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Poststroke Partial Seizures Presenting As Hemifacial Spasm

Hemifacial spasm (HS) is an intermittent involuntary rhythmic tonic or clonic contraction of facial musculature innervated by the ipsilateral facial nerve. Between the episodes of spasms, the face is either normal or slightly weak on the affected side.¹ Although usually there is no identifiable etiology, it is conventionally thought to be a peripheral process due to either compressive lesions of the facial nerve at the root exit zone (e.g., tumor, arteriovenous malformation), or noncompressive lesions (e.g., stroke, multiple sclerosis). It has been hypothesized that an ephaptic transmission results in facial nerve excitation and HS.² Anticonvulsants and botulism toxin are the mainstay of the treatment.² Here, we present the case of a patient who presented with right HS following a left cortical stroke but the cause was epileptic and the treatment involved anti-convulsants.

A 74-year-old, fully independent, right-handed male with history of atrial fibrillation and left cortical hemorrhagic stroke, presented with an acute right HS and worsening of his right hemiparesis starting the night before. Since his prior stroke 2 years ago he had been left with a mild right hemiparesis and a subtle dysarthria. The patient was taking 81 mg aspirin at the time of this presentation. He was seen in an outside hospital the night before where a computed tomography (CT) scan of his head was obtained and he was loaded with 1 g of phenytoin with no effect on his facial spasm.

On initial examination the patient was lying in bed in no distress with almost continuous spasm of the right upper and lower face interrupted only by brief intermissions lasting 1 to 3 seconds. Patient denied any associated pain. His comprehension, naming and repetition were preserved but his speech was dysarthric. When spasms were not present a right facial droop was noted along with a right pronator drift and hemiparesis, with motor strength at 4/5 on the left and 3/5 on the right.

Head CT scan and magnetic resonance imaging (MRI) of the brain demonstrated a left frontal cortical based, acute to subacute, intracerebral hemorrhage overlapping with a chronic bleed with several areas of hemosiderin deposition on gradient echo (Fig. 1).

Upon admission his phenytoin was continued (for seizure prophylaxis after intracerebral bleed) at 100 mg three times a day, with no effect on his HS. Video-EEG monitoring revealed continuous cycles of rhythmic ictal pattern lasting for 90 to 350 seconds interrupted only for 1 to 3 seconds during which the EEG showed a relatively suppressed background. The ictal activities started in the left central or central-frontal-temporal head regions. A high voltage rhythmic muscle activity was present on the right side. Clinically, these discharges were associated with his facial spasms with brief periods of quiescence in between (Fig. 2). These findings were suggestive of simple partial status epilepticus with the primary focus at the site of his hemorrhagic stroke.

Patient's HS was completely controlled after the free phenytoin level was increased from 1.4 to 1.9 mg/mL. Four days later his phenytoin was replaced with levetiracetam at

Published online 26 July 2007 in Wiley InterScience (www. interscience.wiley.com). DOI: 10.1002/mds.21660

500 mg twice a day. His HS remained well controlled during the rest of his hospital course and he was discharged to a subacute rehabilitation facility after one week. A second EEG, performed before discharge, did not show any epileptiform discharges.

There are other reports of HS following stroke. Ambrosetto and Forlani, as well as Kawakami et al., report such patients, but both cases involved pontine strokes, compressing the facial nerve due to edema.^{3,4} Schiess et al., present a case of right HS, in a patient with left ponto-medullary stroke.⁵ Arunabh et al., cite a caudate stroke inducing HS,⁶ but neither one of these cases had seizures. Forss et al., report a patient with history of partial seizures who developed left HS as reflex epilepsy provoked by stimulation of the left lower gums.⁷

A review of 1,500 patients with stroke showed a 3.7% incidence of poststroke movement disorders starting from one day to 10 months after stroke. Chorea was the most common condition among this group while there were no cases of HS.⁸ Our patient's neuroimaging demonstrates a discrete cortical based hemorrhage in the left precentral gyrus correlating to the



FIG. 1. A: Computed tomography demonstrating hemorrhage in the left frontal cortex. B: Gradient Echo (first row) and T2 weighted (second row) MRI images demonstrating subacute hemorrhage in the left frontal cortex.

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FIG. 2. EEG demonstrating the end of a seizure episode and facial spasm followed by a 3 second period of quiescence before the onset of a new cycle. A high voltage sharp discharge in the left central-frontal head area (max C3 and F7) is followed by a build up of rhythmic low voltage fast activity on the left. The muscle artifact on the right was always present during these events. This pattern repeated itself constantly (seizures lasting 90–350 second) and coincided with clinical episodes of right hemifacial spasm during which the patient remained awake and alert (simple partial seizures).

representation of the face. Presence of focal spikes in the left cortex on EEG, and response to antiepileptic medication, demonstrate that HS can, among other etiologies, present as a manifestation of simple partial seizures and status epilepticus following stroke.

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