# Herpes encephalitis : a stroke mimicker

#### Thein Win, Nida Maham and Sahayini Kumar

Department of Internal Medicine, Greater Baltimore Medical Center, Baltimore, MD, USA

#### ABSTRACT

Background: HSV-1 encephalitis (HSVE) usually presents with fever, altered mental status or focal seizures. Aphasia can also be a presenting symptom of HSVE but rarely occurs as the primary symptom. We present a case where aphasia was the primary presenting symptom of HSVE. Case: A 72-year-old physician with a history of hyperlipidemia and obstructive sleep apnea presented to the emergency room with sudden onset of speech difficulty lasting an hour. He did not have a fever, photophobia, neck stiffness, weakness, or numbness. The patient was brought in by the family within an hour to the emergency department. On exam, the only neurological deficit that was found was the use of inappropriate words in sentences and inability to name certain objects. He was diagnosed with an embolic stroke and received tPA. MRI brain that was done 24-hour post tPA showed an increased FLAIR and T2 signal hyperintensity within the medial left temporal lobe with slight effacement of the cysts sulci which was concerning for encephalitis. This was later confirmed by serology. The patient was started on IV Acyclovir and recovered fully after 3 weeks of acute neuro rehabilitation. Conclusion: Aphasia primarily is an unusual presentation of HSVE. It should be considered as one of the possibilities in patients presenting with features suggestive of a stroke involving the language areas of the brain.

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## 1. Introduction

HSV-1 encephalitis (HSVE) is the most common cause of fatal sporadic encephalitis in the USA, accounting for approximately 10 to 20 percent of the 20,000 annual viral encephalitis cases [1,2]. It usually presents with fever, altered mentation or focal seizure [3]. While aphasia is also a known symptom of HSVE it is usually accompanied by other symptoms as well. We present a case where aphasia was the primary presenting symptom of HSVE.

#### 2. Case

A 72-year-old physician with a history of hyperlipidemia and obstructive sleep apnea presented to the emergency room with sudden onset of speech difficulty lasting an hour. He was watching television at home when suddenly his wife noticed that his speech was not making sense despite fluency. The examples that she gave were that he stated he had problems for 6 days instead of 6 hours, he used the word Tuesday instead of Thursday and referred to his shoulder as 'mitochondrial'. The patient was not aware of his speech problem. He also complained of a frontal headache that had been going on for a few days. He did not have a fever, photophobia, neck stiffness, weakness, or numbness. The patient was brought in by the family within an hour to the emergency department.

In ED, he was afebrile, and the rest of his vitals were within normal limits. On physical examination, he was awake and alert. He was oriented to time, place, and person. His speech was fluent without evidence of dysarthria. However, he used incorrect words in sentences without being aware of using them. He also exhibited anomia. His pronunciations of words were incorrect multiple times and he also used incorrect word substitutions. For example, instead of saying Timonium he sounded like saying Tomonium, instead of brick table he said breck table, and for Saturday he said Tatterday. On National Institute of Health Stroke Scale images, he was not able to name a cactus, hammock, and was not able to describe a woman washing dishes. The rest of the neurological exam was normal. CT brain without contrast in the ED did not show any hemorrhage or evidence of an acute large territory of ischemia. He was seen by a neurologist in the emergency department and was clinically diagnosed as having an embolic stroke. He was started on tPA and transferred to the intensive care unit.

He continued to have aphasia and anomia in ICU. He remained afebrile and vitals remained stable. MRI brain was done as a workup for stroke, 24-hour post tPA. It showed an increased FLAIR and T2 signal hyperintensity within the medial left temporal lobe with slight effacement of the cysts sulci which was concerning for encephalitis (Image 1). After the MRI finding, a lumbar puncture was done, and the patient

**CONTACT** Sahayini Kumar skumar@gbmc.org Department of Internal Medicine, Greater Baltimore Medical Center, Baltimore, MD, USA © 2019 The Author(s). Published by Informa UK Limited, trading as Taylor & Francis Group on behalf of Greater Baltimore Medical Center. This is an Open Access article distributed under the terms of the Creative Commons Attribution-NonCommercial License (http://creativecommons.org/licenses/by-nc/4.0/), which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.



**Figure 1.** Initial MRI brain showing increased axial and T2 signal hyperintensity within the medial left temporal lobe with slight effacement of cysts sulci.

was started on Acyclovir. CSF exam showed WBC of 465 (64% lymphocytes, 35% monocytes and 1% eosinophil), RBC of 4, glucose of 54 and protein of 93.6. Gram stain shows no organism but many leucocytes. HSV 1 DNA, QN PCR, CSF came back as 962,013 copies/ml. EEG showed the presence of focal activity occurring rhythmically in the left frontal-temporal region which can be seen in patients with the clinical diagnosis of encephalitis.

He developed a fever on day 4 of hospitalization. MRI on the 4th day of hospitalization showed



Figure 2. MRI brain 4 days after hospitalization, showing increasing edema in left temporal lobe and insula and slight mass effect.

increasing parenchymal edema within the temporal lobe and insula with slight mass effect to the adjacent structures (Image 2). The patient was transferred to acute neurology rehab with 3 weeks of intravenous therapy followed by oral therapy. He eventually recovered fully after the rehabilitation and was able to go back to work 6 weeks later.

## 3. Discussion

Typical acute symptoms of HSVE infections include fever, headache, altered mental status, focal neurological abnormalities and seizures [3]. Fever and abnormal mental status are the primary signs and symptoms of HSVE, occurring in >90% of patients. As herpes encephalitis normally affects temporal lobes, symptoms of herpes encephalitis can include aphasia, confusion and behavioral changes.

The patient we report presented with fluent aphasia as his primary symptom. He had a frontal headache but did not have any other prodromal symptoms like fevers, seizures or altered mental status.

There are only very few case reports of aphasia as presenting symptom of herpes encephalitis. Most HSVE have the above-mentioned typical symptoms associated with aphasia on presentation. To our knowledge, there are only 2 case reports where the patients presented with aphasia which was not associated with a prodrome of fever, seizures or sustained altered mental status. In the first report, the patient presented with fluent aphasia and fluctuation of mentation. She had prodromal symptoms of headache, nausea, vomiting, and fatigue the day prior to the presentation [4]. On admission her temperature was mildly elevated at 100.2 F. In the 2nd report, the patient presented with symptoms suggestive of a typical MCA stroke of global aphasia, left gaze deviation and right-sided hemiparesis [5]. This patient developed a fever of 101.4 F, 11 hours after admission.

Acute stroke is an immediate consideration in anyone presenting with acute aphasia. The patient we presented arrived in the ED within an hour of symptom onset. Since early imaging can be normal in acute stroke, as was in this patient, tPA was administered. The patient who presented with MCA stroke features as reported in the case report above did not receive tPA since it was outside the 3-hour window but was admitted to an acute stroke unit for close monitoring [5]. The MRI at 24 hours after admission in the patient we reported showed an increased FLAIR and T2 signal hyperintensity within the medial left temporal lobe with slight effacement of the cysts sulci which was concerning for encephalitis. CSF analysis confirmed the diagnosis with a finding positive for HSV-1 PCR. This has a 98% sensitivity and 94% specificity for the diagnosis of HSVE [4].

When damage occurs to Brodmann area 22 (Wernicke's area), located in the superior temporal gyrus, it can present as Wernicke's aphasia (e.g. fluent or receptive aphasia), and is characterized by fluent, albeit nonsensical speech which is seen in this case [1,6]. The case report where the patient had fluent aphasia showed MRI signal abnormalities diffusely in the left temporal lobe [4]. Although the initial non-contrast MRI in our patient showed only the medial temporal lobe being affected, the MRI 4 days later showed a diffuse left temporal lobe involvement. Contrast enhanced MRI is more sensitive in detecting signal abnormalities involving inflammation. The initial MRI was non-contrast which could explain the limited abnormal findings.

Timely recognition and treatment can alter the mortality of HSVE from 70% to 14–30% [5,7]. Our patient responded well to the acyclovir which was started a day after admission, and he had a complete recovery in 6 weeks. Poor outcomes have been noted with Acyclovir therapy if started 2 days after admission [5]. However, even in patients who receive prompt treatment, neurocognitive deficits can last long term. A variety of rehabilitation methods have helped improve these deficits along with appropriate pharmacological therapies to improve the associated psychiatric issues [1].

### 4. Conclusion

Aphasia without fever or altered mental status is an unusual presentation of HSVE. It should be considered

as one of the possibilities in patients presenting with features suggestive of a stroke involving the language areas of the brain. Early recognition and treatment of HSVE reduce mortality and adverse outcomes.

#### **Disclosure statement**

No potential conflict of interest was reported by the authors.

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