery (arrow 3).

Case Report

A 74-year-old right-handed Nigerian woman, with pre-existing hypertension treated with amlodipine and bendrofluazide, was brought to the emergency department by her daughter having woken up mute and lacking in initiation. She had two episodes of urinary incontinence but no other symptoms. Despite a history of poor attention and concentration, she was previously independent, and did not smoke cigarettes or drink alcohol. There was no other previous medical history, drug history or family history of note.

On examination, she had no receptive dysphasia but was mute. She displayed spontaneity for left-sided actions but was observed to lack volitional right-sided functions both spontaneously and to command. There was a 'lead-pipe' increase in tone in the right upper limb and an extensor right plantar response but no other neurological deficits. Her blood pressure was 143/82 mmHg. Physical examination and laboratory investigations were otherwise unremarkable. Magnetic resonance brain imaging demonstrated evidence of previous lacunar infarction, multifocal vascular narrowing and an acute left parasagittal infarct, involving the cingulate gyrus, associated with severe short segment narrowing of the A3 segment of the left anterior cerebral artery (*Figure 1*). No extracranial stenosis or source of cardioembolism was found on magnetic resonance angiography, Duplex ultrasound or cardiac investigations. The patient was treated with antiplatelet therapy and within 36 hours returned to baseline.

netic mutism, sphincter dysfunction and dependent functional outcome. In this case, there was a lack of spontaneity associated with neurological functions only contralateral to the side of the lesion, supporting a focal interruption to frontal-subcortical circuitry.

Abulia with mutism may be erroneously diagnosed as depression or delirium, which may be associated with stroke. Distinguishing between them is important as treatment differs and diffusion-weighted magnetic resonance imaging is useful to locate and delineate acute ischaemic lesions within the anterior cerebral artery territory. Intracranial large artery disease is the most important stroke aetiology in anterior cerebral artery infarction (Kang and Kim, 2008) with magnetic resonance angiography, as in this study, proving most helpful in diagnosis. It is becoming increasingly apparent that there is considerable ethnic variation in the prevalence of intracranial arterial disease which is more common in black individuals with ischaemic stroke such as this patient (Markus et al, 2007). BJHM

Kang SY, Kim JS (2008) Anterior cerebral artery infarction: stroke mechanism and clinical-imaging study in 100 patients. *Neurology* **70:** 2386–93

Kumral E, Bayulkem G, Evyapan D, Yunten N (2002) Spectrum of anterior cerebral artery territory infarction: clinical and MRI findings. *Eur J Neurol* **9:** 615–24

Markus HS, Khan U, Birns J et al (2007) Differences in stroke subtypes between black and white patients with stroke: the South London Ethnicity and Stroke Study. *Circulation* **116:** 2157–64

Nagaratnam N, Nagaratnam K, Ng K, Diu P (2004) Akinetic mutism following stroke. *J Clin Neurosci* **11:** 25–30

Yang CP, Huang WS, Shih HT et al (2007) Diminution of basal ganglia dopaminergic function may play an important role in the generation of akinetic mutism in a patient with anterior cerebral arterial infarct. *Clin Neurol Neurosurg* **109:** 602–6

Stumbling into encephalopathy: chest trauma in a patient with alcoholic cirrhosis

A middle-aged man with biopsy-proven alcohol-related liver cirrhosis was admitted to hospital with right-sided chest pain. He was haemodynamically stable with oxygen saturations of 96% on room air. He had reduced air entry on auscultation at his right lung base, which was stony dull to percussion.

The admission chest X-ray (*Figure 1*) shows extensive surgical emphysema with the right eighth and ninth ribs fractured posteriorly and a moderate right effusion. A computed tomography scan (*Figure 2*) shows a fractured ninth rib that is diastased with significant intrathoracic displacement.

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Despite draining the haemothorax, the patient became pyrexial a few days later with a white cell count of $22x10^9$/litre and C-reactive protein of 183 mg/litre. Repeat imaging showed a loculated empyema. Unfortunately, he became encephalopathic, developed multiorgan failure and died.

Since trauma is associated with alcohol-related intoxication (Israel et al, 2006) physicians should be aware of the increased mortality and risk of bleeding in the patient with advanced liver disease. BJHM

Israel Y, Orrego H , Schmidt W et al (2006) Trauma in cirrhosis: An indicator of the pattern of alcohol abuse in different societies. *Alcohol Clin Exp Res* **15**(3): 433–7

Figure 1. The chest X-ray shows extensive right-sided surgical emphysema (arrows) with the right eighth and ninth ribs fractured posteriorly and a moderate right effusion.

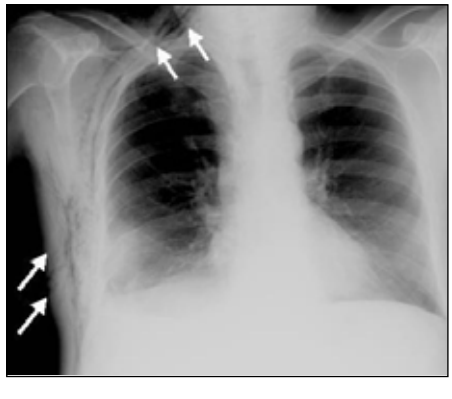

Figure 2. The transaxial computed tomography scan shows a fractured ninth rib that is diastased with significant intrathoracic displacement (arrow a). The liver contour appears irregular in keeping with cirrhosis. A right-sided haemopneumothorax secondary to multiple rib fractures, with extensive subcutaneous emphysema (arrows b), was seen in the remaining computed tomography scan.