

Letter to the Editor

Bilateral apraxia of eyelid closure following bilateral subcortical frontal infarcts



ARTICLE INFO

Keywords:

Apraxia

Stroke

Eyelid closure

1. Background

While apraxia of eyelid opening accompanies several neurological conditions, apraxia of eyelid closure has been reported less frequently. Apraxia of eyelid closure is impaired voluntary eye closure with normal language comprehension and level of alertness, and absent ocular pathology. It has been previously reported in progressive supranuclear palsy, vascular progressive supranuclear palsy, amyotrophic lateral sclerosis, Creutzfeldt-Jakob's disease, Huntington's disease, post encephalitic parkinsonism, and following non-dominant frontal or parietal lobe strokes [1–9]. Here we report a case of apraxia of eyelid closure following bilateral subcortical frontal infarcts.

2. Case report

A 90-year-old right handed man was admitted for failure to thrive. He had a past medical history significant for hyperlipidemia, hypertension and aortic stenosis status post transcatheter aortic valve replacement 9 months prior to presentation. His hospital course was complicated by enterococcus faecalis bacteremia from an unknown source. Several days after starting intravenous antibiotics, he became difficult to arouse. The neurological exam was remarkable for a left facial droop, left sided hemiparesis, and left sided hypoesthesia. The patient was able to follow axial and appendicular commands, such as sticking out his tongue and showing a thumbs-up on the right, but he was unable to voluntarily close his eyes despite effort and intact comprehension. He said "I'm trying" when asked to close his eyes. He could, however, maintain eye closure after he manually closed his eyes. His eyes remained closed during sleep. He was also able to close his eyes in response to perceived threats and with spontaneous blinking, and to open his eyes on command. The remainder of the cranial nerve exam was normal. There was no apraxia with other facial or appendicular tasks.

An MRI brain was remarkable for four punctate subcortical acute infarctions in the right frontal and temporal-occipital lobes, as well as the left frontal lobe, concerning for a cardioembolic source (Fig. 1). CT angiography of the head and neck showed a distal right middle cerebral artery occlusion with partial recanalization. A transthoracic echocardiogram was negative for vegetations. However, given the concurrent presentation of bacteremia and the presence of intracardiac hardware and multifocal strokes, the etiology was thought to be likely septic emboli and the patient was continued on long-term intravenous antibiotics at discharge. Following discharge phone contact was made with the patient's family, who reported that the eyelid closure deficit noted above improved approximately one month after discharge.

3. Discussion

Apraxia of eyelid closure results from impaired relaxation of the levator palpebrae superior muscle [10]. It has been postulated that a lesion in the corona radiata is responsible for the eyelid closure apraxia, secondary to disconnection between the primary and secondary motor cortices [7]. Three cases of unilateral apraxia of eyelid closure have been reported with contralateral frontal infarcts. Traditionally, it has also been seen in patients with non-dominant parietal lobe lesions as a manifestation of constructional apraxia [8]. Apraxia of eyelid closure has also been reported in patients with vascular progressive supranuclear palsy as a chronic manifestation of frontal subcortical small vessel ischemic disease [4]. However, our case is unique in that it was an acute manifestation of bilateral apraxia of eyelid closure in the setting of bilateral subcortical frontal infarcts. Bilateral apraxia of eyelid closure may be an under-recognized sign in hospitalized patients with subcortical frontal infarcts. Without careful examination, it may be confused with lethargy, delirium, or inattention.

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.jns.2018.01.036>.

Disclosures

The authors report no sources of funding or conflicts of interest. The authors do not have financial disclosures for the previous 12 months.

<https://doi.org/10.1016/j.jns.2018.01.036>

Received 15 December 2017; Received in revised form 19 January 2018; Accepted 28 January 2018

Available online 31 January 2018

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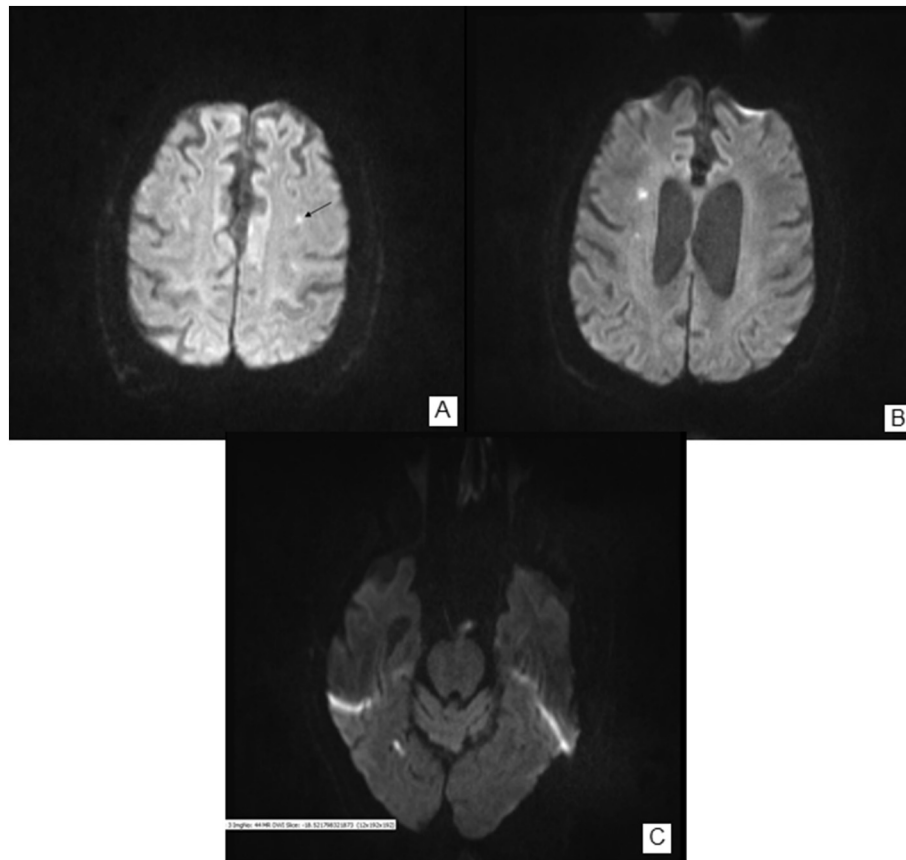


Fig. 1. Brain MRI with DWI B100 sequence shows increased signal in left frontal lobe (A), right frontal lobe (B) and right temporal-occipital lobe (C) concerning for multifocal acute infarctions.

Ethical compliance statement

We confirm that we have read the journal's position on issues involved in ethical publication and affirm that this work is consistent with those guidelines. The authors confirm that the approval of an institutional review board was not required for this work.

Author roles

E.A. writing the first draft, M.D. Review and critique.

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