

A Case of Isolated and Prolonged Global Aphasia: Ischemic Stroke or Aphasic Status Epilepticus?

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Authors' contributions

This work was carried out in collaboration between all authors. Authors EG, MG and AB have contributed to the collection of data and the drafting of the paper. Author AC oversaw the part of the paper relative to Nuclear Medicine. Author CC contributed to the section relative to neuroimaging. Authors MG and MDS reviewed the article. All authors read and approved the final manuscript.

Case Study

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ABSTRACT

Introduction: The most common cause of sudden isolated and prolonged global aphasia is acute stroke, affecting the cortical or subcortical language network. However, an aphasic status epilepticus (ASE) has to be considered as a possible differential diagnosis in awake patients presenting with acute and prolonged language impairment. ASE is suggestive of a localized dysfunction of language processing in the dominant hemisphere. ASE is a rare phenomenon and few cases are reported in the current literature. In the differential diagnosis between ASE and stroke with aphasia, FDG-PET imaging could be used when EEG shows no clear evidence of epileptic activity.

Case Presentation: We described a case of a 74 year-old woman who presented sudden

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onset of isolated and prolonged global aphasia; she suffered 5 months before of a left temporo-occipital hemorrhage and 20 days before a left hemispheric ischemic stroke. A new ischemic or hemorrhagic event was excluded by neuroimaging (CT and MRI, including DWI). Since several EEGs did not show ictal epileptic pattern, but only inter-ictal slow waves in the left temporal region, an FDG-PET was performed, resulting in two hypermetabolic areas in the left temporal and occipital lobes. The aphasia improved after anti-epileptic therapy.

Discussion and Conclusion: In conclusion, this is a case of post-stroke ASE, in which the evidence of hypermetabolism on FDG-PET allowed a definite diagnosis of epilepsy, despite the non-ictal EEG pattern.

Keywords: Aphasic status epilepticus; global aphasia; ischemic stroke; FDG-PET.

1. INTRODUCTION

The most common cause of sudden isolated and prolonged global aphasia is acute stroke affecting the cortical or subcortical language network. Non-convulsive status epilepticus can be associated with impaired speech and comprehension, but other cognitive symptoms such as altered responsiveness and awareness are typically the more salient clinical features. However, in awake patients presenting with acute and prolonged language impairment, aphasic status epilepticus (ASE) has to be considered, and it is suggestive of a localized dysfunction of language processing in the dominant hemisphere.

ASE is a rare phenomenon and few cases are reported in the literature. It has been proposed that, in presence of aphasic symptoms an electrographic seizure pattern is sufficient to confirm or exclude the diagnosis of ASE [1]; nevertheless, in ASE a routine EEG not always demonstrates electrographic seizure activity, even during period of speech impairment [2]. We described a case of a 74 year-old woman who presented sudden onset of isolated and prolonged global aphasia after two cerebrovascular events.

2. CASE PRESENTATION

A 74 year-old woman presented sudden onset of both isolated and prolonged global aphasia. In the personal history, there were temporo-occipital hemorrhages occurred 5 months before and a left hemispheric ischemic stroke (right hemiparesis with motor aphasia, almost completely reversed) occurred 20 days before the onset of aphasia. On admission, neurological examination showed mixed aphasia, without other focal deficits. A further ischemic or hemorrhagic event was excluded by neuroimaging (CT and MRI, including DWI). MRI excluded also other structural abnormalities.

Several EEGs including a prolonged EEG recording showed an inter-ictal pattern, consisting in theta-delta activity with spikes/sharp waves at 3-5 Hz in the left frontal-temporal region Fig. 1. 18F-Fluorodeoxyglucose (FDG)-PET was then performed, resulting in two hypermetabolic areas in the left temporal and occipital lobes. The patient was discharged 15 days after introduction of antiepileptic therapy (Carbamazepine 400 mg bid and Levetiracetam 500 mg bid), with an improvement of the aphasic symptoms. EEG abnormalities gradually decreased after introduction of anticonvulsant therapy Fig. 2.

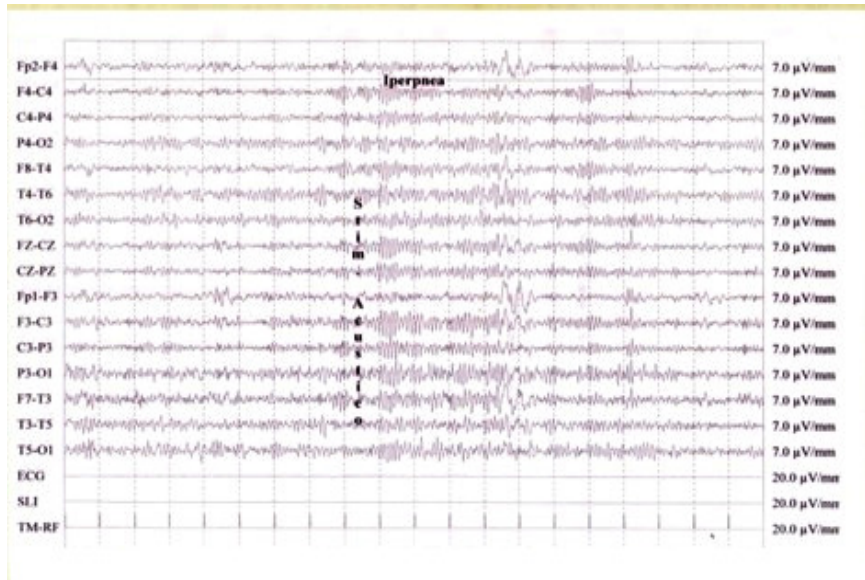


Fig. 1. EEG detected theta-delta waves on fronto-temporal region

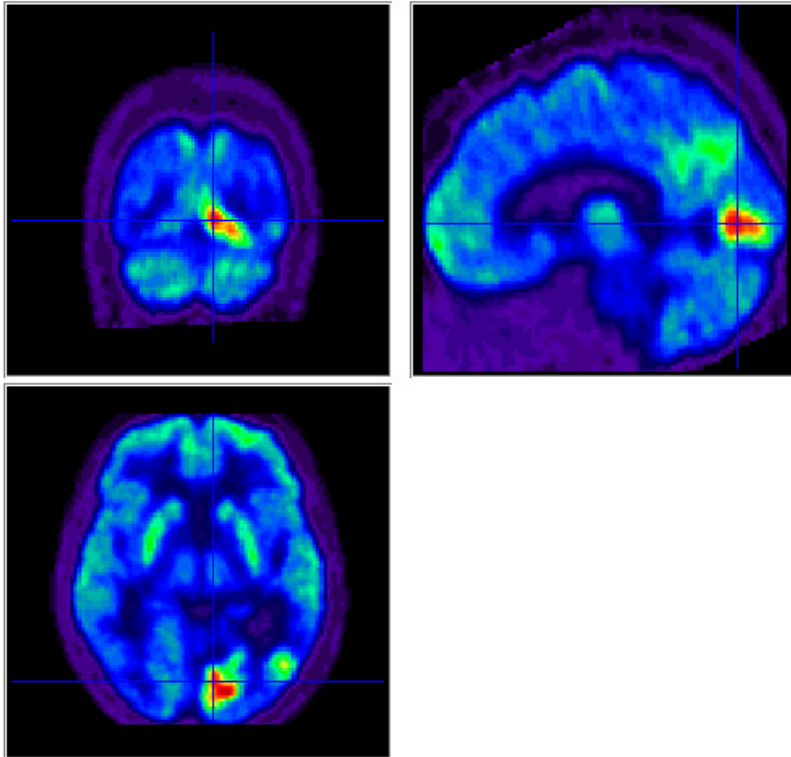


Fig. 2. FDG-PET resulting in two hypermetabolic areas in the left temporal and occipital lobes

3. DISCUSSION

Epileptic aphasia is defined according to Rosembaum's criteria as an aphasic speech in patients who are conscious, who are able to produce at least some speech that show aphasic errors and whose EEG studies confirm unequivocal seizure activity [3]. According to the review of the literature, patients should be considered to be in ASE if seizures persist for more than 5 minutes [4]. Status epilepticus is classified according to International League Against Epilepsy (ILAE) guidelines as either continuous clinical symptoms lasting more than 30 min or episodic clinical changes over a period of 30 min or more, without return to clinical baseline between events [3]. In 1986, Rosembaum proposed that in the presence of aphasic symptoms an EEG seizure pattern is sufficient to confirm or exclude the diagnosis of ASE. This is based on the assumption that the spatial resolution of a surface EEG study is sufficient to capture ictal changes during aphasic status and that clinical symptoms and electrographic seizure activity correlate each other. However, the electro clinical association may be disrupted in patients with status epilepticus; moreover, deficits can be persistent even when electrographic seizure activity is seen only intermittently [5]. In ASE a routine EEG not always demonstrates electrographic seizure activity, even during period of speech impairment [6] and patients may retain some aphasic deficits for several days or weeks after ictal EEG pattern resolution. Clinical examination by itself may not be sufficient to make definitive diagnosis [6]. Thus, patients ASE afflicted could remain inadequately characterized. Ericson and co-authors demonstrate that standard EEG is sensitive for detection of abnormalities in the dominant hemisphere in patients with ASE, yet continuous EEG is necessary to confirm and monitor treatment [7]. Functional imaging may help identify ictal activity. FDG-PET can demonstrate ictal hypermetabolism in the epileptogenic zone [8]. Ictal PET is extremely difficult to arrange for brief seizures, where SPECT done in the acute phase can easily localize the epileptogenic zone for single brief seizures. Furthermore, SPECT has been used to identify focal hyperperfusion in patients with epilepsy partialis continua with facial and extremity twitching and no associated EEG activity [9]. However, FDG-PET has many advantages over ictal SPECT in suspected non-convulsive status epilepticus; in fact, FDG-PET evaluates glucose metabolism, whereas SPECT evaluates perfusion, which is usually, but not consistently, coupled to metabolism. Then PET provides a more direct measure of cerebral activation; in addition, PET has higher spatial resolution than SPECT [2]. Despite the FDG-PET has several advantages over ictal SPECT in suspected non-convulsive SE, it should be regarded that ictal SPECT has a sensibility ranging in 90-100% to localize the epileptogenic focus. In the ASE, the time may not to be adequate, considering the radiopharmaceutical injection, to perform the SPECT [10]. The use of FDG-PET scans has been reported in the presurgical evaluation of patients with epilepsy [11].

ASE events are rare and cannot usually be planned during FDG uptake, so ictal PET is not used in routine clinical practice. Dong and coworkers showed that FDG-PET imaging should be considered as a diagnostic tool in patients with suspected ictal aphasia or amnesia, which fail to show clear evidence of ictal activity in EEG. They report five patients (4 with isolated aphasia and 1 with amnesia), all with hypermetabolism demonstrated by PET, in the absence of clear activity on EEG or structural abnormalities on MRI. FDG-PET imaging can be more sensitive in detecting ictal activity than EEG, and should be considered as a diagnostic tool in patients with suspected ictal aphasia who fail to show clear activity on EEG [2].

4. CONCLUSION

Our case is a post-stroke ASE, in which the evidence of hypermetabolism on FDG-PET allowed a definite diagnosis of epilepsy. In our case, aphasic status epilepticus may be only a reflection of cortical hyperactivity, suggesting a functional etiology for the prolonged language disorder. In conclusion, an aphasic status epilepticus (ASE) has to be considered as a possible differential diagnosis in awake patients presenting with acute and prolonged language impairment; FDG-PET, where possible, can allow definite differential diagnosis.

CONSENT

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

ETHICAL APPROVAL

Not applicable.

ACKNOWLEDGEMENTS

EG, MG, AB have contributed to the collection of data and the drafting of the paper. AC oversaw the part of the paper relative to Nuclear Medicine. CC contributed to the section relative to neuroimaging. MG and MDS reviewed the article.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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