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Letters to the Editor

Acute transient aphemia



Dear Editor,

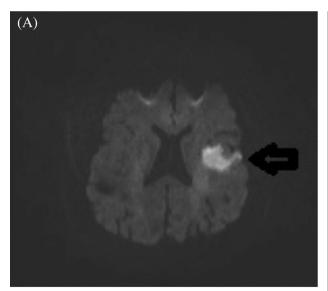
Some years ago in *Clinical Neurology and Neurosurgery*, Fox et al. [1] described a fascinating case of isolated aphemia – loss of the ability to speak with completely preserved understanding and writing. The patient of Fox et al. started speaking single words after one day with further improvement during the hospital stay progressing to mild residual dysarthria on telephone follow-up some months later. We here present a case not dissimilar to the one of Fox et al., but with an even more dramatic recovery.

A 57-year-old left-handed female with past medical history of non-insulin dependent diabetes, two cerebral vascular accidents, hypertension, depression, primary inflammatory myositis, rheumatoid arthritis and aortic valve repair was presented with sudden loss of speech. The patient was rushed to the hospital within the hour where she was found to have a profound speech deficit being unable to speak at all. Specifically she was not able to phonate.

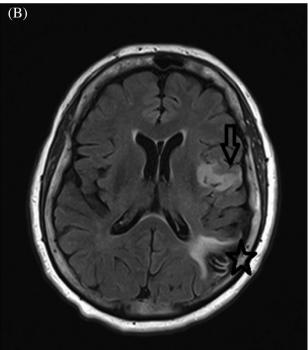
She was able to nod and shake her head appropriately to questions. She was also able to follow complex commands and to write her name and names of objects. The patient was not apraxic. CT scan showed a stable, moderately sized left middle cerebral artery infarct with no sign of acute intracranial hemorrhage. Tissue plasminogen activator (tPA) was not administered. Restricted diffusion MRI image showed an acute left middle cerebral artery (Fig. 1A) infarct involving the frontal lobe. Fig. 1B shows the MRIT2 weighted image that demonstrates the new infarct and old left middle cerebral artery gliosis.

The patient began to show signs of recovery roughly 24 h after admission, beginning with one word repetitions and being able to state the name of the hospital. Her recovery rapidly progressed on day three after admission to almost fluent speech, nearly back to baseline as per family. No likely source of stroke could be identified and the episode was characterized as a cryptogenic, recurrent, and embolic CVA.

Aphemia was the present finding with intact comprehension and writing, as in the patient of Fox et al. [1], but contrast to an



Restricted diffusion image MRI indicates new infarct (arrow).



MRI T2 weighted image. Vertical arrow indicates new infarct. Star indicates gliosis from old infarct.

Fig. 1. (A) Restricted diffusion image MRI indicates new infarct (arrow). (B) MRI T2 weighted image. Vertical arrow indicates new infarct. Star indicates gliosis from old infarct

aphasia in which there are deficits of written as well as spoken language. Interestingly, aphemia was first described by Broca [2] predating his description of the eponymous aphasia. Aphemia has been described in lesion to the left frontal operculum or left precentral gyrus. While aphemia certainly can be persistent [3], curiously, as in our case, that of Fox et al. [1] and another [4] aphemia can show significant recovery within days, while complete recovery of severe aphasia is seen in less than 10% of cases and then only after some weeks at the least [5]. This difference in recovery from the two deficits may warrant further study.

Competing interests

None.

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Dual anticonvulsant and neuroprotective effects of therapeutic hypothermia after status epilepticus



Sir,

We read with a great interest the recent and innovative article by Bennett et al. [1]. Their report is original and at times thoughtprovoking, notably regarding the use of therapeutic hypothermia for super refractory status epilepticus.

Patients with status epilepticus require the symptomatic measures usually taken in the emergency department, prehospital

emergency medical system or in the neurological intensive care unit. Antiepileptic treatment appropriate for the electrical and clinical pattern in the patient should be initiated at scene on an emergency basis. In addition to these symptomatic measures, the treatment strategy should include rigorous aetiological investigations for a cause to the status epilepticus, followed by etiological treatment. However, the existence of functional impairments in half the survivors of convulsive status epilepticus requiring ICU management [2] indicates a need for neuroprotective interventions.

Therapeutic hypothermia has been reported to exert not only anticonvulsant but also neuroprotective effects [3] in studies of rats with self-sustaining status epilepticus. Hyperthermia between 39.5 °C and 42 °C promoted the occurrence of seizures and exacerbated local and hippocampal neuronal damage, whereas cooling to 32.5 °C significantly decreased the occurrence of such damage. Hypothermia between 28 °C and 31 °C decreased the incidence of epileptic activity and associated neuronal lesions. After pre-treatment with mild hypothermia (32-34°C), the rats were protected from the onset of status epilepticus (increased latency to onset of seizures and time to onset of status epilepticus) and also had fewer apoptotic neurons in the hippocampus. Another study has shed light on the pathophysiological mechanisms involved in the neuroprotective effect of hypothermia. Moderate hypothermia (32-34°C) was associated with regulation of the expression of the GluR1 and GluR2 subunits of the AMPA glutamate receptors, an effect that decreased the number of necrotic or apoptotic hippocampal neurons. Furthermore, cerebral oedema induced by ongoing seizures was decreased by the use of therapeutic hypothermia immediately after status epilepticus induced by kainic acid. All these effects were potentiated by the concomitant administration of anticonvulsant benzodiazepine therapy. However, hypothermia was not effective to prevent epilepsy after status epilepticus.

In humans, therapeutic hypothermia has been used as an adjuvant treatment in refractory epilepsy and during neurosurgery. Successful treatment with hypothermia has also been reported in paediatric patients with severely refractory status epilepticus. Finally, therapeutic hypothermia may also target some causes of status epilepticus such as ischaemic or haemorrhagic stroke, subarachnoid haemorrhage, and even traumatic brain injury [4]. Conversely, the use of therapeutic hypothermia in severe bacterial meningitis has been demonstrated to increase the risk of death [5].

In conclusion, whether therapeutic hypothermia is beneficial in humans with status epilepticus remains to be demonstrated. Pathophysiological considerations and experimental data suggest a role for therapeutic hypothermia. A French multicentre randomised study for which patient recruitment is ongoing can be expected to clarify this point (HYBERNATUS, NCT01359332). Until the results are available, all patients with status epilepticus may benefit from non-specific measures aimed at preventing secondary brain damage, including at least targeted temperature management. The use of therapeutic hypothermia must still be restricted to severely refractory status epilepticus, in the absence of contraindications.

Financial support

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Conflict of interest

We declare no conflict of interest.

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