

New Onset Absence Status Epilepticus in Pregnancy: A Case Report

Case Report

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Absence status epilepticus may occur in persons diagnosed with idiopathic/genetic epilepsy as well as *de novo* in adult and elderly patients. Despite being a rare phenomenon, pregnant women with no previous history of epileptic seizures may be presented with new onset status epilepticus. In this report, we describe the case of a 22-year-old pregnant female with no prior history of seizures. The patient was admitted to our center with reduced spontaneous speech and perplexity. Electroencephalography showed continuous, generalized synchronous paroxysms of 3 Hz spike-wave complexes. The patient's clinical condition improved following the administration of diazepam and levetiracetam. To the best of our knowledge, we describe the first case of new onset absence status epilepticus during pregnancy. (2024;14:94-96)

Key words: Absence status, Pregnancy, Status epilepticus, Absence seizure

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Introduction

Absence status epilepticus (ASE), a subtype of status epilepticus (SE), is primarily characterized by altered mental status, spanning from mildly compromised ideation and responsiveness to a state of stupor.¹ It has been reported that ASE typically develops in individuals diagnosed with idiopathic/genetic epilepsy syndrome (I/GGE), but it can also rarely arise *de novo*.¹ *De novo* ASE of late-onset can be precipitated by chronic alcohol abuse, benzodiazepine withdrawal, and metabolic disorders in elderly individuals.^{2,3} In pregnant women without a history of seizures, rarely, newly onset status epilepticus may be encountered. In such cases, underlying causes often include pregnancy-related hemodynamic changes, eclampsia, and cerebral thrombotic events.⁴ Here, we report a *de novo* ASE presentation observed in pregnancy and the possible underlying processes.

Case Report

A 22-year-old primigravid female, at the 13th week of gestation, presented to our outpatient clinic with recurrent episodes of fainting over a span of 2 days. These fainting episodes were characterized by marked pallor, loss of awareness, and involuntary contractions involving all extremities, with a duration up to 10 seconds. Additionally, her family reported that the patient appeared perplexed, and her

spontaneous speech along with the participation in daily life activities had notably diminished since the onset of these episodes. Before the onset of these symptoms, she had not shown any sign of cognitive impairment. Her medical and family history was unremarkable. On neurological examination, the patient was apathetic, and the overall spontaneity of the movement was considerably reduced. She gave one-word responses to the questions and had difficulties in understanding complicated questions and orders. The patient exhibited disorientation in time, and no discernible focal motor or sensory deficits were detected. During neurological assessment, she had staring spells that lasted up to 10 seconds. The blood pressure (BP) was within normal limits. Sodium level was 138 mEq/L (reference range, 136-145), potassium level was 4.2 mEq/L (reference range, 3.5-5.1), calcium level was 9.4 mg/dL (reference range, 8.4-10.2), creatinine was 0.8 mg/dL (reference range, 0.5-0.9), blood urea nitrogen was 15 mg/dL (reference range, 7-20), and thyroid-stimulating hormone was 3.7 µIU/mL (reference range, 0.27-4.2). Other routine laboratory tests and brain magnetic resonance imaging were unremarkable. Routine electroencephalography (EEG) revealed continuous, generalized synchronous paroxysms of 3 Hz spike-wave complexes (Fig. 1A). The described continuous activity persisted intermittently throughout the recording, occasionally accompanied by muscle artifacts, and apart from this, no evidence of distinct background activity could be discerned. After administering intravenous infusion of 10 mg of dia-

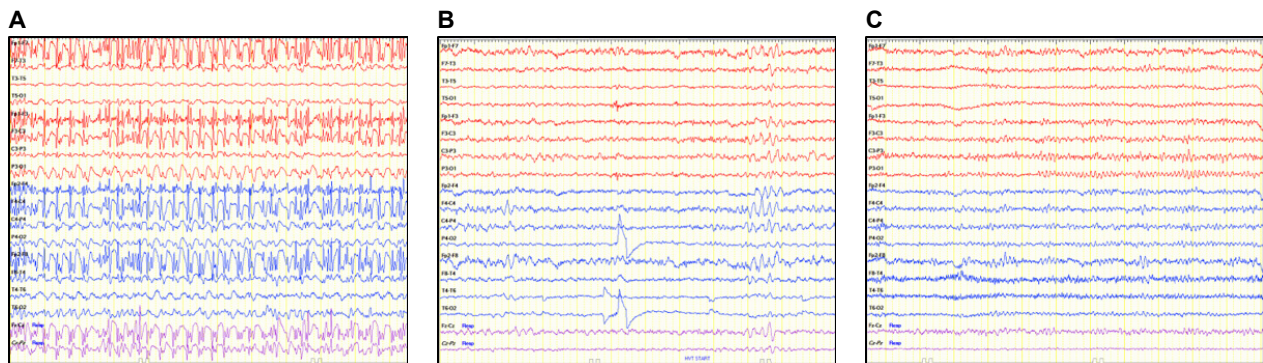


Figure 1. Electroencephalography (EEG) findings of the patient. (A) EEG shows continuous, generalized synchronous paroxysms of 3 Hz spike-wave complexes on day 1. (B) Control EEG demonstrates the suppression of the generalized synchronous activity on day 2. (C) Control EEG in the second month reveals normal background activity.

zepam, a daily regimen of levetiracetam at a dose of 1,000 mg was initiated. The following day, speech of the patient was ordinary, and she was keenly alert. On the second day of her hospitalization, EEG was repeated, and slow-wave paroxysms at the theta-delta frequency, rarely occurring over the low-amplitude background activity, were observed in the bilateral frontal regions (Fig. 1B). The patient, with clinical and electrophysiological findings returning to normal, was discharged on levetiracetam (1,000 mg/day).

Two months after discharge, she was in a seizure-free state and EEG showed normal background activity with posteriorly located alpha rhythm (Fig. 1C). The patient and her family were subsequently reexamined regarding any antecedent instances of epileptic seizures including myoclonia during her childhood and adolescence, to which they conveyed that no such paroxysmal episodes occurred. When the family was questioned about epilepsy, it was stated that no member of the family had a history of epileptic seizures.

The patient delivered a healthy baby at 38 weeks of gestation without any more seizures or pregnancy-related complications. She remained seizure-free on levetiracetam treatment at the 3 months follow-up after delivery.

Discussion

ASE is prominently characterized by prolonged state of altered consciousness and its distinctive EEG pattern features spike-wave complexes with a typical frequency of 3 Hz.¹ ASE has predominantly been linked to I/GGE and occasionally *de novo* ASE of late-onset.¹ Our case stands out as the first reported instance of *de novo* ASE in pregnancy in the literature.

In a pregnant patient with a new onset seizure, one of the clinical conditions that should be first ruled out is eclampsia. In our case, the exclusion of eclampsia was predicated upon the onset of SE before the 20th week of pregnancy coupled with the patient maintaining BP values within the established normal range.⁵

In women of reproductive age, epilepsy stands out as one of the most observed neurological conditions. The course of seizures can vary due to factors such as the limitations of anti-seizure drug usage, changes in their metabolism, alterations in sleep patterns, and hormonal fluctuations. Estrogen has been identified to increase susceptibility to seizures, with the estrogen to progesterone ratio reaching its peak in the first trimester before gradually declining.⁶ While the frequency of seizures remains the same in some women during pregnancy, an increase in seizures has been reported in approximately one-third of cases.⁷

On the other hand, the occurrence of the first seizure during pregnancy is a rare phenomenon. Studies have reported that the onset of the first seizure during pregnancy is observed in approximately 2.3-2.4% of women diagnosed with epilepsy.^{8,9} In our case, we interpreted the presentation of ASE as the possible first episode of I/GGE, particularly juvenile absence epilepsy (JAE), even though there was no previous history. Although JAE is typically diagnosed between the ages of 9 and 13, there have been rare instances reported with a later onset extending into young adulthood.¹⁰ While cases of I/GGE experiencing their first seizures during pregnancy are rare, a study from 1975 in an older cohort demonstrated that three out of 59 pregnant epilepsy patients experienced their first seizure during pregnancy.¹¹ In these reported cases, the described first seizures were generalized tonic-clonic in nature and occurred during the third

trimester and early postpartum period. The presence of seizures in our case as absence seizures is also noteworthy, and represents a rare, and previously unreported phenomenon.

Considering our case as *de novo* ASE, it is typically a clinical presentation encountered in pediatric cases and is often the initial manifestation of I/GGE. It is rarely encountered in adulthood, and in such cases, underlying causes may be provoked by benzodiazepine withdrawal, alcohol abuse, or the initiation of psychotropic drugs.^{2,3} Considering the absence of a pre-existing history of epileptic seizures, or any other precipitating factors in our patient, we highlight that the new onset ASE in our case could be attributed to hormonal changes during pregnancy. This addition underscores the potential role of pregnancy as a *de novo* cause of ASE in our patient.

Several limitations of this report should be acknowledged. First, our laboratory workup did not include the assessment of serum hormone levels. Second, during the short follow-up period, no seizure recurrence or EEG abnormalities were detected to support the preliminary diagnosis of the patient, who was evaluated as possible JAE. Third, it should be considered that there might be recall bias regarding absence seizures in the medical history of the patient, as short absence seizures are easily unrecognized. However, we believe this is unlikely since JAE is known for its high frequency of bilateral tonic-clonic seizures, and the absence of such episodes prior to this presentation strengthens the evidence that ASE could be the initial clinical manifestation.

In conclusion, we wanted to emphasize the rarity of encountering ASE in epilepsy clinical practice, and the infrequent occurrence of *de novo* cases. Our case underscores the significance of being the first reported instance of *de novo* ASE triggered by pregnancy in the literature to the best of our knowledge.

Conflict of Interest

The authors declare that they have no conflicts of interest.

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